

SIXTH EDITION

Exercise Physiology

For Health, Fitness,
and Performance

Denise L. Smith
Sharon A. Plowman
Michael J. Ormsbee



Wolters Kluwer

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**FOR HEALTH, FITNESS, AND
PERFORMANCE**

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Sixth Edition

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Dedication

*To our teachers and students,
past, present, and future:
sometimes one and the same.*

About the Authors



DENISE L. SMITH is the Tisch Family Distinguished Professor of Health and Human Physiological Sciences at Skidmore College. She also serves as the Director of the First Responder Health and Safety Research Laboratory. With a PhD in kinesiology and

specialization in exercise physiology from the University of Illinois at Urbana-Champaign, Dr. Smith has taught for over 30 years, including classes in anatomy and physiology, exercise physiology, clinical aspects of cardiovascular health, cardiorespiratory aspects of human performance, neuromuscular aspects of human performance, and research design. Her research is focused on the cardiovascular strain associated with heat stress, particularly as it relates to cardiac, vascular, and coagulatory responses to firefighting. Her research explores the intersection between the cardiovascular strain of firefighting and cardiac events in the fire service. Dr. Smith has received more than \$15 million in research funding and has published over 100 peer-reviewed articles in such journals as *American Journal of Cardiology*, *Cardiology in Review*, *Medicine & Science in Sports & Exercise*, *Exercise and Sport Sciences Reviews*, *Vascular Medicine*, *Ergonomics*, *European Journal of Applied Physiology*, *Journal of Applied Physiology*, and *Occupational Medicine*. She has been awarded the National Institute of Occupational Safety and Health Bullard-Sherwood Award for Research to Practice. She is also a coauthor of “Advanced Cardiovascular Exercise Physiology,” an upper-level text that is part of the Advanced Exercise Physiology series.

Dr. Smith is a Fellow in the American College of Sports Medicine and has served as secretary for the Occupational Physiology Interest Group and as a member of the National Strategic Health Initiative Committee. She works extensively with the major fire service organizations on issues related to firefighter health and safety. She is a member of the National Fire Protection Agency Technical Committee on Fire Service Occupational Safety and Health. She is also a Research Scientist at the University of Illinois Fire Service Institute at Urbana-Champaign.



SHARON A. PLOWMAN earned her PhD at the University of Illinois at Urbana–Champaign under the tutelage of Dr. T. K. Cureton Jr. She is a professor emeritus from the Department of Kinesiology and Physical Education at Northern Illinois University. Dr. Plowman taught for 36 years, including classes in exercise physiology, stress testing, and exercise bioenergetics. She has published over 75 scientific research articles in exercise physiology and applied articles on physical fitness with emphasis on accurate and appropriate test items, females and children in such journals as *ACSM's Health & Fitness Journal*, *Annals of Nutrition and Metabolism*, *Human Biology*, *Medicine & Science in Sports & Exercise*, *Pediatric Exercise Science*, and *Research Quarterly for Exercise and Sport*. She is a coauthor of the *Dictionary of the Sport and Exercise Sciences* (M. H. Anshel, ed., 1991), has published several chapters in other books, and is coeditor of the

2013 *FitnessGram® Reference Guide.*

Dr. Plowman is a Fellow Emeritus of the American College of Sports Medicine and served on the Board of Trustees of that organization from 1980 to 1983. In 1992, she was elected as an Active Fellow by the American Academy of Kinesiology and Physical Education (National Academy of Kinesiology). She has served on the Advisory Board for FitnessGram® from 1987 to the present. The American Alliance for Health, Physical Education, Recreation and Dance (AAHPERD) (now the Society of Health and Physical Educators [SHAPE]) recognized her with the Mabel Lee Award in 1976, the Physical Fitness Council Award in 1994, and the Measurement and Evaluation Council Lifetime Achievement Award in 2021. Dr. Plowman received the Excellence in Teaching Award (at Northern Illinois University at the department level in 1974 and 1975 and at the university level in 1975) and the Distinguished Alumni Award from the Department of Kinesiology at the University of Illinois at Urbana-Champaign in 1996. In 2006, the President's Council on Physical Fitness and Sports presented her with their Honor Award in recognition of her contributions to the advancement and promotion of the science of physical activity.



MICHAEL J. ORMSBEE is a Professor and Graduate Program Director in the Department of Nutrition and Integrative

Physiology and the Director of the Institute of Sports Sciences and Medicine at Florida State University (FSU). He is also an honorary research fellow at the University of KwaZulu-Natal in Durban, South Africa. Dr. Ormsbee earned his PhD in Bioenergetics from East Carolina University, his MS in Exercise Science from South Dakota State University, and his BS in Exercise Science from Skidmore College. He has taught classes in both exercise physiology and nutrition, including sports nutrition and endocrinology. His research expertise involves the interaction of exercise training, nutrition, and supplementation to improve metabolism and achieve optimal body composition, human performance, and health in athletic and clinical populations. Dr. Ormsbee has been awarded over \$6 million in industry and federal research funding and has more than 80 scientific publications. He published several book chapters on exercise and nutrition in sport and wrote “Changing Body Composition through Diet and Exercise,” which is a book and video series available to the public.

Dr. Ormsbee is a Fellow of the American College of Sports Medicine and the International Society of Sports Nutrition, and he is a Certified Strength & Conditioning Specialist through the National Strength & Conditioning Association (NSCA). Dr. Ormsbee was honored as the 2013 Transformation through Teaching Award Winner, 2014 Teacher of the Year, 2017 Nutrition Researcher of the Year (NSCA), 2018 Student Mentor of the Year, 2020 Distinguished Teacher of the Year, and the 2020 Sport Scientist of the Year (NSCA). You can follow him online [@mikeormsbee](#).

Preface

The sixth edition of *Exercise Physiology for Health, Fitness, and Performance* builds upon and expands the strength of the first five editions. The purpose of the current edition, however, remains unchanged. That is, the goal is to present exercise physiology concepts in a clear and comprehensive way that will allow students to apply fundamental principles of exercise physiology in the widest variety of possible work situations. The primary audience is kinesiology, exercise science, health, coaching, and physical education majors and minors, including students in teaching preparation programs and students in exercise and sport science tracts where the goal is to prepare for careers in fitness, rehabilitation, athletic training, or allied health professions.

As with other textbooks in the field, a great deal of information is presented. Most of the information has been summarized and conceptualized based on extensive research findings. However, we have occasionally included specific research studies to illustrate certain points, believing that students need to develop an appreciation for research and the constancy of change that research precipitates. **Focus on Research** boxes, including some that are labeled as **Clinically Relevant**, are integrated into the text to help students understand how research informs our understanding of exercise physiology and how research findings can be applied in the field. Our definition of the designation “Clinically Relevant” is used in the broadest sense to refer to a variety of situations that students of exercise physiology might find themselves in during an internship situation or eventual employment. All Focus on Research boxes highlight important classic or recent basic and applied studies in exercise physiology, as well as relevant experimental design considerations. **Literature Search** exercises at the end of each chapter help reinforce the importance of reading literature and

provide opportunity to strengthen literature searching skills.

All chapters are thoroughly referenced, and a complete list of references is provided at the end of each chapter. These references should prove to be a useful resource for students to explore topics in more detail for laboratory reports or term projects. The extensive referencing also reinforces the point that our knowledge in exercise physiology is based on a foundation of rigorous research.

The body of knowledge in exercise physiology is extensive and growing every day. Each individual faculty member must determine what is essential for his or her students. To this end, we have tried to allow for choice and flexibility, particularly in the organization of the content of the book.

A Unique Integrative Approach

The intent of this textbook is to present the body of knowledge based on the traditions of exercise physiology but in a way that is not bound by those traditions. Instead of proceeding from a unit on basic science, through units of applied science, to a final unit of special populations or situations (which can lead to the false sense that scientific theories and applications can and should be separated), we have chosen a completely integrative approach to make the link between basic theories and applied concepts both strong and logical.

Flexible Organization

The text begins with an introductory chapter: The Warm-Up. This chapter is intended to prepare students for the chapters that follow. It explains the text's organization, provides an overview of exercise physiology, and establishes the basic terminology and concepts that will be covered in each unit. Paying close attention to this chapter will help the student when studying the ensuing chapters.

Four major units follow: Metabolic System, Cardiovascular-Respiratory System, Neuromuscular-Skeletal System, and

Neuroendocrine-Immune System. Although the units are presented in this order, each unit can stand alone and has been written in such a way that it may be taught before or after each of the other three with the assumption that [Chapter 1](#) (The Warm-Up) will always precede whichever unit the faculty member decides to present first. [Figure 1.1](#) depicts the circular integration of the units reinforcing the basic concepts that all of the systems of the body respond to exercise in an integrated way and that the order of presentation can logically begin with any unit. Unit openers and graphics throughout the text reinforce this concept.

Consistent Sequence of Presentation

To lay a solid pedagogical foundation, the chapters in each unit follow a consistent sequence of presentation: basic anatomy and physiology, the measurement and meaning of variables important to understanding exercise physiology, exercise responses, training principles and adaptations, and special applications, problems, and considerations.

Basic Sciences

It is assumed that the students using this text will have had a basic course in anatomy, physiology, chemistry, and math. However, sufficient information is presented in the basic chapters to provide a background for what follows if this is not the case. For those students with a broad background, the basic chapters can serve as a review; for those students who do not need this review, the basic chapters can be de-emphasized.

Measurement

Inclusion of the measurement sections serves two purposes—to identify how the variables most frequently used in exercise physiology are obtained and to contrast criterion or laboratory test results with field test results. Criterion or laboratory results are essential for accurate determination and understanding of the

exercise responses and training adaptations, but field test results are often the only items available to professionals in school or health club settings.

Exercise Responses and Training Adaptations

The chapters or sections on **exercise responses** and **training adaptations** present the definitive and core information for exercise physiology. Exercise response chapters are organized by exercise modality and intensity. Specifically, physiological responses to the following six categories of exercise (based on the duration, intensity, and type of muscle contraction) are presented when sufficient data are available: (1) short-term, light to moderate submaximal aerobic exercise; (2) long-term, moderate to heavy submaximal aerobic exercise; (3) incremental aerobic exercise to maximum; (4) static exercise; (5) dynamic resistance exercise; and (6) very short-term, high-intensity anaerobic exercise. Training principles for the prescription of exercise training programs are presented for each physical fitness component: aerobic and anaerobic metabolism, body composition, cardiovascular endurance, muscular strength and endurance, flexibility, and balance. These principles are followed by the training adaptations that will result from a well-prescribed training program.

Special Applications

The special application chapters always relate the unit topic to health-related physical fitness and then deal with such diverse topics as altitude and thermoregulation (Cardiovascular-Respiratory Unit); making weight and eating disorders (Metabolic Unit); muscle fatigue and soreness (Neuromuscular-Skeletal Unit); and Overreaching/Overtraining Syndrome (Neuroendocrine Immune Unit). **Focus on Application** and **Focus on Application—Clinically Relevant** boxes emphasize how research and underlying exercise physiology principles are relevant to the practitioner.

Complete Integration of Age Groups and Sexes

A major departure from tradition in the organization of this text is the complete integration of information relevant to all age groups and both sexes. In the past, there was good reason to describe evidence and derive concepts based on information from male college students and elite male athletes. These were the samples of the population most involved in physical activity and sport, and they were the groups most frequently studied. As more women, children, and older adults began participating in sport and fitness programs, information became available on these groups. Chapters on females, children/adolescents, and the elderly were often added to the back of an exercise physiology text as supplemental material. However, most physical education, kinesiology, exercise science, and allied health professionals will be dealing with both male and female children and adolescents in school settings, average middle-aged adults in health clubs or fitness centers, older adults in special programs, and both sexes of all ages in allied health settings. Very few will be dealing strictly with college-aged students, and fewer still will work with elite athletes. This does not mean that information based on young adult males has been excluded or even de-emphasized. However, it does mean that it is time to move coverage of the groups that make up most of the population from the back of the book and integrate information about males and females at various ages throughout the text. That being said, these sections are typically stand-alone, allowing the faculty member to give the individual students freedom to select a population they are primarily interested in learning about.

Pedagogical Considerations

This text incorporates multiple pedagogical techniques to support student learning. These techniques include a chapter table of contents and a list of learning objectives at the beginning of each chapter as well as a chapter summary, review questions, and

references at the end of each chapter. Another pedagogical aid is the use of a running glossary. Terms are presented in definition boxes as they are introduced and are colored in bold type and defined in the text where they first appear to emphasize the context in which they are used. A glossary is included in the back matter of the book for easy reference. Additional important technical terms with which students should be familiar are italicized in the text to emphasize their importance. Because so many are used, a list of commonly used symbols and abbreviations with their meanings is printed on the front endpapers of the text for quick and easy reference. Each chapter contains a multitude of tables, charts, diagrams, and photographs to underscore the pedagogy, to aid in the organization of material, and to enhance the visual appeal of the text. Figure and table numbers are highlighted in color in the text to identify the relevant textual material instantly.

Unique Color Coding

A unique aspect of the graphs is color coding, which allows for quick recognition of the condition represented. Because it is so critical to recognize the differences among exercise responses to different types of exercise, we use a specific background color for each category of exercise. Further, we differentiate the responses to an acute bout of exercise from training adaptations that occur as a result of a consistent training program with a specific background color. For exercise response patterns, each of the six exercise categories has its own shaded representative color and accompanying icon. A key to these colors and icons is included in [Table 1.2](#). Population comparisons (male-female, children/adolescents-adults, trained-untrained) are also color coded on graphs where applicable.

Active Learning

Throughout the text, **Check Your Comprehension** and **Check Your Comprehension-Case Study** boxes engage the student in

active learning beyond just reading. The number of these was expanded in the fifth edition at faculty request. In some instances, the boxes require students to work through problems that address their understanding of the material. In other instances, students are asked to interpret a set of circumstances or deduce an answer based on previously presented information. Scattered throughout the text and occasionally used in Check Your Comprehension boxes are equations and problems used to calculate specific variables in exercise physiology. Examples using all equations are included in discrete sections in the text. Individual faculty members can determine how best to use or not use these portions of the text to fit their individual situations and student needs. Each chapter ends with a set of essay review questions.

Appendices

[Appendix A](#) provides information on the metric system, units, symbols, and conversion both with and between the metric and English systems. [Appendix B](#) offers supplementary material, consisting of three parts that deal with aspects of oxygen consumption calculation. [Appendix C](#) provides answers to the Check Your Comprehension/Check Your Comprehension—Case Study boxes that appear throughout the text.

Online Resources

A comprehensive set of ancillary materials designed to facilitate classroom preparation and ease the transition into a new text is available to students and instructors using *Exercise Physiology for Health, Fitness, and Performance*, sixth edition.

For Students

- Quiz bank of multiple choice questions intended to assist in studying the material or for self-testing. Answers are accessible.

- Worksheets that include true/false questions (with space for correcting false statements), fill-in tables, figure labeling, matching, and calculations to assist in studying or for self-testing. Worksheet answers are also available to students.
- Laboratory manual.
- Online animations.

For Faculty

- Image bank of all figures in the text
- PowerPoint lecture outlines
- Test bank
- Answers to in-text chapter review questions

User's Guide

This User's Guide explains the key features found in the sixth edition of *Exercise Physiology for Health, Fitness, and Performance*.

Get the most out of your learning and study time so you can master exercise physiology principles and move on to career success!

Commonly Used Symbols and Abbreviations

You can find this useful resource just inside the front cover of the text.

Commonly Used Symbols and Abbreviations

| | | | |
|---------------------------|---|--------------------------------|---|
| (A-a)PO ₂ diff | difference between partial pressure of oxygen in alveoli and arterial blood | EMG | electromyogram |
| a-vO ₂ diff | difference in oxygen content between arterial and venous blood | EPOC | excess postexercise oxygen consumption |
| A | actin | ERT | oxygen replacement therapy |
| ACh | acetylcholine | ESV | end-systolic volume |
| ACTH | adrenocorticotrophic hormone | ETAP | exercise-related transient abdominal pain |
| ADH | antidiuretic hormone | ETS | electron transport system |
| ADL | activities of daily living | F _i CO ₂ | fraction of expired carbon dioxide |
| ADP | adenosine diphosphate | F _i N ₂ | fraction of expired nitrogen |
| AI | adequate intake | F _i O ₂ | fraction of expired oxygen |
| AIDS | acquired immune deficiency syndrome | F _i | fraction of a gas |
| AMP | adenosine monophosphate | F _i CO ₂ | fraction of inspired carbon dioxide |
| AN | anorexia nervosa | F _i N ₂ | fraction of inspired nitrogen |
| ANS | autonomic nervous system | F _i O ₂ | fraction of inspired oxygen |
| AP | action potential | f | frequency |
| ATP | adenosine triphosphate | FAD | flavin adenine dinucleotide |
| ATP-PC | ATP-phosphocreatine | FEV | forced expiratory volume |
| ATPS | atmospheric temperature and pressure, saturated air | FEA | free fatty acids |
| AV | atrioventricular | FFB | fat-free body mass |
| BCAA | branched chain amino acids | FFM | fat-free mass |
| BED | binge-eating disorder | FFW | fat-free weight |
| BF | body fat | FG | fast-twitch, glycolytic muscle fibers |
| BMC | bone mineral content | FI | fatigue index |
| BMD | bone mineral density | FOG | fast-twitch, oxidative-glycolytic muscle fibers |
| BMI | body mass index | FT | fast-twitch muscle fibers |
| BMR | basal metabolic rate | GAS | general adaptation syndrome |
| BN | bulimia nervosa | GH | growth hormone |
| BP | blood pressure | GI | glycemic index |
| BTPS | body temperature and pressure, saturated | GLUT-1 | non-insulin-regulated glucose transporter |
| air | | GLUT-4 | insulin-regulated glucose transporter |
| BW | body weight | GTO | Golgi tendon organ |
| CAD | coronary artery disease | Hb | hemoglobin |
| CHD | coronary heart disease | HbO ₂ | oxyhemoglobin |
| CHO | carbohydrate | HDL-C | high-density lipoprotein cholesterol |
| CNS | central nervous system | HIV | human immunosuppression virus |
| CO ₂ | carbon dioxide | HR | heart rate |
| CP | creatine phosphate | HRmax | maximal heart rate |
| CR-10 | category ratio scale of perceived exertion | HRR | heart rate reserve |
| CVD | cardiovascular disease | HRrec | heart rate recovery |
| D ₅₀ | density of the body | HRV | heart rate variability |
| D _w | density of water | HT | height |
| DBP | diastolic blood pressure | ICD | isocitrate dehydrogenase |
| DOMS | delayed-onset muscle soreness | ICP | isovolumetric contraction period |
| DRI | daily reference intake | IRP | isovolumetric relaxation period |
| DXA | dual-energy x-ray absorptiometry | LA | lactic acid/glycolytic system |
| E | epinephrine | LBM | lean body mass |
| ECG | electrocardiogram | LBP | low back pain |
| EDV | end-diastolic volume | LDL-C | low-density lipoprotein cholesterol |
| EF | ejection fraction | LS | long, slow distance |
| EIAH | exercise-induced arterial hypoxemia | LT | lactate threshold |
| | | M _a | mass of the body in the air |
| | | M _w | mass of the body underwater |

Chapter Objectives

These are the learning objectives that you need to meet after reading the chapter.



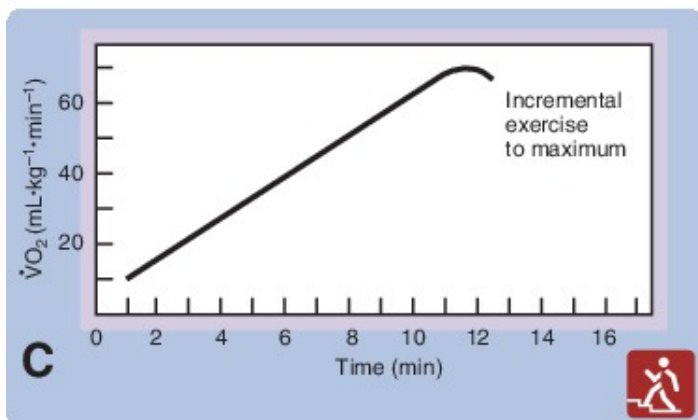
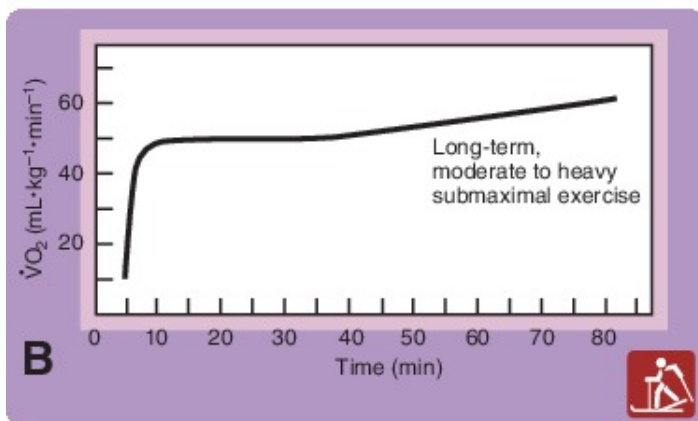
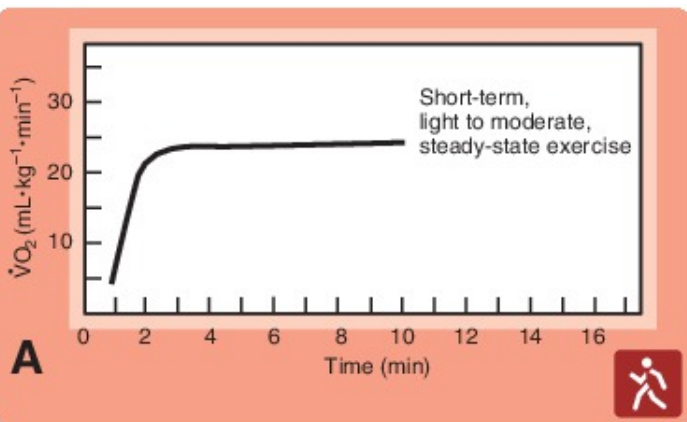
OBJECTIVES

After studying the chapter, you should be able to:










- Articulate the importance of an exercise professional having a strong understanding of the influence of heat stress on safety and performance.
- Identify environmental factors that affect thermoregulation and be able to use indices of heat stress and windchill to assess the risks associated with exercise under various conditions.
- Describe thermal balance and discuss factors that contribute to heat gain and heat loss.
- Define the mechanisms by which heat is lost from the body, and describe how they differ under exercise conditions.
- Describe the body's regulatory system for temperature control in terms of the sensory input, neural integration, and effector responses to increase or decrease heat loss.

Data Graphs

Each chapter contains a multitude of graphs, tables, charts, and diagrams that clarify and enhance points made in the text.



Color tints and bold icons within figures and figure legends help you quickly distinguish the exercise response to six different categories of exercise.

| TABLE 1.2 Color and Icon Interpretation for Exercise Response Patterns | | |
|--|---|---|
| Exercise Category | Color | Icon |
| Short-term, light to moderate submaximal aerobic |  |  |
| Long-term, moderate to heavy submaximal aerobic |  |  |
| Incremental aerobic to maximum |  |  |
| Static |  |  |
| Dynamic resistance |  |  |
| Very short-term, high-intensity anaerobic |  |  |

Clinically Relevant Boxes

Specially identified boxes highlight clinical information, situations, or case studies that you may experience during an internship or future employment.

Focus on Research Boxes

Classic, illustrative, and cutting-edge research studies are presented to help you develop an appreciation for how research affects changing practices in the field.

FOCUS ON RESEARCH Clinically Relevant

Childhood Obesity and CVD Risk Factors

A total of 1,166 White (C) and 1,213 African American (AA) girls were tested in the National Heart, Lung, and Blood Institute Growth and Health Study annually between ages 9 or 10 years and 18 years and were contacted for self-reported measures at age 21–23 years. At the time the study began, BMI values above the CDC's age-specific 95th percentile were labeled as overweight. Because these values are now labeled obese, the term obesity will be used here. Young adult obesity was defined as a BMI $\geq 30 \text{ kg m}^{-2}$.

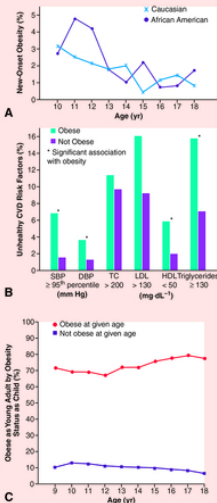
The rate of obesity increased throughout adolescence from 7 to 10% in the C girls and from 17 to 24% in the AA girls (not shown). Graph A shows the percent of new-onset cases (incidence) of obesity by race over the age span. The incidence ranged from 2 to 5% through age 12, after which the annual increase was generally in the 1–2% range. The important part of this information is the fact that it is the so-called tween years (ages 9–12 years) when girls are especially at risk of getting fat. Thus, particular attention and intervention should be directed toward this age group to prevent the increase in body weight and fat.

Graph B shows the percentage of selected cardiovascular risk factors in the C and AA groups combined, based on being or not being obese. Girls who were obese were 3–10 times as likely as those who

were not obese to be assessed as “at risk” on four of the six cardiovascular risk factors: unhealthy levels of systolic and diastolic blood pressure (SBP and DBP), high-density lipoproteins (HDL), and triglyceride levels. The increased risk was already evident at age 9 years. Thus, there are meaningful health reasons for not delaying interventions.

Graph C shows the percentage of obese girls in young adulthood who were obese or not at each age in childhood. For example, 71.3% of the obese 9-year-olds were obese at approximately 21 years, but only 10.3% of the nonobese at age 9 were obese at approximately 21 years. Overall, girls who were obese during childhood were 11 to 30 times more likely to be obese in young adulthood. Given that the height and weight values were self-reported by the young adults, and underreporting weight is a known problem, the risk could be even greater. These data clearly show that obesity tracks from childhood to adulthood and needs to be addressed with children.

Source: Thompson, D. R., E. Obarzanek, D. L. Franko, et al.: Childhood overweight and cardiovascular disease risk factors: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics*. 150:18–25 (2007).



childhood, rather than in old age (Bray, 1987; Burton et al., 1985; Lee et al., 2007; Pi-Sunyer, 1993; Simopoulos, 1987; Wang et al., 2004). Worldwide, the trends are similar. Epidemiological studies from not only the United States but also Europe and Asia have found that higher BMI values are significantly associated with increased incidence of coronary artery disease and ischemic stroke (Yatsuya et al., 2014). A recent 2020 meta-analysis found that higher BMI levels are associated with increased rates of cardiovascular events. Furthermore, among those with cardiovascular diseases, overall mortality rates displayed a U-shaped relationship with BMI (Dwivedi et al., 2020).

However, several studies have found that waist circumference or waist-to-hip ratio may be a better predictor of CVD than BMI (Ji et al., 2018; Lavie et al., 2018; van Dijk et al., 2012).

A study of 276,835 Dutch children showed that higher BMI values during childhood (7–13 years of age) were associated with an increased risk of coronary heart disease in adulthood. The associations were stronger in boys than girls and increased as the child became older in both sexes (Baker et al., 2007). Among other things, the Focus on Research: Clinically Relevant box provides evidence of increased CVD risk factors in female children and adolescents with BMI values greater than the 95th percentile (Thompson et al., 2007).

As mentioned previously, the risk is higher for those who store their fat in the android pattern than in the gynoid pattern. While coronary heart disease is linked to diabetes mellitus, hypertension, and hyperlipidemia (primarily metabolic changes), congestive heart failure is more related to the increase in total fat mass per se.

Focus on Application Boxes

These features apply basic concepts, principles, or research findings to relevant practical situations, concerns, or recommendations.

FOCUS ON APPLICATION

Caloric Cost and Exercise Machines

After studying exercise physiology for hours, to take a break, you go to the campus recreation center to exercise. Your goal is to burn 300 kcal, so you hop on your favorite exercise equipment, punch in your BW, select manual protocol, and begin. When the console reads 300 kcal, you stop—proud of having attained your goal. But did you really?

The answer to that question depends on a number of factors. The console number for kcal is derived mathematically from a prediction equation that typically takes into account BW and one or more measures of workload, such as stride rate, stride length, belt speed, elevation, and resistance or power output, depending on whether the equipment is a stair-stepping machine, treadmill, elliptical strider, rowing machine, or cycle ergometer. Research studies have generally shown that under identical conditions, caloric cost estimations are very consistent (reliable). However, the same cannot be said for the accuracy (validity) of the caloric cost values. For example, Swain et al. (1999) found that an elliptical motion machine significantly overestimated caloric cost (from 39% to 79%), with the larger overestimations occurring at the higher exercise intensities. In another study on an elliptical striding machine, Heseltown et al. (2000) found that although the mean caloric cost values at two of three workloads were not significantly different, the estimated values were systematically overestimated at levels above 300 kcal for a

30-minute workout, and the percentage of individuals whose measured caloric cost fell within 30 kcal of their estimated caloric cost was only 60%, averaged over two trials. A third study using an elliptical strider by Mier and Feito (2006) measured overestimations averaging 20–30% in caloric expenditure regardless of whether the participants used their legs only or arms and legs combined. Recently, it was suggested that elliptical trainers tend to overestimate caloric cost by at least 100 calories for every 30 minutes of exercise (Glave et al., 2018). Similar overestimations have been found for stair-stepping machines (Riddle and Orringer, 1990; Ryan et al., 1998), and several studies have reported that holding on to the point where part of the BW is supported results

in a significant overestimation of caloric expenditure for both stair-steppers and the treadmill (Åstrand, 1984; Butts et al., 1993; Howley et al., 1992). The manufacturers of these machines try to provide accurate information to the exerciser, but errors are part of predictions, and the mathematical program cannot adjust if you “cheat” by holding on. Therefore, the answer to the question of whether you actually burned 300 kcal is “probably not.” Console values for caloric expenditure should be interpreted as approximations rather than absolutes, and that approximation is probably high.

Sources: Åstrand (1984); Butts et al. (1993); Glave et al., 2018; Heseltown et al. (2000); Howley et al. (1992); Mier and Feito (2006); Riddle and Orringer (1990); Ryan et al. (1998); Swain et al. (1999).



expected to be sustained for only 1–2 hours occasionally. (Note: 12 METs = $42 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \text{ VO}_2 \text{ max}$) Generally, sedentary behaviors are considered to be MET values less than 1.5, light-intensity activities 1.6–2.9, moderate-intensity 3–6, and vigorous 6 or more (Ainsworth et al., 2011). Happily we can see that sitting in class, taking notes, participating in discussions, and studying are relatively light metabolic work that can be sustained indefinitely.

Table 4.8 presents a classification of metabolic intensities in MET values for selected physical activities. This allows an individual to find activities for any of the intensity levels for exercise found in **Table 4.6**. For example, on average, water skiing (6 METs) would be appropriate as a moderate activity for an individual whose $\text{VO}_2 \text{ max}$ is 10 or 12 METs but not for those with lower MET max levels. Walking at $3.0 \text{ mi} \cdot \text{hr}^{-1}$ (3.5 METs) would be an appropriate activity for an individual whose $\text{VO}_2 \text{ max}$

Body System Responses to Exercise

Consistently formatted diagrams clearly show how each body system responds to exercise in an integrated fashion and how those responses are interdependent.

by rearranging Fick's equation (see Equation 11.13) to the following equation:

$$\dot{V}O_2 \max = \dot{Q} \max \times (a-vO_2 \text{ diff} \max)$$

The rectilinear increase in cardiac output during a maximal incremental exercise test is described above. The changes in the $a-vO_2 \text{ diff}$ (discussed in Chapter 10) are an increase with a plateau at approximately 60% $\dot{V}O_2 \max$. The result of these changes is the rectilinear rise in oxygen up to maximum, which has been discussed.

Maximal exercise tests that include the measurement of oxygen consumption for the determination of $\dot{V}O_2 \max$ are routinely administered by coaches and trainers to determine an athlete's fitness or to track changes in fitness, by researchers to better understand the mechanisms that limit exercise or to probe questions related to physiological function under stressful conditions, and by medical personnel to assess cardiorespiratory function. The criteria for determining if an individual reaches maximal oxygen consumption during an exercise test are discussed in Chapter 4.

$\dot{V}O_2 \max$ is commonly used as the criterion measure of cardiorespiratory (also called aerobic) fitness. In reality, $\dot{V}O_2 \max$ is an integrated measure of fitness that encompasses the ability of the body to take in (respiratory system), transport (cardiovascular system), and utilize (the metabolic system) oxygen. Thus, $\dot{V}O_2 \max$ may be considered a cardiovascular, respiratory, and metabolic variable, and, indeed, for that reason, it is also discussed in other chapters. However, $\dot{V}O_2 \max$ has important implications for cardiovascular health and is normally thought to be limited by cardiovascular function; therefore, we will consider $\dot{V}O_2 \max$ primarily as a cardiovascular variable, and it is discussed in depth in this chapter.

FACTORS LIMITING At some point, an individual cannot continue to increase the intensity of the exercise load or to work at maximum effort because the body cannot provide and utilize more oxygen to support an additional workload. But what specifically limits $\dot{V}O_2 \max$?

Figure 12.10 summarizes possible limitations to oxygen

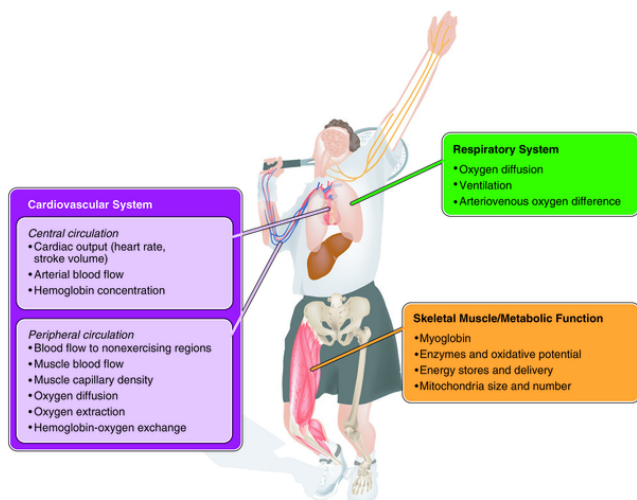


Figure 12.10. Possible Limitations to Maximal Oxygen Consumption.
Source: Modified from Rowell (1993).



Check Your Comprehension Boxes

These engaging mini-quizzes challenge you to work through problems, interpret circumstances, analyze information, or deduce answers to reinforce your learning as you move through each chapter.

$D_b = 1.0982 - (0.000815 \text{ sum of skinfolds} + 0.0000084 \text{ sum of skinfolds}^2)$ (ACSM, 1983)

EXAMPLE

If a 17-year-old wrestler weighs 165 lb and his sum of skinfolds for the selected sites is 46 mm, the calculation would be

$$D_b = 1.0982 - [0.000815 (46) + 0.0000084 (2116)] \\ = 1.0429 \text{ cc}^{-1}$$

The D_b value is then substituted into the age-appropriate formula presented in Table 7.2 in Chapter 7 for a male adolescent to determine %BF. For a 17-year-old, this is

$$\%BF = \left[\frac{5.03}{D_b} - 4.59 \right] \times 100 = 23.31$$

Equations 7.3, 7.4, and 7.5 are then used to determine the wrestler's most appropriate competitive weight. Using Equation 7.3,

$$FFW = 165 \text{ lb} \times \left[\frac{100\% - 23.3\%}{100} \right] = 126.6 \text{ lb}$$

Using Equation 7.3,

$$WT_2 = \left[\frac{100 \times 126.6}{100\% - 16.3\%} \right] = 151.3 \text{ lb}$$

Note: 16% is used here as the desirable %BF, not 5%, which is the lowest recommended %BF for a wrestler of this age. The 16.3% complies with the recommendation that weight loss not exceed 7% of body weight.

To get down to 5% BF, this wrestler would need to lose 18.3% of his body weight and that is too much. Using Equation 7.5,

$$151.3 \text{ lb} - 165 \text{ lb} = -13.7 \text{ lb}$$

To achieve his recommended body weight, this wrestler needs to lose 13.7 lb.

Despite several decades of work, harmful weight-making practices still are followed and the standards themselves are not without problem—one of which is uneven enforcement. It is important that expert guidance by a sports nutrition professional is available to each weight-category sport athlete to establish a workable long-term approach to body mass and body fat management. Such advice needs to take into account the nuances of the particular sport while attempting to achieve both positive

health and competitive performance outcomes for the athlete (Burke et al., 2021). Complete the case study in the Check Your Comprehension 3 box.

**CHECK YOUR COMPREHENSION
3-CASE STUDY 3**

Calculate the weight at which the following 14-year-old wrestler should compete.

| | |
|----------------|----------------------------|
| Name: Zachary | Triceps skinfold: 8 mm |
| Weight: 138 lb | Subscapular skinfold: 9 mm |
| | Abdominal skinfold: 12 mm |

How much weight does Zachary need to gain or lose to achieve this weight?

Check your answer in Appendix C.

Several studies have investigated compliance with the 1998 regulations. A 1999 survey of 43 collegiate teams (Oppliger et al., 2003) found that the most weight lost during the season was 6.9% of body weight, but average weekly weight loss was 4.3%. Although 40.2% indicated that the then-new NCAA rules deterred extreme weight loss behaviors, approximately 55% fasted, approximately 28% used saunas, and approximately 27% used vapor barrier suits at least once a month. Overall, however, compared to college wrestlers in the 1980s, weight behavior was less extreme. A follow-up study (Oppliger et al., 2006) of 811 competitors in Division I, II, and III national championship tournaments from 1999 to 2004 showed that weight and %BF decreased from preseason to postseason competition. However, the preseason certified minimum weight remained unchanged ($68.0 \pm 9.2 \text{ kg}$ vs. $67.9 \pm 9.1 \text{ kg}$), thus showing good agreement between the preseason recommendations and the actual end-of-season weights. Rapid weight loss before weigh-in was found to be statistically significant but small ($\sim 1.7\%$ of body weight). The average wrestlers at the tournaments were competing at 9.5% BF, down from the preseason average of $12.3 \pm 3.4\%$, but well above the minimum of 5%. The investigators concluded that the NCAA weight management program appears to be effective in reducing unhealthy weight-cutting behaviors (although the wrestlers were not asked how they achieved weight goals) and promoting competitive equity.

Summary

1. Weight gain, loss, and stabilization follow the first law of thermodynamics as expressed in the caloric balance equation. The components of this equation are food ingestion (+), resting or basal metabolic rate (–), thermogenesis (–), and exercise (–).

Clear and Accurate Artwork

Detailed anatomic illustrations and practice-related photos place key concepts in context.

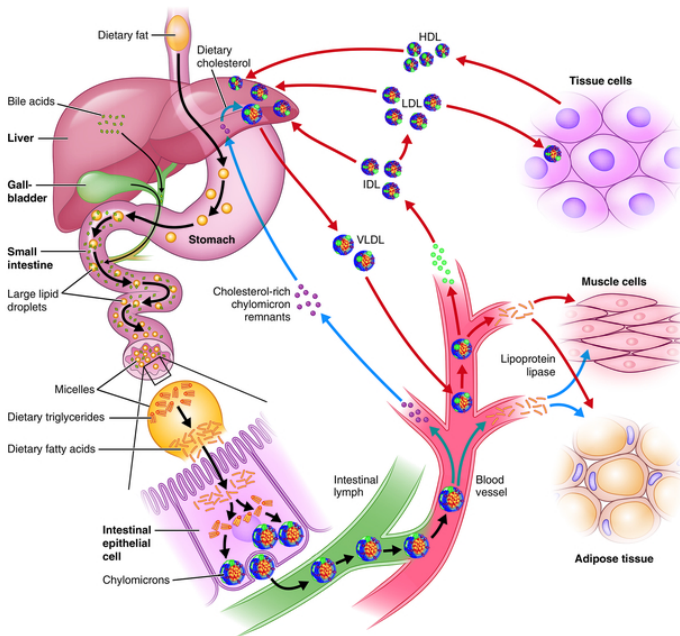


Figure 15.5. Fat and Cholesterol Transport in the Body.

(VLDLs) deliver triglycerides and cholesterol to adipose tissue for storage or to muscle for use as fuel. The removal of triglyceride from a VLDL results in an *intermediate-density lipoprotein* (IDL), which in turn is converted in the liver to a **low-density lipoprotein (LDL-C)**. LDL-C is composed of protein, a small portion of triglyceride, and a large portion of cholesterol. LDL-C transports 60–70% of the total cholesterol in the body to all cells except liver cells. The major apolipoprotein of LDL-C is called Apo-B. As mentioned earlier, LDL-C is involved in the formation of atherosclerotic plaque.

High-density lipoprotein (HDL-C) is a lipoprotein in blood plasma composed primarily of protein and a minimum of cholesterol or triglyceride. The purpose of HDL-C is to transport cholesterol from body tissues to

the liver where the cholesterol can be broken down and eliminated in bile.

Triglycerides (but not the associated VLDLs) represent an independent risk factor (Summary of the Third Report of the National Cholesterol Education Program

Low-Density Lipoprotein (LDL-C) A lipoprotein in blood plasma composed of protein, a small portion of triglyceride, and a large portion of cholesterol whose purpose is to transport cholesterol to the cells.

High-Density Lipoprotein (HDL-C) A lipoprotein in blood plasma composed primarily of protein and a minimum of cholesterol or triglyceride whose purpose is to transport cholesterol from the tissues to the liver.

Example Boxes

These highlighted equations enable you to visualize working out problems and calculate specific variables in exercise physiology.

Definition Boxes

Important terms are boldfaced in the text where they first appear to emphasize the context in which they are used. Definitions are provided in a callout box to create an on-the-spot glossary.

Chapter Summaries

Concise copy points review the chapter's core content.

Summary

1. A cardiovascular training program depends on the individual's age, health status, and the program's goals.
2. Any activity involving large muscle groups for a prolonged time has the potential to increase cardiovascular fitness. The choice of exercise modalities should be based on interest, availability, and a low risk of injury.
3. Training using different exercise modalities causes the same overall benefits with central cardiovascular adaptations, but peripheral cardiovascular adaptations are specific to the muscles being exercised.
4. Intensity is very important for improving maximal oxygen consumption ($\dot{V}O_{2\max}$) primarily in conjunction with duration, which determines training volume. Intensity can be prescribed in relation to heart rate, oxygen consumption, or rating of perceived exertion (RPE). Training intensity is the most important factor for maintaining cardiovascular fitness.
5. The ACSM recommends the following training goals to develop and maintain cardiorespiratory fitness in healthy adults: frequency of 3–5 d·wk⁻¹, intensity of 64–95% $\dot{V}O_{2\max}$, 40–89% $\dot{V}O_{2R}$ or %HRR, and duration of 20–60 minutes of continuous aerobic activity.
6. The Canadian Society for Exercise Science recommends a total of 150 min·wk⁻¹ of moderate to vigorous physical activity more activity leads to more health benefits.
7. Children and adolescents should participate in at least 60 min·d⁻¹ of moderate to vigorous physical activity that is age appropriate while minimizing sedentary activity the rest of the day.
8. $\dot{V}O_{2\max}$ work rate with endurance training. It is unchanged at the same relative submaximal workload and at maximal exercise.
13. $\dot{V}O_{2\max}$ increases with endurance training; improvements of 15% are routinely reported with training programs that meet the recommendations of ACSM.
14. Endurance training leads to positive structural and functional adaptations in the vasculature because of vascular remodeling and improved endothelial function.
15. Blood pressure changes little or not at all at rest, during submaximal exercise, or during maximal exercise in normotensive individuals with endurance training.
16. Endurance training results in increased blood volume, with highly trained endurance athletes having 20–25% greater volume than untrained subjects. Changes in plasma volume occur early in a training program, with an 8–10% change occurring within the first week. Early changes (at 1 month) are due almost entirely to increases in plasma volume, whereas increases in red blood cells and hemoglobin occur later.
17. Endurance training results in changes in blood formation and clot breakdown that decrease the likelihood of unnecessary clot formation.

Review Questions

1. How is overload manipulated to bring about cardiorespiratory adaptation? Consider exercise recommendations for fitness and physical activity guidelines for health benefit in your response.
2. Differentiate between central and peripheral cardiovascular adaptations.

Chapter Review Questions

Essay-style questions help you build your critical thinking, problem-solving, and decision-making skills.

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*Denise L. Smith
Sharon A. Plowman
Michael J. Ormsbee*

Commonly Used Symbols and Abbreviations

| | |
|-----------------------|---|
| $P_{aO_2} - P_{aO_2}$ | difference between partial pressure of oxygen in alveoli and arterial blood |
| $C_{aO_2} - C_{vO_2}$ | difference in oxygen content between arterial and venous blood |
| A | actin |
| ACh | acetylcholine |
| ACTH | adrenocorticotrophic hormone |
| ADH | antidiuretic hormone |
| ADL | activities of daily living |
| ADP | adenosine diphosphate |
| AI | adequate intake |
| AIDS | acquired immune deficiency syndrome |
| AMP | adenosine monophosphate |
| AN | anorexia nervosa |
| ANS | autonomic nervous system |
| A _p | action potential |
| ATP | adenosine triphosphate |
| ATPCr | phosphocreatine |
| T_{aPO_2} | atmospheric temperature and pressure, saturated air |
| AV | atrioventricular |
| BCAA | branched chain amino acids |
| BED | binge-eating disorder |
| BF | body fat |
| BCM | bone mineral content |
| BMD | bone mineral density |
| BMI | body mass index |
| BMR | basal metabolic rate |
| BN | bulimia nervosa |

BP blood pressure
 BT body temperature and pressure, saturated air
 BW body weight
 CAD coronary artery disease
 CHD coronary heart disease
 CHO carbohydrate
 CNS central nervous system
 CO₂ carbon dioxide
 CP creatine phosphate
 CR₁₀ category ratio scale of perceived exertion
 CVD cardiovascular disease
 D density of the body
 D_w density of water
 DBP diastolic blood pressure
 DOMS delayed-onset muscle soreness
 DRI daily reference intake
 DXA dual-energy x-ray absorptiometry
 E epinephrine
 ECG electrocardiogram
 EDV end-diastolic volume
 EF ejection fraction
 EIA exercise-induced arterial hypoxemia
 EMG electromyogram
 EPOC excess postexercise oxygen consumption
 EST estrogen replacement therapy
 ESV end-systolic volume
 ET exercise-related transient abdominal pain
 ETS electron transport system
 FE_{CO₂} fraction of expired carbon dioxide
 FE_{N₂} fraction of expired nitrogen
 FE_{O₂} fraction of expired oxygen
 FG fraction of a gas
 FI_{CO₂} fraction of inspired carbon dioxide
 FI_{N₂} fraction of inspired nitrogen
 FI_{O₂} fraction of inspired oxygen
 f frequency
 FAD flavin adenine dinucleotide
 FEV₁ forced expiratory volume
 FFA free fatty acids
 FFM fat-free body mass
 FFM fat-free mass
 FFM fat-free weight
 FG fast-twitch, glycolytic muscle fibers
 FI fatigue index

FC Fast-twitch, oxidative-glycolytic muscle fibers
 FT Fast-twitch muscle fibers
 GA General adaptation syndrome
 GH Growth hormone
 GI Glycemic index
 GLUT1 Insulin-regulated glucose transporter
 GLUT4 Insulin-regulated glucose transporter
 GT Golgi tendon organ
 Hb Hemoglobin
 HbO₂ Hemoglobin
 HDL High-density lipoprotein cholesterol
 HIV Human immunosuppression virus
 HR Heart rate
 HR_{max} Maximal heart rate
 HR_R Heart rate reserve
 HR_{rec} Heart rate recovery
 HRV Heart rate variability
 H Height
 ICD Isocitrate dehydrogenase
 IC₅₀ Isovolumetric contraction period
 IR₅₀ Isovolumetric relaxation period
 LA Lactic acid/glycolytic system
 LB Lean body mass
 LB_{BP} Low back pain
 LDL Low-density lipoprotein cholesterol
 LS Long, slow distance
 LT Lactate threshold
 M Mass of the body in the air
 M_w Mass of the body underwater
 M Myosin
 MAOD Maximal accumulated oxygen deficit
 MAP Mean arterial pressure
 MCT1 Mitochondrial and intracellular monocarboxylate lactate transporter
 MCT4 Mitochondrial monocarboxylate lactate transporter
 ME Metabolic equivalent
 MLSS Maximal lactate steady state
 MP Mean power
 MVIC Maximal voluntary contraction
 MVV Maximal voluntary ventilation
 NAD Nicotinamide adenine dinucleotide
 NE Norepinephrine
 NK Natural killer
 NKCA Natural killer cell activity
 NM Neuromuscular junction

NM neuromuscular spindle
 NT neurotransmitter
 O oxygen
 OBL net of blood lactate accumulation
 OI osteogenic index
 OR oxidative phosphorylation
 OR overreaching
 OT overtraining syndrome
 P pressure in the alveoli
 PA partial pressure of carbon dioxide in the alveoli
 PA partial pressure of oxygen in the alveoli
 PB barometric pressure
 P partial pressure of a gas
 Pi inorganic phosphate
 P pressure
 PA partial pressure of carbon dioxide in arterial blood
 PA partial pressure of oxygen in arterial blood
 PC phosphocreatine
 PC partial pressure of carbon dioxide
 PF phosphofructokinase
 pH hydrogen ion concentration
 PN partial pressure of nitrogen
 PN proprioceptive neuromuscular facilitation
 PN peripheral nervous system
 PO partial pressure of oxygen
 PP peak power
 PET peripheral quantitative computed tomography
 PR protein
 PV partial pressure of oxygen in venous blood
 PV partial pressure of carbon dioxide in venous blood
 Q cardiac output
 R rate of appearance
 R rate of disappearance
 R resistance
 RB red blood cells
 RD recommended daily allowance
 RE relative energy deficiency in sport
 RE respiratory exchange ratio
 RH relative humidity
 RH resting heart rate
 RM repetition maximum
 RM resting metabolic rate
 RM respiratory muscle training
 RM range of motion

RPE Rating of perceived exertion
 RPP Rate pressure product
 RQ Respiratory quotient
 RV Residual volume
 SaO_2 Percent saturation of arterial blood with oxygen
 SpO_2 Percent saturation of blood with oxygen
 SvO_2 Percent saturation of venous blood with oxygen
 SBP Systolic blood pressure
 SO Slow-twitch, oxidative muscle fibers
 SR Sarcoplasmic reticulum
 SS Stretch shortening cycle
 ST Slow-twitch muscle fibers
 STD Standard temperature and pressure, dry air
 SV Stroke volume
 T temperature
 T_{amb} Ambient temperature
 TC Total cholesterol
 T_{co} Core temperature
 TEF Thermic effect of feeding
 TEM Thermic effect of a meal
 $Target HR$ Target exercise heart rate
 $\dot{V}O_2$ target exercise oxygen consumption
 TEX
 TG Triglycerides
 TL Total lung capacity
 TPR Total peripheral resistance
 T_{re} Rectal temperature
 T_{sk} Skin temperature
 T_{ty} Tympanic temperature
 $URTI$ Upper respiratory tract infection
 \dot{V}_A alveolar ventilation
 V_D volume of dead space
 \dot{V}_E volume of expired air
 V_G volume of a gas
 \dot{V}_I volume of inspired air
 V_T tidal volume
 \dot{V} volume per unit of time
 V volume
 VAT Visceral abdominal tissue
 VC vital capacity

$\dot{V}CO_2$ volume of carbon dioxide produced

VEP ventricular ejection period

VPF ventricular filling period

VLDL very-low-density lipoprotein

$\dot{V}O_2$ volume of oxygen consumed

$\dot{V}O_{2max}$ maximal volume of oxygen consumed

$\dot{V}O_{2peak}$ peak volume of oxygen consumed

$\dot{V}O_{2R}$ oxygen consumption reserve

VT ventilatory threshold

$\dot{V}O_{2max}$ velocity at maximal oxygen consumption

W_{waist} waist-to-hip ratio

WBC white blood cells

WT weight

Icon Identification Guide

Short-term, light to moderate submaximal aerobic exercise



Long-term, moderate to heavy submaximal aerobic exercise



Incremental aerobic to maximum exercise



Static exercise



Dynamic resistance exercise



Very short-term, high-intensity anaerobic exercise



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OBJECTIVES

After studying the chapter, you should be able to:

- Describe what exercise physiology is and discuss why you need to study it.
 - Identify the organizational structure of this text.
 - Differentiate between exercise responses and training adaptations.
 - List and explain the six categories of exercise whose responses are discussed throughout this book.
 - List and explain the factors involved in interpreting an exercise response.
 - Describe the graphic patterns that physiological variables may exhibit in response to different categories of exercise and as a result of training adaptations.
 - List and explain the training principles.
 - Describe the differences and similarities between health-related and sport-specific physical fitness.
 - Define and explain periodization.
 - Define detraining.
 - Relate exercise and exercise training to Selye's theory of stress.
-

Introduction

In the science fiction movie *Fantastic Voyage* (CBS/Fox), a military medical team is miniaturized in a nuclear-powered submarine and injected through a hypodermic needle into the carotid artery. Anticipating an easy float into the brain, where they plan to remove a blood clot by laser beam, they are both awed by what they see and imperiled by what befalls them. They see erythrocytes turning from an iridescent blue to vivid red as oxygen bubbles replace carbon dioxide; nerve impulses appear as bright flashes of light; and when their sub loses air pressure, all they need to do is tap into an alveolus. Not all of their encounters are so benign, however. They are sucked into a whirlpool caused by an abnormal fistula between the carotid artery and jugular vein. They have to get the outside team to stop the heart so that they will not be crushed by its contraction. They are jostled about by the conduction of sound waves in the inner ear. They are attacked by antibodies. And finally, their submarine is destroyed by a white blood cell—they are, after all, foreign bodies to the natural defense system. Of course, in the end, the “good guys” on the team escape through a tear duct, and all is well.

Although the journey you are about to take through the human body will not be quite so literal, it will be just as incredible and fascinating, for it goes beyond the basics of anatomy and physiology into the realm of the moving human. The body is capable of great feats, whose limits and full benefits in terms of exercise and sport are still unknown. The health benefits of physical activity and physical fitness are becoming more obvious and leading to many exciting career opportunities.

Consider these events and changes, listed below, all of which point out how dramatically our understanding of performance and health have changed in a relative short period of time.

- President Dwight D. Eisenhower suffered a heart attack on September 23, 1955. At that time, the normal medical treatment was 6 weeks of bed rest and a lifetime of curtailed activity ([Hellerstein, 1979](#)). Eisenhower's rehabilitation, including a return to golf, was, if not revolutionary, certainly progressive. Today, cardiac

patients are mobilized within days and frequently train for and safely run marathons.

- The 4-minute mile was considered an unbreakable limit until May 6, 1954, when Roger Bannister ran the mile in 3:59.4. Hundreds of runners (including some high school boys) have since accomplished that feat. The men's world record for the mile, which was set in 1999, is 3:43.13. The women's mile record, of 4:12.33, set in 2019, is approaching the old 4-minute "barrier."
- The 800-m run was banned from the Olympics from 1928 to 1964 for women because females were considered to be "too weak and delicate" to run such a "long" distance. In the 1950s when the 800-m run was reintroduced for women in Europe, ambulances were stationed at the finish line, motors running, to carry off the casualties (Ulyot, 1976). In 1963, the women's world marathon record (then not an Olympic sport for women) was 3:37:07, a time now commonly achieved by females not considered to be elite athletes. The women's world best "mixed gender" time (meaning achieved in a race in which men also competed) was set in 2019 at 2:14:04, an improvement of 1:23:03 (38.3%).
- In 1954, Kraus and Hirschland published a report indicating that American children were less fit than European children (Kraus and Hirschland, 1954). These results started the physical fitness movement. At that time, being fit was defined as being able to pass the Kraus-Weber test of minimal muscular fitness, which consisted of one each of the following: bent-leg situp; straight-leg sit-up; standing toe touch; double-leg lift, prone; double-leg lift, supine; and trunk extension, prone. Today (as is discussed in detail later in this chapter), physical fitness is more broadly defined in terms of both physiology and specificity (health related and sport related), and its importance for individuals of all ages is widely recognized.
- The American Heart Association first acknowledged physical inactivity as a risk factor for cardiovascular disease in a 1992 publication (Fletcher et al., 1992) and mounting research led to the 1996 Surgeon General Report (US Department of Health and Human Services, 1996),

which promoted physical activity as a means of improving life expectancy, quality of life, and overall health. In 2008, the United States Department of Health and Human Services released Physical Activity Guidelines for Americans ([US Department of Health and Human Services, 2008](#)).

- In 2007, the Exercise is Medicine (EIM) campaign was developed by the American Medical Association (AMA) and American College of Sports Medicine (ACSM) with the purpose to “make physical activity assessment and promotion a standard in clinical care, connecting health care with evidence-based physical activity resources for people everywhere and of all abilities ([American College of Sports Medicine, 2020](#)).” A key part of this initiative was to establish Physical Activity as a Vital Sign (PAVS) in which physical activity was added to health history forms and then translated to electronic medical records. This provided doctors with the information to prescribe exercise for their patients and connect them to resources such as physical trainers and other health professionals.
- On October 12, 2019, Kenyan distance runner Eliud Kipchoge ran a 1:59:40 marathon to achieve the first ever sub-2-hour marathon. This was not technically a world record as it was a nonsanctioned event that allowed multiple pacers and fueling strategies; however, the pace of less than 4 minutes and 35 seconds per mile for 26 miles straight shows the power of the human body.

These changes and a multitude of others that we readily accept as normal have come about as a combined result of formal medical and scientific research and informal experimentation by individuals with the curiosity and courage to try new things. The rapidly changing knowledge about the relationship between physical activity/physical fitness and health/performance means there is a great deal of information to cover in an exercise physiology textbook. It also means there are more professional opportunities than ever before for properly educated students to find fulfilling careers in this exciting area of human health.

What Is Exercise Physiology and Why Study It?

The events and changes described above exemplify concerns in the broad area of exercise physiology, that is, athletic performance, physical fitness, health, and rehabilitation. **Exercise physiology** can be defined as both a basic and an applied science that describes, explains, and uses the body's response to exercise and adaptation to exercise training to maximize human physical potential.

Exercise Physiology A basic and an applied science that describes, explains, and uses the body's response to exercise and adaptation to exercise training to maximize human physical potential.

No single course or textbook, of course, can provide all the information a prospective professional will need. However, knowledge of exercise physiology and an appreciation for practice based on research findings help set professionals in the field apart from mere practitioners. It is one thing to be able to lead yoga routines. It is another to be able to design routines based on predictable short- and long-term responses of given class members, to evaluate those responses, and then to modify the sessions as needed. To become respected professionals in fields related to exercise science and physical education, students need to learn exercise physiology in order to:

1. Understand how the basic physiological functioning of the human body is modified by various types of exercise as well as the mechanisms causing these changes. Unless one knows what responses are normal, one cannot recognize an abnormal response or adjust to it.
2. Understand how the basic physiological functioning of the human body is modified by various training programs and the mechanisms responsible for these changes. Adaptations will be specific to the training program used.

3. Provide quality fitness programming and physical education programs in schools that stimulate children and adolescents both physically and intellectually. To become lifelong exercisers, individuals need to understand how physical activity can benefit them, why they take physical fitness tests, and what to do with fitness test results.
4. Apply the results of scientific research to maximize health, rehabilitation, and/or athletic performance in a variety of subpopulations.
5. Respond accurately to questions and advertising claims, as well as recognize myths and misconceptions regarding exercise. Good advice should be based on scientific evidence.

Overview of the Text

Just as the fitness participant, athlete (**Figure 1.1**), or even musician warms up before working out, competing, or performing, this chapter is intended to provide you, the learner, with an essential warm-up for the rest of the text. That is, it provides the basic information that will prepare you to successfully understand what follows in the text and accomplish the goals stated above. To address the way that exercise interacts with the entire body, the textbook is divided into four units: metabolic system, cardiovascular-respiratory system, neuromuscular-skeletal system, and neuroendocrine-immune system. While the units are independent and may be covered in any order, to facilitate learning, each unit follows a consistent format:



Figure 1.1 Warming Up in Preparation for Performance.

1. Basic information

- a. Anatomical structures
- b. Physiological function
- c. Laboratory techniques and variables typically measured

2. Exercise responses

3. Training

- a. Application of the training principles
- b. Adaptations to training

4. Special applications, problems, and considerations

Each unit first deals with basic anatomical structures and physiological functions necessary to understand the material that follows. Then, each unit describes the acute responses to exercise. Following are specific applications of the training principles and discussion of the typical adaptations that occur when the training principles are applied correctly. Finally, each unit ends with one or more special application topics, such as thermal concerns, weight control/body composition, and osteoporosis. This integrated approach demonstrates the relevance of applying basic

information.

More exercise physiology research has been done on college-age males and elite male athletes than on any other portion of the population. Nonetheless, wherever possible, we provide information about both sexes as well as children and adolescents at one end of the age spectrum and older adults at the other, throughout the unit.

Each unit is independent of the other three, although the body obviously functions as a whole. Your course, therefore, may sequence these units of study in a different order other than just going from [Chapter 1](#) to [Chapter 22](#). After this first chapter, your instructor may start with any unit and then move in any order through the other three. This concept is represented by the circle and cross arrows in **Figure 1.2**.

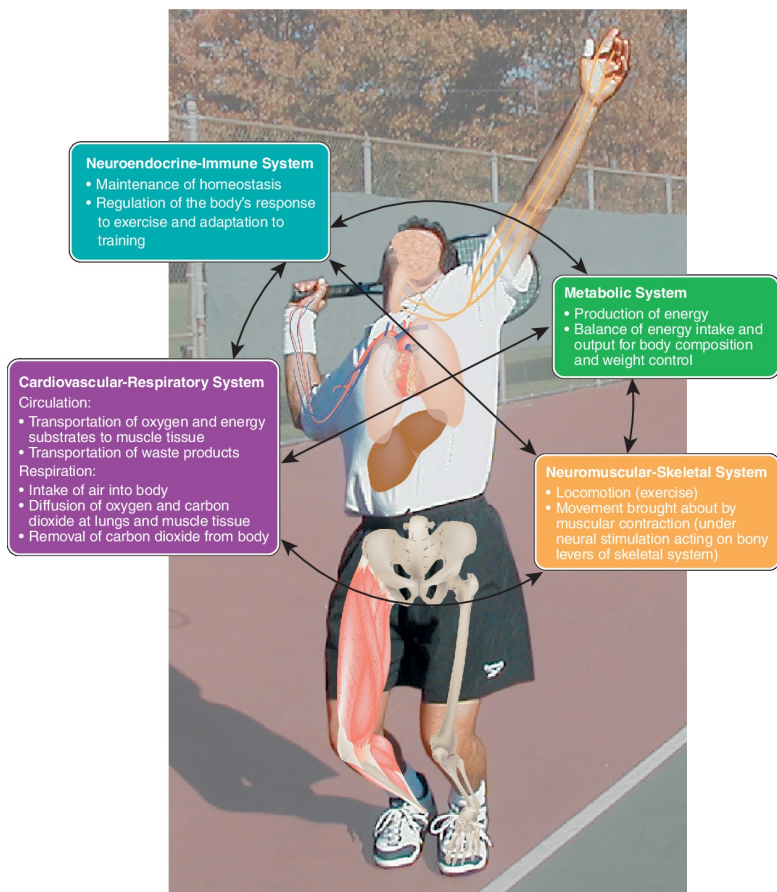


Figure 1.2 Schematic Representation of Text Organization.

Figure 1.2 also illustrates two other important points: (1) all of the systems respond to exercise in an integrated fashion and (2) the responses of the systems are interdependent. The metabolic system produces cellular energy in the form of adenosine triphosphate (ATP). ATP is used for muscular contraction. For the cells (including muscle cells) to produce ATP, they must be supplied with oxygen and fuel (foodstuffs). The respiratory system brings oxygen into the body via the lungs, and the cardiovascular system distributes oxygen and nutrients to the cells of the body via the blood pumped by the heart through the blood vessels. During exercise, all these functions must increase.

The neuroendocrine-immune system regulates and integrates both resting and exercise body functions.

Each unit is divided into multiple chapters depending on the amount and depth of the material. Each chapter begins with a list of learning objectives that present an overall picture of chapter content and help you understand what you should learn. Definitions are highlighted and boxed as they are introduced. Each chapter ends with a summary and review questions. Appearing throughout the text are Focus on Research and Focus on Application boxes, which present four types of research studies:

1. Analytical—an evaluation of available information in a review
2. Descriptive—a presentation of some variable (such as heart rate or blood lactate) or population (such as children or highly trained endurance athletes)
3. Experimental—a design in which treatments have been manipulated to determine their effects on selected variables
4. Quasi-experimental—designs such as used in epidemiology that study the frequency, distribution, and risk of disease among population subgroups in realworld settings

Focus on Research boxes present classic, illustrative, or cutting-edge research findings. Focus on Application boxes show how research may be used in practical contexts. Some of each type of focus box have been designated as Clinically Relevant. Clinically Relevant boxes present information, situations, or case studies related to clinical experiences students of exercise physiology often have. Research studies provide the scientific foundation of exercise physiology. While we have used many research articles for each chapter, and while you may feel that your textbook is very detailed, the truth is that each section only contains a small percentage of available research. In order to help you recognize the depth of science supporting this text, and to help you become a stronger reader of research, special features will prompt you to do relevant Literature Searches to explore areas of interest.

An additional feature is the Check Your Comprehension box.

The Check Your Comprehension boxes are problems for you to complete. Answers to these problems are presented in [Appendix C](#). When appropriate, calculations are worked out in examples. The appendices and endpapers provide supplemental information. For example, [Appendix A](#) contains a listing of the basic physical quantities, units of measurement, and conversions within the *Système International d'Unités* (SI or metric system of measurement commonly used in scientific work) and between the metric and English measurement systems. In the front of the book, you will find a list of the symbols and abbreviations used throughout the book, along with their full names. You may need to refer to these appendices/endpapers frequently if these symbols and measurement units are new to you.

Exercise physiology is a dynamic area of study with many practical implications. Over the next few months, you will gain an appreciation for the tremendous range in which the human body can function. At the same time, you will become better prepared as a professional to carry out your responsibilities in your particular chosen field. Along the way, you will probably also learn things about yourself. Enjoy the voyage.

The Exercise Response

Let's begin with some definitions and concepts required for understanding all the units to come. Many of these terms are common and the meaning may seem obvious at first, but knowing a precise definition for fundamental terms will make the reading of literature and studying of exercise physiology much easier. **Exercise** is a single acute bout of bodily exertion or muscular activity that requires an expenditure of energy above resting level and that in most, but not all, cases results in voluntary movement. Exercise sessions are typically planned and structured to improve or maintain one or more components of physical fitness. The term *physical activity*, in contrast, generally connotes movement in which the goal (often to sustain daily living or recreation) is different from that of exercise but which also requires the expenditure of energy and often provides health benefits. For example, walking to school or work is physical

activity, while walking around a track at a predetermined heart rate is exercise. Exercise is sometimes considered a subset of physical activity with a more specific focus (Caspersen et al., 1985). From a physiological standpoint, both involve the process of muscle action/energy expenditure and bring about changes (acute and chronic). Therefore, the terms exercise and physical activity are often used interchangeably in this textbook. Where the amount of exercise can actually be measured, the terms workload or work rate may be used as well.

Exercise A single acute bout of bodily exertion or muscular activity that requires an expenditure of energy above resting level and that in most, but not all, cases results in voluntary movement.

Homeostasis is the state of dynamic equilibrium (balance) of the body's internal environment. Exercise disrupts homeostasis, causing changes that represent the body's response to exercise. An **exercise response** is the pattern of change in physiological variables during a single acute bout of physical exertion. A physiological *variable* is any measurable bodily function that changes or *varies* under different circumstances. For example, heart rate is a variable with which you are undoubtedly already familiar. You probably also know that heart rate increases during exercise. However, to state simply that heart rate increases during exercise do not describe the full pattern of the response. For example, the heart rate response to a 400-m sprint is different from the heart rate response to a 50-mi bike ride. To fully understand the response of heart rate or any other variable, we need more information about the exercise itself. Three factors are considered when determining the acute response to exercise:

Homeostasis The state of dynamic equilibrium (balance) of the internal environment of the body.

Exercise Response The change in physiological variables during a single acute bout of physical exertion.

1. The exercise modality (or mode)
2. The exercise intensity
3. The exercise duration

Exercise Modality

Exercise modality (or **mode**) means the type of activity or the particular sport. For example, rowing has a very different effect on the cardiovascular-respiratory system than does football. Modalities are often classified by the type of energy demand (aerobic or anaerobic), the major muscle action (continuous and rhythmical, dynamic resistance, or static), or a combination of the energy system and muscle action. Walking, cycling, and swimming are examples of continuous, rhythmical aerobic activities; jumping, sprinting, and weight lifting are anaerobic and/or dynamic resistance activities. To determine the effects of exercise on a particular variable, you must first know what type of exercise is being performed.

Exercise Modality or Mode The type of activity or sport, usually classified by energy demand or type of muscle action.

Exercise Intensity

Exercise intensity is most easily described as maximal or submaximal. **Maximal (max) exercise** is straightforward; it simply refers to the highest intensity, greatest load, or longest duration an individual is capable of doing. Motivation plays a large part in the achievement of maximal levels of exercise. Most maximal values are reached at the end point of an *incremental exercise test to maximum*; that is, the exercise task begins at a level the individual is comfortable with and gradually increases until he or she can do no more. The values of the physiological variables measured at this time are labeled as “max”; for example, maximum heart rate is symbolized as HRmax.

Maximal (max) Exercise The highest intensity, greatest load,

or longest duration exercise of which an individual is capable.

Submaximal exercise may be described in one of two ways. The first involves a *set load*, which is a load that is known or is assumed to be below an individual's maximum. This load may be established by some physiological variable, such as working at a specific heart rate (perhaps $150 \text{ b}\cdot\text{min}^{-1}$), at a specific work rate (e.g., $600 \text{ kgm}\cdot\text{min}^{-1}$ on a cycle ergometer), or for a given distance (perhaps a 1-mi run). Such a load is called an **absolute submaximal workload**. If an absolute workload is used, and the individuals being tested vary in fitness, then some individuals will be challenged more than others. Generally, those who are more fit in terms of the component being tested will be less challenged and so will score better than those who are less fit and more challenged. For example, suppose the exercise task is to lift 80 lb in a bench press as many times as possible, as in the YMCA bench press endurance test for males. As illustrated in **Table 1.1**, if the individuals tested were able to lift a maximum of 160, 100, and 80 lb once, respectively, it would be anticipated that the first individual could do more repetitions of the 80-lb lift than anyone else. Similarly, the second individual would be expected to do more repetitions than the third, and the third individual would be expected to do only one repetition. In this case, the load is not submaximal for all the individuals, because Terry can lift the weight only one time (making it a maximal lift for Terry). Nonetheless, the use of an absolute load allows for the ranking of individuals based on the results of a single exercise test and is, therefore, often used in physical fitness screenings or tests.

Absolute Submaximal Workload A set exercise load performed at any intensity from just above resting to just below maximum.

TABLE 1.1 Absolute and Relative Submaximal Workloads

| | Absolute Workload | | Relative Workload | |
|-------|-------------------|----------------------------------|---------------------|--------------------------------|
| | Maximal Lift | No. of Times 80 lb can be Lifted | 75% of Maximal Lift | No. of Times 75% can be Lifted |
| Jose | 160 | 12 | 120 | 10 |
| Pat | 100 | 6 | 75 | 10 |
| Terry | 80 | 1 | 60 | 10 |

The second way to describe submaximal exercise is as a percentage of an individual's maximum. A work load may be set at a percentage of the person's maximal heart rate, maximal ability to use oxygen, or maximal workload. This value is called a **relative submaximal workload** because it is prorated or relative to each individual. All individuals are, therefore, expected to be equally challenged by the same percentage of their maximal task. This should allow the same amount of time or number of repetitions to be completed by most, if not all, individuals. For example, for the individuals described in **Table 1.1**, suppose that the task now is to lift 75% of each one's maximal load as many times as possible. The individuals will be lifting 120, 75, and 60 lb, respectively. If all three are equally motivated, they should all be able to perform the same total number of repetitions. Relative workloads are occasionally used in physical fitness testing. They are more frequently used to describe exercises that are light, moderate, or heavy in intensity or to prescribe exercise guidelines.

Relative Submaximal Workload A workload above resting but below maximum that is prorated to each individual; typically set as some percentage of maximum.

There is no universal agreement about what exactly constitutes light, moderate, or heavy intensity. In general, this book uses the following classifications:

1. Low or light: $\leq 54\%$ of maximum
2. Moderate: 55–69% of maximum
3. Hard or heavy: 70–89% of maximum
4. Very hard or very heavy: 90–99% of maximum
5. Maximal: 100% of maximum
6. Supramaximal: $>100\%$ of maximum

Maximum is defined variously in terms of workload or work rate, heart rate, oxygen consumption, weight lifted for a specific number of repetitions, or force exerted in a voluntary contraction. Specific studies may use percentages and definitions of maximum that vary slightly.

Exercise Duration

Exercise duration is simply a description of the length of time the muscular action continues. Duration may be as short as 1–3 seconds for an explosive action, such as a jump, or as long as 12 hours for a full triathlon (3.2-km [2-mi] swim, 160-km [100-mi] bicycle ride, and 42.2-km [26.2-mi] run). In general, the shorter the duration, the higher the intensity that can be used. Conversely, the longer the duration, the lower the intensity that can be sustained. Thus, the amount of homeostatic disruption depends on both the duration and intensity of the exercise.

Exercise Categories

There are a nearly endless number of possible exercise categories. Recognizing some major categories of exercise, and in order to support learning, this textbook combines the descriptors of exercise modality, intensity, and duration into six primary categories of exercise. Where sufficient information is available, the exercise response patterns for each are described and discussed:

1. *Short-term, light to moderate submaximal aerobic exercise.* Exercises of this type are rhythmical and continuous in nature and utilize aerobic energy. They are performed at a constant workload for 10–15 minutes at approximately 30–69% of maximal work capacity.
2. *Long-term, moderate to heavy submaximal aerobic exercise.* Exercises in this category also utilize rhythmical and continuous muscle action. Although predominantly aerobic, anaerobic energy utilization may be involved. The duration is generally between 30 minutes and 4 hours at constant workload intensities ranging from 55 to 89% of maximum.

3. *Incremental aerobic exercise to maximum.* Incremental exercises start at light loads and continue by a predetermined sequence of progressively increasing workloads to an intensity that the exerciser cannot sustain or increase further. This point becomes the maximum (100%). The early stages are generally light and aerobic, but as the exercise bout continues, anaerobic energy involvement becomes significant. Each workload/work rate is called a stage, and each stage may last from 1 to 10 minutes, although 3 minutes is most common. Incremental exercise bouts typically last between 5 and 20 minutes for the total duration.
4. *Static exercise.* Static exercises involve muscle contractions that produce an increase in muscle tension and energy expenditure but do not result in meaningful movement. Static contractions are measured as some percentage of the muscle's **maximal voluntary contraction (MVC)**, the maximal force that the muscle can exert. The intent is for the workload to remain constant, but fatigue sometimes makes that impossible. The duration is inversely related to the percentage of maximal voluntary contraction (%MVC) that is being held but generally ranges from 2 to 10 minutes.

Maximal Voluntary Contraction (MVC) The maximal force that the muscle can exert.

5. *Dynamic resistance exercise.* These exercises utilize muscle contractions that exert sufficient force to overcome the presented resistance, so that movement occurs, as in weight lifting. Energy is supplied by both aerobic and anaerobic processes, but anaerobic is dominant. The workload is constant and is based on some percentage of the maximal weight the individual can lift (**1-RM**) or a resistance that can be lifted for a specified number of times. The number of repetitions, not time, is the measure of duration.

1-RM The maximal weight that an individual can lift once during a dynamic resistance exercise.

6. *Very short-term, high-intensity anaerobic exercise.* Activities of this type last from a few seconds to approximately 3 minutes. They depend on high-power anaerobic energy and are often supramaximal.

Complete the [Check Your Comprehension 1](#) box.

CHECK YOUR COMPREHENSION 1

Describe each of the following activities using the terms of the six exercise response categories.













1. A male cheerleader holds a female cheerleader overhead.
2. A body builder poses.
3. A new mother pushes her baby in a stroller in the park for 20 minutes.
4. A freshman in high school takes the FitnessGram® PACER (Progressive Aerobic Cardiovascular Endurance Run) test in physical education class.
5. An adult male completes a mini triathlon in 2:35.
6. A basketball player executes a fast break ending with a slam dunk.
7. A volleyball player performs two sets of six squats.
8. A cyclist completes a 25-mi time trial in 50:30.6.
9. An exercise physiology student completes a graded exercise test on a cycle ergometer with 3-minute stages and + 50 kgm·min⁻¹ per stage to determine **VO₂max**.
10. A barrel racer warms up her horse for 15 minutes prior to competition.
11. A middle-aged individual performs 18 repetitions in the YMCA bench press endurance test.
12. A college athlete participates in a 400-m track race.

Check your answers in [Appendix C](#).

Exercise Response Patterns

Throughout this book, the exercise response patterns for the six categories of exercise are described in text and depicted graphically. For ease of recognition, consistent background colors and icons represent each category of exercise (**Table 1.2**). **Figure 1.3** presents six of the most frequent graphic patterns resulting from a constant workload/work rate, that is, all of the exercise categories except incremental exercise to maximum and very short-term, high-intensity anaerobic exercise. Frequent patterns for incremental exercise to maximum are depicted in **Figure 1.4**. The verbal descriptors used throughout the book are included on these graphs and in the following paragraphs. Note that the y-axis can be any variable that is measured with its appropriate unit of measurement. Examples are heart rate ($\text{b}\cdot\text{min}^{-1}$), blood pressure (mmHg), and oxygen consumption ($\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$). Only specific graphic patterns are applicable to any given variable. These combinations of pattern and variable are described in the exercise response sections in each unit. Some students feel like there are many graphs in the book. That may be true, but remember, the graphic responses shown depict the typical response to essentially every type of exercise (based on our six categories) for the most important physiological variables. If you know these graphs, you will “know” how the body responds to exercise. For each exercise response, the baseline, or starting point against which the changes are compared, is the variable’s resting value. Your goal here is to become familiar with the graphic patterns and the terminology used to describe each.

TABLE 1.2 Color and Icon Interpretation for Exercise Response Patterns

| Exercise Category | Color | Icon |
|--|---|---|
| Short-term, light to moderate submaximal aerobic |  |  |
| Long-term, moderate to heavy submaximal aerobic |  |  |
| Incremental aerobic to maximum |  |  |
| Static |  |  |
| Dynamic resistance |  |  |
| Very short-term, high-intensity anaerobic |  |  |

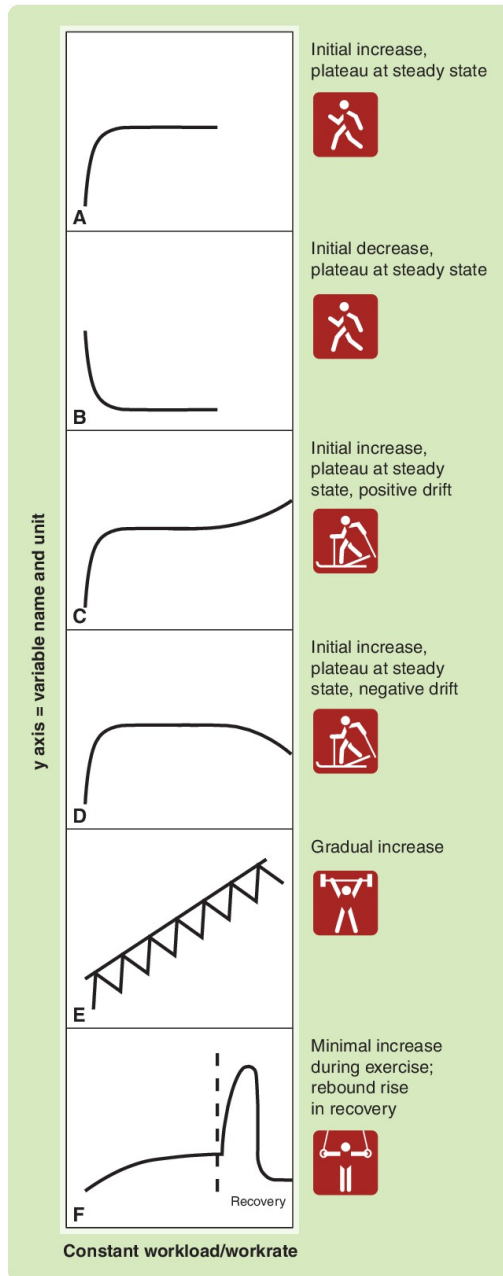


Figure 1.3 Graphic Patterns and Verbal Descriptors for Constant Workload/Work Rate Exercise Responses.

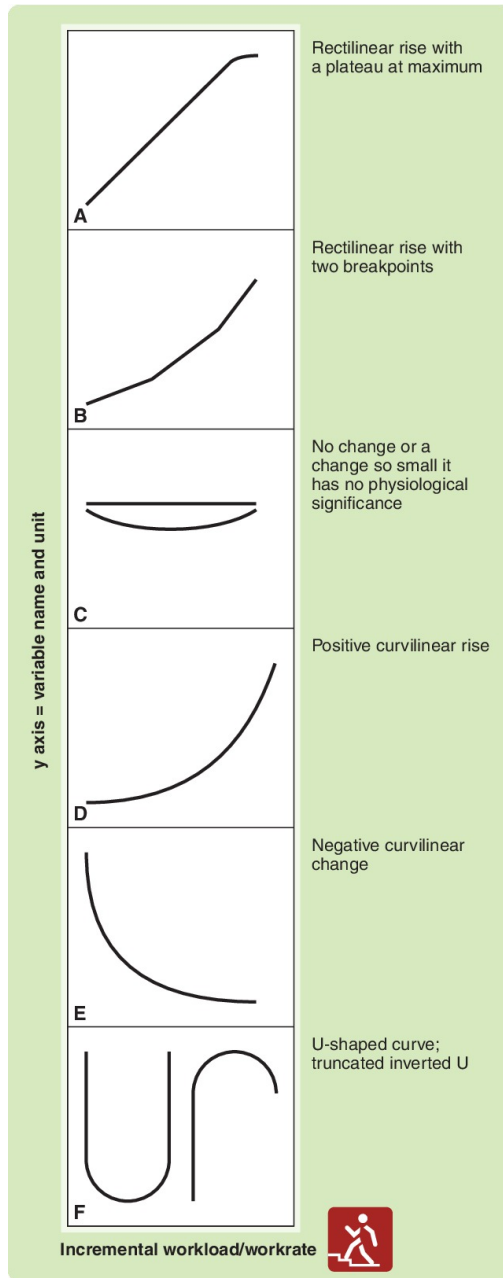


Figure 1.4 Graphic Patterns and Verbal Descriptors for Incremental Workload/Work Rate Exercise Responses.

The patterns showing an initial increase or decrease with a plateau at steady state (**Figure 1.3A and B**) are the most common responses to short-term, light to moderate submaximal aerobic exercise. Think about HR increasing during a light jog; it increases above resting when you start jogging, but levels off (plateaus) as you continue to run. Patterns that include a drift seen as the gradual curvilinear increase or decrease from a plateau despite no change in the external workload (**Figure 1.3C and D**), typically result from long-term, moderate to heavy submaximal aerobic exercise. As an example, imagine doing a half marathon; even if you try to run at a consistent pace, your heart rate is likely to drift higher toward the latter half of the run. Another form of gradual increase, despite no change in the external workload (**Figure 1.3E**), is frequently seen during dynamic resistance exercise as a sawtooth pattern resulting from the sequential lifting and lowering of the weight. Finally, some categories of exercise may show a smooth, gradual increase (the straight rising line of **Figure 1.3E**). Minimal change during exercise with a rebound rise in recovery is almost exclusively a static exercise response (**Figure 1.3F**).

As the title of **Figure 1.4** indicates, all of these patterns of response routinely result from incremental exercise to maximum. Panel **1.4F** shows two versions of the U-shaped pattern. You may see either a complete or truncated (shortened) U, either upright or inverted. No specific patterns are shown for very short-term, high-intensity anaerobic exercise, because these tend to be either abrupt rectilinear or curvilinear increases or decreases.

Exercise Response Interpretation

When interpreting the response of variables to any of the exercise categories, keep four factors in mind:

1. Characteristics of the exerciser
2. Appropriateness of the selected exercise
3. Accuracy of the selected exercise
4. Environmental and experimental conditions

Characteristics of the Exerciser

Certain characteristics of the exerciser can affect the magnitude of the exercise response. The basic pattern of the response is similar, but the magnitude of the response may vary with the individual's sex, age (child/adolescent, adult, older adult), and/or physiological status, such as health and training level. Where possible, these differences will be pointed out. See the Focus on Research Box for an example.

Appropriateness of the Selected Exercise

Exercise scientists use exercise tests to describe exercise responses or attributes of an individual. It is important that the exercise test used match the physiological system or physical fitness component that is being evaluated or the research question that is being asked. For example, you cannot determine cardiovascular endurance using dynamic resistance exercise. However, if the goal is to determine how selected cardiovascular variables respond to dynamic resistance exercise, then, obviously, that is the type of exercise that must be used.

The modality used within the exercise category should also match the intended outcome. For example, if the goal is to demonstrate changes in cardiovascular-respiratory fitness for individuals training on a stationary cycle, then an incremental aerobic exercise to maximum test should be conducted on a cycle ergometer, not a treadmill or other piece of equipment.

Accuracy of the Selected Exercise Test

The most accurate tests are called **criterion tests**. They represent a standard against which other tests are evaluated. Most criterion tests are **laboratory tests**—precise, direct measurements of physiological function that usually involve monitoring, collection, and analysis of expired air, blood, or electrical signals. Typically, these require expensive equipment and trained technicians. Not all laboratory tests, however, are criterion tests.

Criterion Test The most accurate tests for any given variable; the measurement standard against which other tests are

judged.

Laboratory Test Precise, direct measurement of physiological functions for the assessment of exercise responses or training adaptations; usually involves monitoring, collection, and analysis of expired air, blood, or electrical signals.

Field tests can be conducted almost anywhere, such as a school gymnasium, playing field, or health club. Field tests are often performance based and estimate the values measured by the criterion test. The mile run is a field test used to assess cardiovascular-respiratory fitness, which is more directly and accurately measured by the criterion test of maximal oxygen consumption ($\dot{V}O_2 \text{ max}$). Both laboratory and field tests are discussed in this text.

Field Test A performance-based test that can be conducted anywhere and that estimates the values measured by the criterion test.

Environmental and Experimental Conditions

Many physiological variables are affected by environmental conditions, most notably temperature, relative humidity (RH), and barometric pressure. Normal responses typically occur at neutral conditions ($\sim 20\text{--}29^\circ\text{C}$ [$68\text{--}84^\circ\text{F}$], 50% RH, and 630–760 mmHg, respectively). Likewise, when a response to exercise is described, it is assumed that the exerciser had adequate sleep, was not ill, had not recently eaten or exercised, and was not taking any prescription or nonprescription drugs or supplements that could affect the results. If any of these assumed conditions is not met, the expected exercise response might not occur.

FOCUS ON RESEARCH

The Effects of Age, Sex, and Physical Training on the Response to Exercise

Exercise professionals and exercise participants have long been interested in how personal characteristics influence the body's response to exercise. In this study, the authors investigated the effects of age, sex, and physical training on cardiovascular responses to exercise. They separated 110 healthy subjects into eight groups based on three variables: age (young [mid 20s] or old [mid 60s]), sex (male or female), and physical training (trained or untrained). The table below identifies the eight groups based on these three subject characteristics.

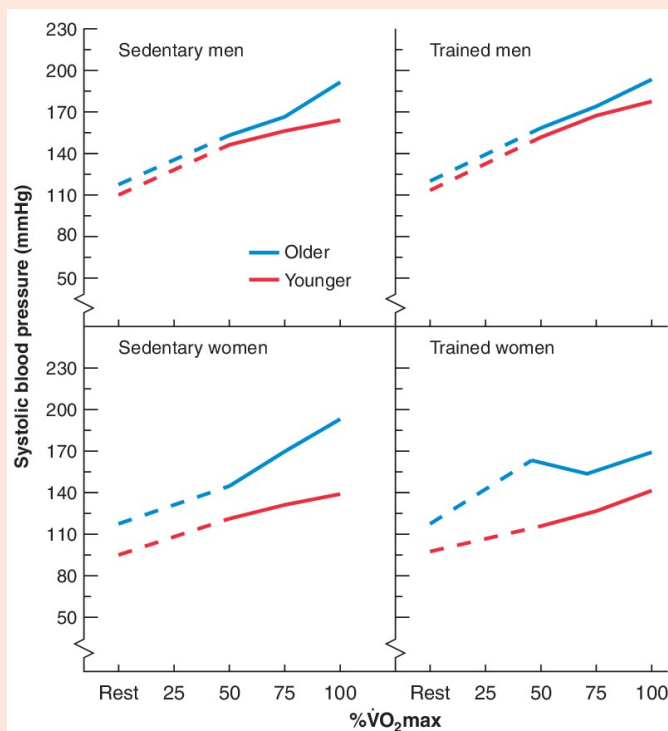
| | Males | Females |
|---------|----------------|----------------|
| Younger | Trained (TR) | Trained (TR) |
| | Untrained (UT) | Untrained (UT) |
| Older | Trained (TR) | Trained (TR) |
| | Untrained (UT) | Untrained (UT) |

Results of this study are shown in the figure at the right, which depicts for each group the systolic blood pressure responses to incremental treadmill tests to maximum. These data reveal the following:

1. Systolic blood pressure response to incremental exercise to maximum was significantly greater in older persons than in younger persons. This is true for males and females regardless of training status.
2. Maximal systolic blood pressure was significantly lower in trained females than in untrained females.

Although the authors investigated many variables, we describe only systolic blood pressure because the purpose here is only to demonstrate how characteristics of the

exerciser affect exercise response.



Source: Reprinted with permission from Ogawa, T., R. J. Spina, W. H. Martin, W. M. Kohrt, K. B. Schectman, J. O. Holloszy, & A. A. Ehsani: Effects of aging, sex, and physical training on cardiovascular responses to exercise. *Circulation*. 86(2):494–503 (1992). Copyright © 1992 by American Heart Association.

Training

Training is a consistent or chronic progression of exercise sessions designed to improve physiological function for better health or sport performance. The two main goals for exercise training are (1) health-related physical fitness and (2) sport-specific physical fitness (sometimes called athletic fitness or

sport-related physical fitness).

Training A consistent or chronic progression of exercise sessions designed to improve physiological function for better health or sport performance.

Health-Related versus Sport-Specific Physical Fitness

In this textbook, the phrase **health-related physical fitness** refers to that portion of physical fitness directed toward the prevention of or rehabilitation from disease, the development of a high level of functional capacity for the necessary and discretionary tasks of life, and the maintenance or enhancement of physiological functions in biological systems that are not involved in performance but are influenced by habitual activity. The individual's goal may be to participate minimally in an activity to achieve some health benefit before disease occurs. The goal may be to participate in a substantial amount of exercise to improve or maintain a high level of physical fitness. Or an injured or disabled individual's goal may be to participate in an activity to recover and/or attain the maximal function possible. All goals should include avoiding injury during the process.

Health-Related Physical Fitness That portion of physical fitness directed toward the prevention of or rehabilitation from disease, the development of a high level of functional capacity for the necessary and discretionary tasks of life, and the maintenance or enhancement of physiological functions in biological systems that are not involved in performance but are influenced by habitual activity.

Three components of health-related physical fitness are generally recognized: cardiovascular-respiratory endurance (aerobic power), body composition, and musculoskeletal fitness (muscular strength, muscular endurance, muscular power, and flexibility) ([American College of Sports Medicine, 2022](#); [Canadian](#)

Society for Exercise Physiology, 2004; Plowman and Meredith, 2013). As depicted in **Figure 1.5** (inner circle), these health-related components form the core of physical fitness. However, the Committee on Fitness Measures and Health Outcomes in Youth, tasked with recommending items “... for assessment of youth fitness components that are associated with health outcomes... suitable for use in a national survey of youth fitness” (Institute of Medicine, 2012, p. 3) modified the definition of musculoskeletal fitness to exclude flexibility and include muscular power. Although some have argued that flexibility should be de-emphasized because of modest health gains associated with flexibility and poor predictive ability relative to health or performance, others caution that there are benefits associated with flexibility and calls to de-emphasize it are premature (Kruse, 2020; Nuzzo, 2020). The role of flexibility as a component of health-related physical fitness has been debated for some time, and whether flexibility will be replaced in the core of health-related physical fitness remains to be seen (Plowman, 2014).

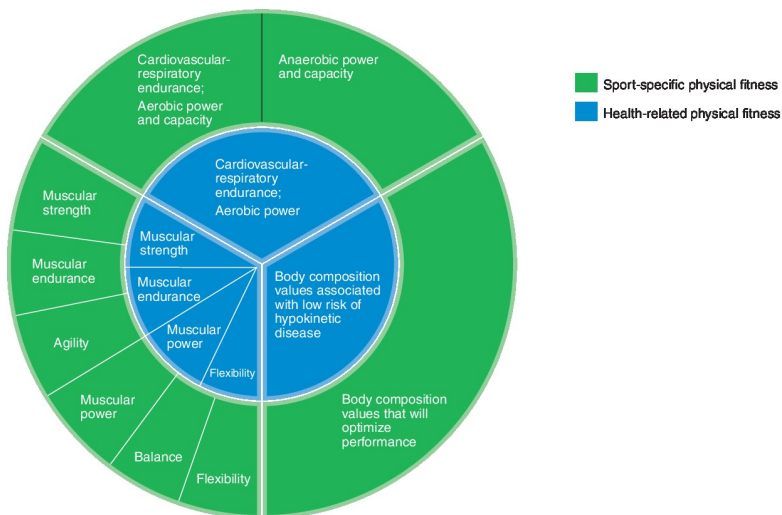


Figure 1.5 Physical Fitness.

Physical fitness consists of health-related physical fitness (*inner circle*) and sport-specific physical fitness (*outer circle*). Health-related physical fitness is composed of components

representing cardiovascular-respiratory endurance, metabolism, and musculoskeletal (muscular strength, muscular endurance, muscular power, and flexibility). Sport-specific physical fitness builds on health-related physical fitness and adds motor attributes (such as agility and balance) and anaerobic power and capacity, as needed.

The relationships between each of these fitness components and **hypokinetic disease** are described in appropriate units. Hypokinetic diseases are diseases caused by and/or associated with a lack of physical activity. Health-related physical fitness is important for everyone.

Hypokinetic Diseases Diseases caused by and/or associated with lack of physical activity.

Sport-specific physical fitness has a more narrow focus; it is that portion of physical fitness directed toward optimizing athletic performance. **Figure 1.5** shows that sport-specific (athletic) fitness (outer circle) expands from the core of health-related physical fitness. Higher levels of cardiovascular-respiratory endurance and anaerobic power and capacity are generally needed for successful performance. Body composition values may be more specific than health levels in order to optimize performance. The musculoskeletal fitness attributes of power, balance, and flexibility are frequently more specific in certain athletic performances than for health.

Sport-Specific Physical Fitness That portion of physical fitness directed toward optimizing athletic performance.

To determine the importance of each component of fitness and develop a sport-related fitness program, you first analyze the specific sport's physiological demands. Then the athlete is evaluated in terms of those requirements. These elements allow for a specifically designed, individualized program. This program

should:

- Work-specific musculature while achieving a balance between agonistic and antagonistic muscle groups
- Incorporate all motor fitness attributes that are needed
- Use the muscles in the biomechanical patterns of the sport
- Match the cardiovascular and metabolic energy requirements of the sport
- Attend realistically to body composition issues
- The demands of the sport will not change to accommodate the athlete. The athlete must be the one to meet the demands of the sport to be successful.

Putting all of these elements together, **physical fitness** may be defined as a physiological state of wellbeing that provides the foundation for the tasks of daily living, a degree of protection against hypokinetic disease, and a basis for participation in sport ([American Alliance for Health, 1988](#)). It is a product, the result of the process of doing physical activity/exercise.

Physical Fitness A physiological state of well-being that provides the foundation for the tasks of daily living, a degree of protection against hypokinetic disease, and a basis for participation in sport.

Dose-Response Relationships

Major questions in exercise physiology revolve around “how much exercise/activity is enough?” and “what is the relationship between specific amounts of exercise/activity or physical fitness levels and the benefits achieved?” To the exercise scientist, these are what are called dose-response relationship questions. A **dose-response relationship** describes how a change in one variable is associated with a corresponding change in another variable. In this context, the training dose refers to the characteristics of the training program, that is, the type, intensity, frequency, duration, and/or volume of the exercise program or physical activity undertaken by the individual or group. The response means the changes that occur when a specific volume or dose of exercise/

physical activity is performed. Thus, for physical activity and health, doseresponse describes the health-related changes obtained for the particular level of physical activity performed. For example, researchers may ask, does the number of days per week that a person walks affect measures of bone mineral density in middle-aged women? Likewise, for physical fitness and health, the dose-response describes the health-related changes that occur with experimentally documented changes or levels of fitness (Haskell, 2007). In this case, the question maybe is as follows: how does blood pressure differ among individuals with differing levels of cardiovascular fitness? These experimentally derived relationships can be graphed and are often called curves. Although it is clear, for example, that exercise/physical activity reduces the risk of many diseases and improves cardiovascular function, it is far less clear what the minimal dose of physical activity may be to acquire risk reduction or how much additional activity/fitness is needed to confer additional benefits. The shape of the dose-response curve may vary (large benefit for minimal increase, small benefit for large increase, etc.) depending upon the health benefit or physiological variable being measured, and the population that is being studied.

Dose-Response Relationship A description of how a change in one variable is associated with a corresponding change in another variable.

Two examples of dose-response curves are presented in **Figure 1.6**. Panel A is an actual curve and depicts an inverse or negative dose-response relationship. That is, the higher the weekly energy expenditure, the lower the relative risk of mortality. Panel B is an example where the curve is implied by the relative heights of the bars in the graph. In contrast to Panel A, this dose-response relationship is a direct or positive one. That is, the higher the amount of daily sitting time, the higher the pro-rated all-cause death rate. Note that the mortality rate varies between those who are active and those who are inactive. However, although lower, even within the active group (defined as participating in at least 30 minutes of moderate-intensity exercise 5 d·wk⁻¹), there is a strong association between sitting and risk of mortality.

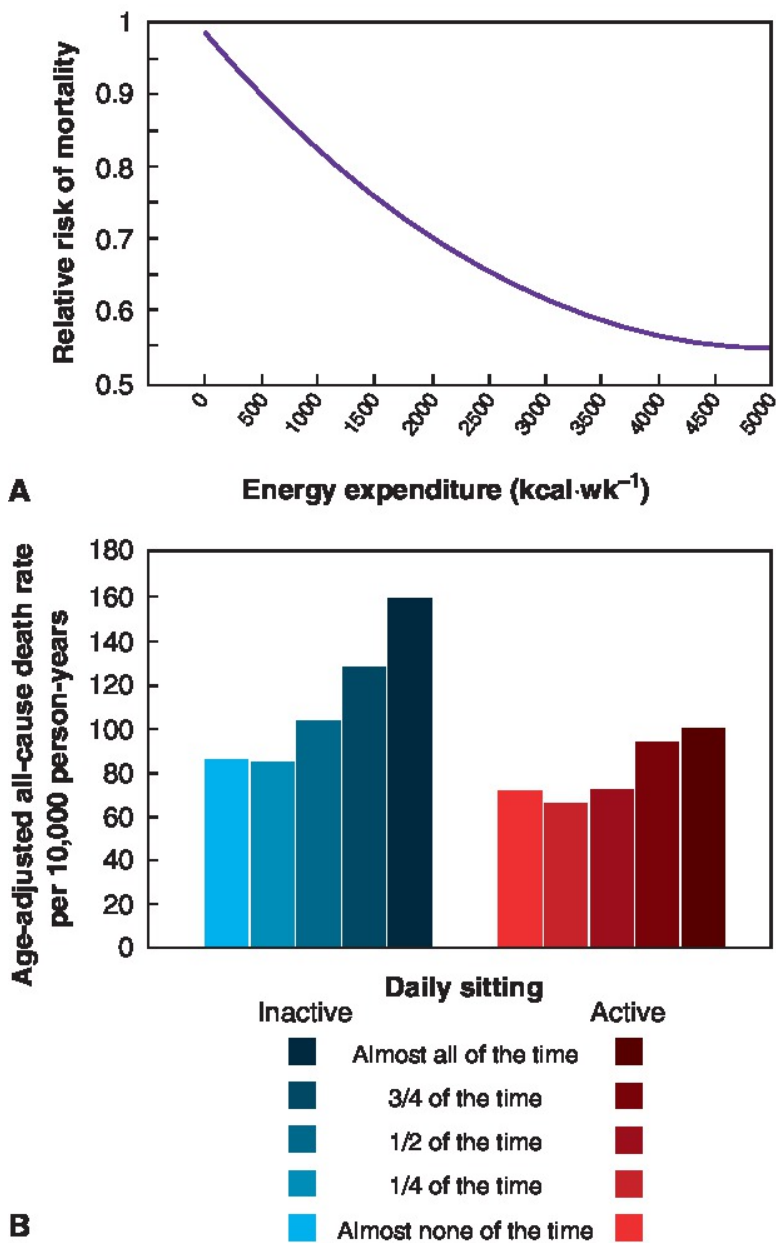


Figure 1.6 Data Showing Dose-Response Relationships.

Panel A shows the relative risk for all-cause mortality and its relationship to baseline physical activity energy expenditure in elderly males and females with coronary artery disease.

Panel B shows age-adjusted all-cause mortality and its relationship in both male and females to daily sitting amount. **Sources:** (A) Reprinted with permission from Janssen, I., & C. J. Jolliffe: Influence of physical activity on mortality in elderly with coronary artery disease. *Medicine & Science in Sports & Exercise*. 38(3):418–423 (2006). Copyright ©2006 The American College of Sports Medicine. (B) Modified with permission from Katzmarzyk, P. T., T. S. Church, C. L. Craig, & C. Bouchard: Sitting time and mortality from all causes, cardiovascular disease, and cancer. *Medicine & Science in Sports & Exercise*. 41(5):998–1005 (2009). Copyright ©2009 The American College of Sports Medicine.

Considerable research is currently being done to discern the shapes of the dose-response curves in order to clarify exercise/physical activity recommendations for various benefits and populations. Until more dose-response information is available, we can only cite variations for the upper and lower recommendations in order to acknowledge that these guidelines do not represent a threshold below which no benefits are achieved. There is undoubtedly a point of diminishing return, where more exercise is not necessarily better. Conversely, it is also absolutely clear that some activity is always better than no activity. Somewhere between the minimal and maximal doses of exercise/physical activity may be an optimal dose. Such a dose would provide the greatest health benefit for the least amount of time and effort and the least risk of injury (American College of Sports Medicine, 2022; Haskell, 2007). Similarly, both athletes and coaches would like to know dose-response and optimal levels of training or fitness to maximize performance. Ideally, one dose would also be optimal for all possible desirable health/performance outcomes and all populations. This is highly unlikely, but unknown at this point. As a result, a variety of public health statements on the amount of exercise/physical activity/fitness necessary for obtaining healthrelated benefits are available. These are discussed in this text, as well as considerations for sport performance in the context of the

application of the training principles.

Training Principles

Although there is much we do not know about training, and new training techniques appear often, eight fundamental guidelines are well established and should form the basis for the development of any training program. These **training principles** are defined and briefly explained in the following sections, but the specific details for applying each principle, as well as the anticipated results or adaptations, are discussed in appropriate later units.

Training Principles Fundamental guidelines that form the basis for the development of an exercise training program.

1. *Specificity*. This principle is sometimes called the SAID principle, which stands for “specific adaptations to imposed demands”; that is, what you do is what you get.

When you develop an exercise training program, you must first determine the goal of the training. Fitness programs for children and adolescents, for example, differ from those for older adults. Training programs for individuals seeking health benefits differ from training programs for athletes. Athletic training programs vary by sport, by event, or even by position within the same sport.

Second, you analyze the physiological requirements necessary for meeting the goal. What physiological system is being primarily stressed: the cardiovascular-respiratory, the metabolic, or the neuromuscular-skeletal? What is the major energy system involved? What motor fitness attributes (agility, balance, flexibility, strength, power, muscular endurance) need to be developed? The more closely the training program matches these factors, the greater its chance for success.

2. *Overload*. Overload is a demand placed on the body greater than that to which it is accustomed. To determine the overload, first evaluate the individual's current

physiological/physical capacity by assessing key variables (specificity). Then in determining how the overload will be achieved, consider three factors: frequency—the number of training sessions daily or weekly; intensity—the level of work, energy expenditure, or physiological response in relation to the maximum; and duration—the amount of time spent training per session or per day. **Training volume** is the quantity or amount of overload (frequency times duration for anaerobic or aerobic continuous exercise or number of sets times number of repetitions for resistance exercise), whereas training intensity represents the quality of overload.

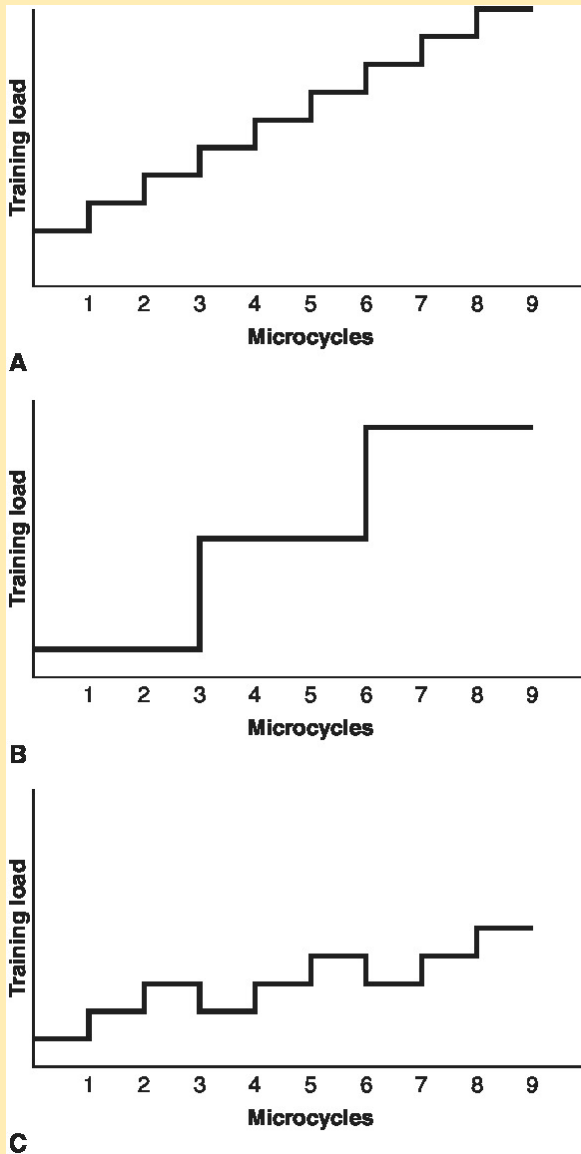
Training Volume The quantity of training overload calculated as frequency times duration for anaerobic or aerobic continuous exercise or number of sets times number of repetitions for resistance exercise.

3. *Rest/Recovery/Adaptation.* Adaptation is the change in physiological function that occurs in response to training. Adaptation occurs during periods of rest, when the body recovers from the acute homeostatic disruptions and/or residual fatigue and, as a result, may compensate to above-baseline levels of physiological functioning. This is sometimes called *supercompensation* (Bompa, 1999; Freeman, 1996). It is, therefore, critical for exercisers to receive sufficient rest between training sessions, after periods of increased training overload, and both before and after competitions. Adaptation allows the individual to either do more work or do the same work with a smaller disruption of baseline values. Keeping records and retesting individuals are generally necessary to determine the degree of adaptation.
4. *Progression.* Progression is the change in overload in response to adaptation. The best progression occurs in a series of incremental steps (called *steploading*), in which every third or fourth change is actually a slight decrease in training load (Bompa, 1999; Freeman, 1996). This step-down allows for recovery, which leads to adaptation. Each step should be small, controlled, and flexible. A continuous unbroken

increase in training load should be avoided. Complete the [Check Your Comprehension 2](#) box.

CHECK YOUR COMPREHENSION 2

Below are three patterns of overload progression in the general conditioning phase of an athlete's training. Select the one that is best, and justify your answer.



Check your answer in [Appendix C](#).

5. *Retrogression/Plateau/Reversibility*. Progress is rarely linear, predictable, or consistent. When an individual's adaptation or performance decreases, retrogression has occurred. If

performance levels off, a plateau has been reached. A plateau should be interpreted relative to the training regimen. Too much time spent doing the same type of workout using the same equipment in the same environment can lead to a plateau. Either too little or too much competition can lead to a plateau. Plateaus are a normal consequence of a maintenance overload and may also occur normally, even during a well-designed, well-implemented steploading progression. Variety and rest may help the person move beyond a plateau. However, if a plateau continues for some time or if other signs and symptoms appear, then the plateau may be an early warning signal of overreaching or overtraining ([Chapter 22](#)). Retrogression may also signal overreaching or overtraining. Reversibility is the reversal of achieved physiological adaptations that occurs after training stops and is also known as detraining. Detraining depends upon the training status of the individual, the degree of reduction in the exercise training, which overload component is impacted most, and the length of time training is interrupted. The timeline for detraining is different for different physiological variables.

6. *Maintenance.* Maintenance is sustaining an achieved adaptation with the most efficient use of time and effort. At this point, the individual has reached an acceptable level of physical fitness or training. The amount of time and effort required to maintain this adaptation depends on the physiological systems involved. For example, more time and effort are needed to maintain adaptations in the cardiovascular system than in the neuromuscular system. In general, intensity is the key to maintenance. That is, as long as exercise intensity is maintained, frequency and duration of exercise may decrease without losing positive adaptations.
7. *Individualization.* Individuals require personalized exercise prescriptions based on their fitness levels and goals. Individuals also adapt differently to the same training program. The same training overload may improve physiological performance in one individual, maintain physiological and performance levels in a second individual, and result in maladaptation and performance decreases in a third. Such differences often result from lifestyle factors,

particularly nutritional and sleep habits, stress levels, and substance use (such as tobacco or alcohol). Age, sex, genetics, disease, and the training modality also all affect individual exercise prescriptions and adaptations.

8. *Warm-Up/Cooldown.* A warm-up prepares the body for activity by elevating the body temperature. Conversely, a cooldown allows for a gradual return to normal body temperature. The best type of warmup is specific to the activity that will follow and individualized to avoid fatigue.

Another important element beyond the physiological training principles is motivation. Except at a military boot camp, it is very difficult to force anyone to train. Therefore, any training program should also be fun. Intersperse games, variations, and special events with the training strive to make normal training sessions as enjoyable as possible.

Complete the [Check Your Comprehension 3 box now](#).

CHECK YOUR COMPREHENSION 3

Mark, a 30-year-old teacher, was disappointed when he ran the local 10-km (6.2 mi) Corn Harvest race in 49:04, which was within seconds of his previous year's time under similar weather conditions. To prepare for the race, he had been training for the previous 6 months. His training consisted of walking for 30 minutes on the Stairmaster at level 10, 2 d-wk -1; running 3 mi in 25–30 minutes on another 2 days; and swimming or bicycling for 45–60 minutes on a 5th day. He did his runs and cycling with Kristi, a 25-year-old fellow teacher, and let her set the pace. She did not run the race.

Considering all eight training principles, identify three principles that apply here and that Mark may not have followed very well. For each one, make a suggestion as to how he might better apply it to prepare more successfully for the upcoming Turkey Trot 10-km race in 3 months. Check your answers in [Appendix C](#).

Periodization

A training program should be implemented in a pattern that is most beneficial for adaptations. This pattern is called the training cycle or periodization. **Periodization** is a plan for training based on a manipulation of the sport's fitness components and the training principles. The objective is to peak the athlete's performance for the competitive season or some part of it while managing fatigue and avoiding overtraining (Morgans et al., 2014; Plisk and Stone, 2003). In general, periodized training progresses from high-volume, low-intensity training to high-intensity, low-volume training for both endurance and resistance weight training as the program progresses (Plisk and Stone, 2003). There are a variety of ways of organizing periodization schedules. An individual training for health-related physical fitness should also use periodization to build in cycles of harder or easier training, or to emphasize one component or another, to prevent monotony.

Periodization Plan for training based on a manipulation of the fitness components with the intent of peaking the athlete for the competitive season or varying health-related fitness training in cycles of harder or easier training.

Figure 1.7 is an example of how periodization might be arranged for an athlete, in this case a basketball player whose season lasts approximately 4.5 months. This is intended as an example only, because periodization depends on the individual's situation and abilities. The time frame in our example is for 1 year—presented as 52 weeks (outer circle)—and is divided into four phases or cycles (Bompa, 1999; Freeman, 1996; Kearney, 1996):

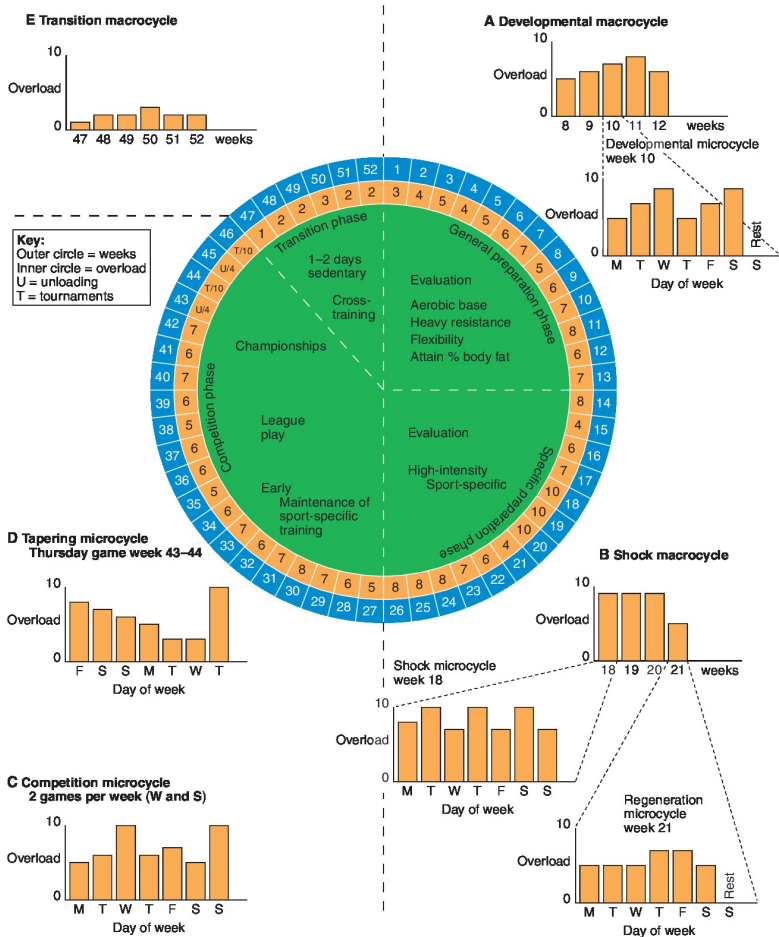


Figure 1.7 Periodization Phases and Overload.

The annual training plan consists of four phases: the general preparation phase (which concentrates on developing the health-related physical fitness components), the specific preparation phase (which emphasizes development of the sport-specific physical fitness components), the competition phase (which emphasizes maintenance of all the physical fitness components), and the transition phase (which allows rest and recovery from the season but emphasizes cross-training to avoid complete detraining). Overload is rated on a scale of 0 (complete rest) to 10 (maximal) and is shown in the tan boxes on the *inner circle*. Three of the five types of

macrocycles are illustrated [A (developmental), B (shock), and E (transition)] in the phases where they typically are used. Four of the five types of microcycles are also shown [A (developmental), B (shock), C (competition), and B and D (regeneration/tapering)] where they are typically used.

1. The general preparation phase (sometimes labeled off-season)
2. The specific preparation phase (also known as preseason)
3. The competition (or in-season) phase
4. The transition (active rest) phase

Overload is rated on a scale of 0 (complete rest) to 10 (maximal) and is shown in the boxes on the inner circle. These values are relative to the individual athlete and do not represent any given absolute training load.

General Preparation Phase

The general preparation (off-season) phase should be preceded by a sport-specific fitness evaluation to guide both the general and specific preparation training program. Another evaluation might be conducted before the season if desired, or evaluations might be conducted systematically throughout the year to determine how the individual is responding to training and to make any necessary adjustments. All evaluation testing should be done at the end of a regeneration cycle so that fatigue is not a confounding factor. As the name implies, the general preparation phase (off-season) is a time when total body health-related physical fitness components are emphasized to develop cardiovascular-respiratory endurance (an aerobic base), flexibility, as well as muscular strength, muscular power, and muscular endurance. Any needed changes in body composition should be addressed during this phase (Kibler and Chandler, 1994). An aerobic base is important for all athletes, even those whose event is primarily anaerobic. A high aerobic capacity allows the individual to work at a higher intensity before accumulating large quantities of lactate. A high aerobic capacity also allows the individual to recover faster, which is important in

itself and allows for a potentially greater total volume of work during interval sessions (Bompa, 1999).

The general preparation phases may occupy most of the year for a fitness participant. During this phase, overload progresses by steps in both intensity and volume (frequency times duration/sets times repetitions), with volume typically being relatively more important than intensity (Bompa, 1999).

Specific Preparation Phase

During the specific preparation phase (preseason phase), the athlete shifts to exercises for the fitness and physiological components needed to succeed in the intended sport. The training program is very heavy and generally occupies at least 6–8 weeks before the first competition or longer (in the example it is 12 weeks) before league competition. About midway through the specific preparation phase, intensity may surpass volume in importance. Alternately, this shift to more emphasis on intensity than volume may not occur until the following competition phase. This varies according to the physiological demands of particular sports and the relative length of the two phases. Sport technical skill training typically increases as the specific preparation phase progresses (Kibler and Chandler, 1994; Plisk and Stone, 2003).

Competition Phase

Once the athlete begins the competition phase, the emphasis first shifts to maintaining the sport-specific fitness developed during the preseason. Although both volume and intensity may be maintained or intensity may even increase, heavy work should immediately follow a competition instead of directly preceding one. During the late season, when the most important competitions are usually held (such as conference championships or bowl games), the athlete should do only a minimum of training or taper gradually so that he or she is rested without being detrained. An analysis of 27 tapering studies revealed that the optimal taper for maximizing performance consists of 2 weeks during which training volume is exponentially reduced by 41–60% without any modification of either training intensity or

frequency (Bosquet et al., 2007).

Transition Phase

The transition phase begins immediately after the last competition of the year. The athlete should take a couple days of complete rest and then participate in active rest using noncompetitive physical activities outside the primary sport. This type of activity is often called crosstraining. In this transition phase, neither training volume nor intensity should exceed low levels (Kibler and Chandler, 1994).

Macrocycles

Each periodization phase is typically divided into several types of *macrocycles* that may each vary in length from 2 to 6 weeks (Fry et al., 1992; Kibler and Chandler, 1994). It should be noted that some systems use the term macrocycle to indicate the total yearly plan and the term mesocycle to indicate phases that last 2–6 weeks (Plisk and Stone, 2003). In this text, we will use the definitions as indicated. Each type of cycle aims for an optimal mixture of work and rest. Macrocycles have five basic goals or patterns, described in the following sections. Different types of macrocycles may be used in a single phase of training.

1. *Developmental macrocycle.* **Figure 1.7A** illustrates a developmental macrocycle typically used in the preparation stages. It is designed to improve either general or specific fitness attributes, such as strength, progressively. Overloading is achieved by a stepwise progression from low to medium to high by gradually increasing the load for three cycles (e.g., weeks 9, 10, and 11 from week 8), followed in week 12 by a regeneration cycle back to the level of the second load or first increase, week 9. This level then becomes the base for the next loading cycle. This is what is meant by steploading.
2. *Shock macrocycle.* Shock macrocycles, such as the one illustrated in **Figure 1.7B**, are used primarily during the two preparation phases and are designed to increase training demands suddenly. They should always be followed by an

unloading regeneration cycle consisting of a drastically reduced training load.

3. *Competition macrocycle*. Competition macrocycles are based on maintaining physiological fitness while optimizing performance for competitions. Obviously, competition macrocycles occur during the competition phase of periodization.
4. *Tapering or unloading regeneration macrocycle*. Tapering or unloading regeneration macrocycles involve systematic decreases in overload to facilitate a physiological fitness peak or supercompensation (Bompa, 1999). As noted, unloading regeneration cycles are used both as breaks between other cycles and as the basis of the active transition phase (Bompa, 1999; Freeman, 1996; Kibler and Chandler, 1994).
5. *Transition macrocycles*. Transition or regression macrocycles (Figure 1.7E) occur during the transition phase and involve very little overload. Tapering (regeneration) and transition phases are intended to remove fatigue, emphasize relaxation, and prevent overreaching/overtraining. Some reversal (regression) of conditioning is expected. These phases are just as valuable for the fitness participant as the athlete.

Microcycles

Macrocycles are further divided into *microcycles*, each lasting 1 week (Fry et al., 1992; Kibler and Chandler, 1994) (Figure 1.7A–D). Similar to macrocycles, microcycles can be developmental, shock, competition or maintenance, tapering or unloading regeneration, or transition or regression. Different types of microcycles may also be used in a single phase or macrocycle. For example, Figure 1.7B illustrates a shock macrocycle that contains both shock microcycles and a regeneration (unloading) microcycle. Likewise, Figure 1.7C and D depict a tapering microcycle and a competition microcycle in the competition phase. Microcycles are further subdivided into specific daily workouts or lesson plans designed by the coach for the athlete. Depending on the athlete's maturity and experience and the level of competition, a training day may entail one, two, or three workouts (Bompa, 1999).

Where possible throughout this text, periodization will be considered in the application of the training principles. See the Focus on Application box for an example of a study that tested two variations of periodization in previously untrained female college students.

Training Adaptations

Training (**Figure 1.8**) brings about physical and physiological changes typically labeled adaptations. **Training adaptations** are physiological changes or adjustments resulting from an exercise training program that promote optimal functioning. One of the most fundamental concepts that a student must understand is the difference between an exercise response (acute change in physiological variables during exercise) and a training adaptation (chronic changes in physiological variables as a result of a training program). Whereas exercise responses (those physiological changes that occur during a single acute exercise bout) use resting values as the baseline (e.g., the pattern of heart rate rise to min 30 of a long-term, moderate to heavy submaximal aerobic exercise is compared to the resting heart rate), training adaptations are evaluated against the same condition prior to training. That is, posttraining values for the variable of interest (on the y-axis) at rest are compared to pretraining values of that variable at rest (e.g., is the resting systolic blood pressure higher, lower, or the same after training as the resting value was before?). Posttraining values in the variable of interest during submaximal exercise are compared to pretraining values under the same submaximal exercise conditions. Similarly, posttraining maximal values of the variable of interest can be compared to pretraining maximal values. Time or exercise intensity, as always, is on the x-axis for the line graphs. Training adaptations may be presented as exercise response patterns using a line graph where T = trained state and UT = untrained state (**Figure 1.9A–C**) or simply as specific values using a bar graph where T1 (Time 1) indicates the pretraining value and T2 (Time 2) indicates the posttraining value (**Figure 1.9D**).



Figure 1.8 Training to Improve Physiological Function and Skill for Improved Performance.

y axis = variable and unit

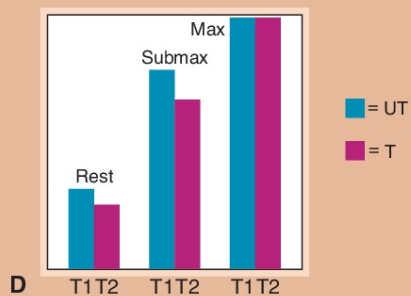
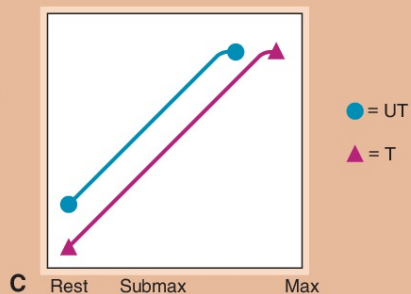
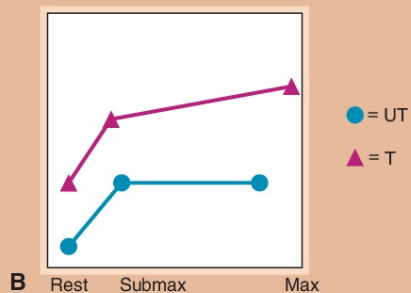
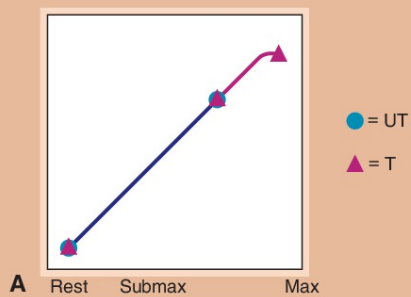


Figure 1.9 Graphic Patterns Depicting Training Adaptations.

Training adaptations are evaluated by comparing variables of interest before and after the training program during the same condition; that is, at rest, during submaximal exercise, or at maximal exercise. Before and after are depicted either by separate lines for untrained (UT) or trained (T) individuals or by the designations T1 and T2 indicating the first test and second test separated by the training program. Compared with the untrained state, training may cause no change, an increase, or a decrease in the measured variable.

Training Adaptations Physiological changes or adjustments resulting from an exercise training program that promote optimal functioning.

Training programs are undertaken in order to bring about a change (adaptation) in physical ability or physiological variable. Training can result in adaptations that are an increase, a decrease, or unchanged state compared to the untrained (baseline) state. To see this concept graphically, refer to **Figure 1.9**. This figure is showing how exercise responses vary between individuals who are trained or untrained. Thus, *the figure depicts both an exercise response and a training adaptation*. In **Figure 1.9A**, we see an example where there is no difference in the values of this theoretical variable between the trained and untrained individuals either at rest or during submaximal exercise. However, the trained group had a higher maximum value than the untrained group. In **Figure 1.9B**, training resulted in an increase at rest, during submaximal exercise, and at maximum. **Figure 1.9C and D** presents the same adaptations in both a line graph and a bar graph to show how each might look. Both graphs indicate that training resulted in a decrease at rest and submaximal work, but no change at maximum.

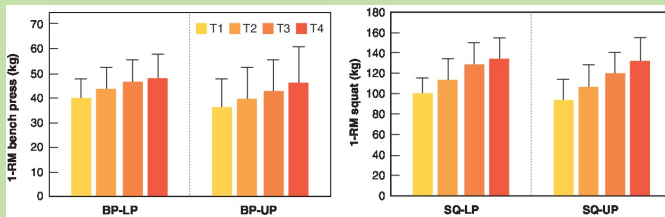
FOCUS ON APPLICATION

Linear versus Undulating Periodization

Two of the basic periodization models for resistance training are the linear (also known as traditional or stepwise) model and the undulating (also known as the nontraditional, nonlinear, or mixed methods) model. Linear periodization starts with the typical high-volume/low-intensity training and progresses through an emphasis on hypertrophic adaptation (strength-endurance) to basic strength and finally to strength and power, thus gradually reversing the emphasis to high-intensity/low-volume training. In undulating training, however, volume and intensity vary on a weekly or daily basis.

Kok et al. (2009) compared these two methods in 20 college students when total workload and average training intensity were matched by the end of the training. All participants received four familiarization sessions prior to testing and 3 weeks of pretraining conditioning before baseline testing. In the linear training group (LP), the exercise prescription was by phase and varied only in terms of the % of 1-RM (percentage of the maximal amount of weight the individual could lift one time) for each set, whereas for the undulating training group (UP), both the number of repetitions per set and the % 1-RM varied by day.

Results of the training adaptations for the 1-RM bench press (kg) and 1-RM squat (kg) are presented in the accompanying figure. Both groups improved significantly in these two lifts at T2 (week 6), T3 (week 9), and T4 (week 12) tests when compared to the baseline testing at T1 (week 3). Squat jump height and bench press throw, indicators of power (not shown), improved similarly. It was concluded that both programs improved strength and power equally, and therefore, either technique may be used.



Source: Adapted with permission from Kok, L. -Y., P. W. Hamer, & D. J. Bishop: Enhancing muscular qualities in untrained women: Linear versus undulating periodization. *Medicine & Science in Sports & Exercise*. 41(9):1797-1807 (2009). Copyright ©2009 The American College of Sports Medicine.

Training adaptations at rest show more variation than either submaximal or maximal changes. In general, if the exercise test is an absolute submaximal test, the physiological responses will probably be decreased after training. For example, heart rate at a work rate of 600 kgm·min⁻¹ might be 135 b·min⁻¹ for an individual before training but 128 b·min⁻¹ after training. If the exercise test is a relative submaximal test, the physiological responses will probably show no change after training. That is, if

an individual were to cycle at 75% $\dot{V}O_2 \text{ max}$ both before and after training, it would be assumed that the $\dot{V}O_2 \text{ max}$, and therefore the amount of external work done at 75%, had increased because of the training. However, both the before and after training heart rate could be 142 b·min⁻¹. If the comparison is made at maximal effort, most physiological responses will increase, such as the $\dot{V}O_2 \text{ max}$ in the preceding absolute example. These results do not hold for all variables but are general patterns.

The predominant way of looking at training adaptations (as opposed to during exercise responses) is that not only do they result from the chronic application of exercise but also they themselves represent *chronic changes*. Such adaptations become greater with harder training, are thought to exist as long as the

training continues, and gradually return to baseline values when training stops (detraining). In reality, however, not all training adaptations follow this standard pattern. Some benefits occur only immediately after the exercise session. These effects are called *last-bout effects* and should not be confused with the exercise response. For example, a last-bout effect can occur with blood pressure levels. The acute response of blood pressure to continuous aerobic endurance exercise is an increase in systolic blood pressure but little or no change in diastolic blood pressure. In the recovery period after exercise, systolic blood pressure decreases. In normal individuals, the decrease is back to the exerciser's normal resting value in a matter of minutes. In individuals with high blood pressure, this postexercise decrease can result in values below their abnormally high resting level for up to 3 hours after exercise. After 3 hours, the high resting values return. In some cases, the last-bout effect can be augmented. That is, assuming the individual participates in a training program of sufficient frequency, intensity, and duration for a period of one to several weeks, the positive change occurring after each exercise bout may be increased. In the example above, the decrease in systolic blood pressure might be 2 mmHg initially, but after several weeks, the postexercise blood pressure decrease might be 6 mmHg for several hours. However, the adjustments that can occur are finite. Once the level of the *augmented last-bout effect* is reached, no further increase in training will bring about additional benefit ([Haskell, 1994](#)). This may be the reason why frequency and consistency are so important in overload for adaptation. Overall, then, the adaptations that result from training can occur on three levels:

1. A chronic change
2. A last-bout effect
3. An augmented last-bout effect

The majority of the training adaptations are dealt with in this book as if they are chronic changes unless otherwise specified.

Exercise and Training as Stressors

Exercise and training are often considered only in a positive manner, but both acute exercise and chronic training are stressors.

Selye's Theory of Stress

A stressor is any activity, event, or impingement that causes stress. **Stress** is defined most simply as a disruption in body homeostasis and all attempts by the body to regain homeostasis. Selye defines stress more precisely as “the state manifested by a specific syndrome that consists of all the nonspecifically induced changes within a biological system.” The biological system here is the human body. The specific syndrome is the general adaptation syndrome (GAS), a step-by-step description of the bodily reactions to a stressor. It consists of three major stages (Selye, 1956):

Stress The state manifested by the specific syndrome that consists of all the nonspecifically induced changes within a biological system; a disruption in body homeostasis and all attempts by the body to regain homeostasis.

1. The Alarm-Reaction: shock and countershock
2. The Stage of Resistance
3. The Stage of Exhaustion

In the *Alarm-Reaction stage*, the body responds to a stressor with a disruption of homeostasis (shock). It immediately attempts to regain homeostasis (countershock). If the body can adjust, the response is mild and advantageous to the organism; the *Stage of Resistance* or adaptation ensues. If the stress becomes chronic or the acquired adaptation is lost, the body enters the *Stage of Exhaustion*. At this point, the nonspecifically induced changes, which are apparent during the Alarm-Reaction but disappear during the Stage of Resistance, become paramount. These changes are labeled the triad of symptoms and include enlargement of the adrenal glands, shrinkage of thymus and lymphatic tissue, and bleeding ulcers of the digestive tract.

Specifically induced changes directly related to the stressor may also occur; for example, if the stressor is cold (shock), the body may shiver to produce heat (countershock). Ultimate exhaustion is death (Selye, 1956).

Selye's Theory of Stress Applied to Exercise and Training

In the context of Selye's theory of stress, the pattern of responses exhibited by physiological variables during a single bout of exercise results directly from the disruption of homeostasis. This is the shock phase of the Alarm-Reaction stage. For many physiological processes (respiration, circulation, energy production, and so forth), the initial response is an elevation in function. The degree of elevation and constancy of this elevation depends on the intensity and duration of the exercise. Appropriate changes in physiological function begin in the countershock phase of the Alarm-Reaction and stabilize in the Stage of Resistance if the same exercise intensity is maintained for at least 1–3 minutes. The Stage of Exhaustion that results from a single bout of exercise, even incremental exercise to maximum, is typically some degree of fatigue or reduced capacity to respond to stimulation, accompanied by a feeling of tiredness. This fatigue is temporary and readily reversed with proper rest and nutrition.

Training programs are made up of a series of acute bouts of exercise organized in such a way as to provide an overload that puts the body into the Alarm-Reaction stage followed by recovery processes that not only restore homeostasis but also encourage supercompensation or adaptation (Kenttä and Hassmén, 1998; Kuipers, 1998; O'Toole, 1998). This can be manifested by altered homeostatic levels at rest, dampened homeostatic disruptions to absolute submaximal exercise loads, and/or enhanced maximal performances or physiological responses. When these adaptations occur, the body has achieved a Stage of Resistance. **Table 1.3** shows how the training principles previously introduced operate in the three stages of Selye's general adaptation syndrome.

TABLE 1.3 Selye's Theory of Stress Applied to

Exercise Physiology

| Stage | Exercise Response | Training Principle | Training Adaptation/ Maladaptation |
|--|--|---|--|
| I. Alarm-Reaction a. Shock b. Countershock | Neuroendocrine system stimulated a. Homeostasis disrupted b. Begin to attain elevated steady state | Warm-up/cooldown Overload Progression* | Dampened response to equal acute exercise stimulus |
| II. Stage of Resistance | Elevated homeostatic steady state maintained if exercise intensity is unchanged | Adaptation Maintenance Specificity (SAID) individualization Reversibility | Enhanced function/physical fitness/ health; increased maximal exercise depending on imposed demand and individual neuroendocrine physiology Adaptation is reversible with detraining Overreaching† |
| III. Stage of Exhaustion | Fatigue, a temporary state, reversed by proper rest and nutrition | Retgression/plateau reversibility | Overreaching Overtraining syndrome Maladaptation changes in neuroendocrine systems |

*The cycle of adaptation and progression occurs repeatedly during a training program.

†If overreaching is planned and recovery is sufficient, positive adaptation results; if overreaching is accompanied by insufficient recovery and additional overload, overtraining will result.

The goal of a training program is to alternate the exerciser between stages I and II and to avoid time in stage III where recovery is not possible in a reasonable time. This process primarily proceeds by the cyclical interaction (shown by the arrows in **Table 1.3**) between adaptation (changes that occur in response to an overload) and progression (change in overload in response to adaptation). Each progression of the overload should allow for adaptation. However, this is not always accomplished.

Training Adaptation and Maladaptation

The results of exercise training can be positive or negative depending on how the stressors are applied. Training is related to fitness goals and athletic performance on a continuum that is best described as an inverted U (**Figure 1.10**) (Fry et al., 1991; Kuipers, 1998; Rowbottom et al., 1998). At one end of the continuum are individuals who are undertrained and whose fitness level and performance abilities are determined by genetics, disease, and nonexercise lifestyle choices. Individuals whose training programs lack sufficient volume, intensity, or progression for either improvement or maintenance of fitness or performance are also undertrained. The goal of optimal

periodized training is the attainment of peak fitness and/or performance. However, if the training overload is too much or improperly applied, then *maladaptation* may occur.

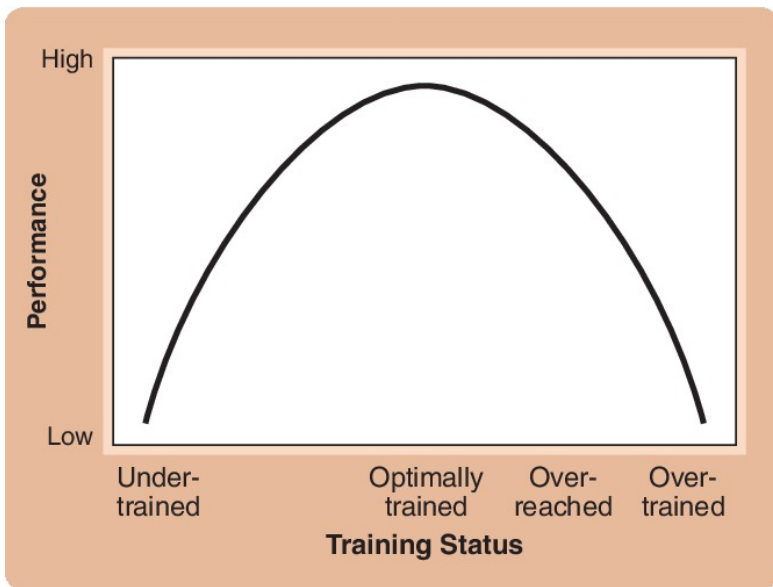


Figure 1.10 Training Status and Performance.

The first step toward maladaptation may be *overreaching* (OR), a short-term decrement in performance capacity that is easily recovered from and generally lasts only a few days to 2 weeks. Overreaching may result from planned shock microcycles, as described in the periodization section, or result inadvertently from too much stress and too little planned recovery (Fry et al., 1991; Fry and Kraemer, 1997; Kuipers, 1998). If overreaching is planned and recovery is sufficient, positive adaptation and improved performance, sometimes called *supercompensation*, result. If, however, overreaching is left unchecked or the individual or coach interprets the decrement in performance as an indication that more work must be done, overreaching may develop into overtraining. Overtraining, more properly called the **overtraining syndrome (OTS)** (or staleness), is a state of chronic decrement in performance and ability to train. It is triggered by metabolic, immune, hormonal, and other dysfunctions that result

from imbalances between training stress and proper recovery. It may take several weeks, months, or even years to recover from (Armstrong and vanHeest, 2002; Cadegiani and Kater 2017; Fry et al., 1991; Fry and Kraemer, 1997; Kreider et al., 1998). The neuroendocrine-immune basis of all stress responses and the maladaptations of overreaching and the overtraining syndrome are discussed in the neuroendocrine-immune unit of this text.

Overtraining Syndrome (OTS) A state of chronic decrement in performance and ability to train that may take several weeks, months, or even years to recover from.

The stress theory enhances our understanding of exercise, exercise training, and physical fitness. As emphasized previously, both exercise and exercise training are stressors. Thus, from the standpoint of stress theory, physical fitness may be defined as achieved adaptation to the stress imposed by muscular exercise. It results as an adaptation from a properly applied training program, is usually exhibited in response to an acute exercise task, and implies avoidance of the OTS.

Strictly speaking, according to stress theory, supercompensation occurs because of a cause-effect relationship between fatigue and fitness. Recent periodization literature emphasizes the opposing effects of fitness and fatigue and is guided by the attempt to maximize the fitness responses to training stimuli while minimizing fatigue. Because fatigue is a natural consequence of training stress and because adaptations are manifested during recovery periods, fatigue management tactics become extremely important (Brown and Greenwood, 2005; Plisk and Stone, 2003).

Summary

1. This chapter presents the general organization of the text and provides background information that will help you interpret and understand the information presented in later

chapters.

2. This chapter differentiates between exercise responses to a single acute bout of exercise and training adaptations to a program of regular exercise.
3. The response to exercise, which is always a disruption in homeostasis, depends on the exercise modality, intensity, and duration. Interpretation of exercise responses must consider characteristics of the exerciser (age, sex, training status), appropriateness of the exercise test used (match between the intended physiological system and outcome), accuracy of the selected exercise (criterion or field test), and environmental and experimental conditions (temperature, relative humidity, barometric pressure, and subject preparation).
4. The baselines against which the exercise-caused disruptions of homeostasis (the exercise response) are compared are normal resting values of the measured variables.
5. Health-related physical fitness is composed of components representing cardiovascular-respiratory endurance, metabolism, and musculoskeletal fitness (muscular strength, muscular endurance, muscular power, and flexibility).
6. Sport-specific physical fitness builds on health-related physical fitness and adds motor fitness attributes (such as agility and balance) and anaerobic power and capacity, as needed.
7. Dose-response relationships describe how a change in one variable (such as exercise training, physical activity, or physical fitness level) is associated with a corresponding change in another variable (e.g., training adaptations, health risk factors, or mortality).
8. Eight general training principles provide guidance for establishing and applying training programs: specificity, overload, rest/recovery/adaptation, progression, retrogression/plateau/reversibility, maintenance, individualization, and warm-up/cooldown.
9. Periodization provides a timeline for the planning of training programs that progresses through four phases or cycles: the general preparation phase (offseason), the specific preparation phase (preseason), the competition phase (in-season), and the transition phase (active rest).

10. To prescribe a training program
 - a. Analyze the physiological demands of the physical fitness program, rehabilitation, or sport goal.
 - b. Evaluate the individual relative to the established physiological demands.
 - c. Apply the training principles relative to the established physiological demands in periodization cycles that allow for a steploading pattern of varying levels of exercise and rest or recovery.
11. Exercise training brings about adaptations in physiological function. Training adaptations are compared to corresponding pretraining conditions (the baseline).
12. Training adaptations may occur on at least three levels: a last-bout effect, an augmented last-bout effect, or a chronic change.
13. In the context of Selye's theory of stress, exercise is a stressor that causes a disruption of the body's homeostasis. During an acute bout of exercise, the body may progress from the Alarm-Reaction stage to the Stages of Resistance and (occasionally) the Stage of Exhaustion. Training programs should be designed to provide an overload that allows adaptation and gradual progression but avoids nonrecoverable time in the Stage of Exhaustion and the overtraining syndrome.

Review Questions

1. Define exercise physiology, exercise, and exercise training.
2. Define and differentiate between exercise response and training adaptation.
3. Graph the most frequent responses physiological variables might exhibit in response to a constant workload/work rate exercise. Verbally describe these responses.
4. Graph the most frequent responses physiological variables might exhibit in response to an incremental exercise to

maximum. Verbally describe these responses.

5. Differentiate between an absolute and relative submaximal workload/work rate exercise, and give an example other than weight lifting.
6. Fully describe an exercise situation, including all elements needed to accurately evaluate the exercise response.
7. Compare the components of health-related physical fitness with those of sport-specific physical fitness.
8. Define dose-response relationship. Give an example relevant to exercise physiology.
9. List and explain the training principles.
10. Explain the phases and cycles of a periodization training program.
11. Differentiate among the three levels of training adaptation, and state which level of adaptation is most common.
12. Explain the relationship of Selye's theory of stress to exercise, training, and physical fitness.

For further review and study tools, visit Lippincott Connect.

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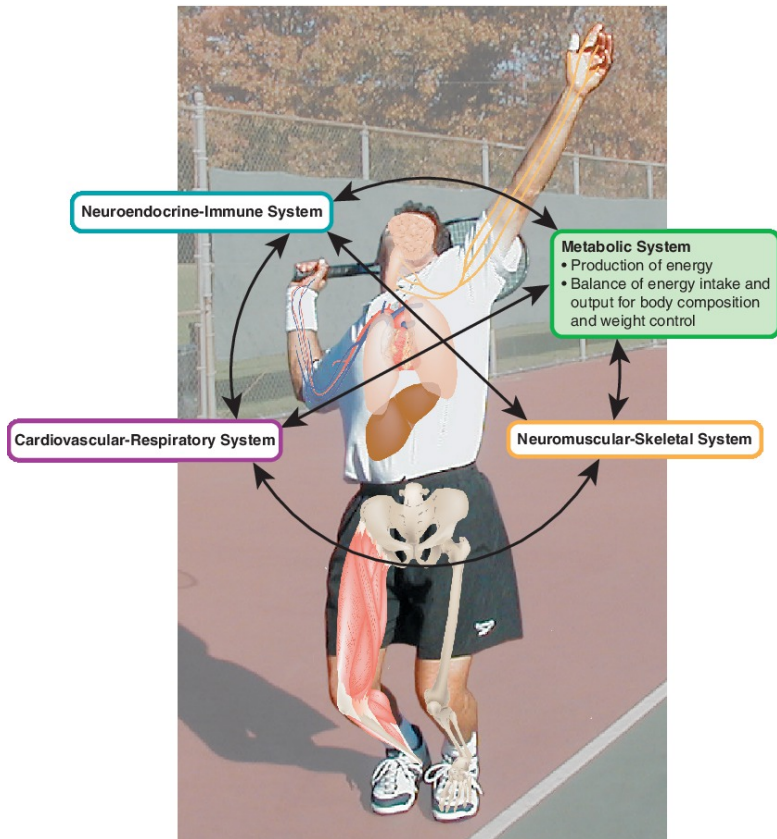
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Metabolic System Unit



Metabolism refers to the sum of all chemical processes that occur within the body. For the study of exercise physiology, the most important metabolic process is how muscle cells convert foodstuffs with or without oxygen to chemical energy (in the form of adenosine triphosphate) for physical activity. Without the production of adenosine triphosphate through metabolic processes, there could be no movement by the neuromuscular system; indeed, there could be no life. Because most energy production requires the presence of oxygen, metabolism largely depends on the functioning of the cardiorespiratory system.

2 Energy Production



CHAPTER OUTLINE

Introduction

Adenosine Triphosphate

Cellular Respiration

Carbohydrate Metabolism

Stage I: Glycolysis Overview

Stage II: Formation of Acetyl Coenzyme A

Stage III: Krebs Cycle

Stage IV: Electron Transport and Oxidative Phosphorylation

ATP Production from Carbohydrate

Fat Metabolism

Beta-Oxidation

ATP Production from Fatty Acids

Ketone Bodies and Ketosis

Protein Metabolism

Transamination and Oxidative Deamination

ATP Production from Amino Acids

The Regulation of Cellular Respiration and ATP Production

Intracellular Regulation

Extracellular Regulation

Fuel Utilization at Rest and During Exercise

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Describe the role of adenosine triphosphate (ATP).
- Summarize the processes of cellular respiration for the production of ATP from carbohydrate, fat, and protein fuel substrates.
- Calculate the number of ATP produced from glucose or glycogen, fatty acid, and amino acid precursors.
- Describe the goals of metabolic regulation during exercise.
- Explain how the production of energy is regulated by intracellular and extracellular factors.
- Compare the relative use of carbohydrate, fat, and protein fuel substrates based on the intensity and duration of exercise.

Introduction

Most individuals eat at least three meals a day. Eating is necessary to provide the energy that is essential for all cellular—and thus bodily—activity. To provide this energy, food must be

transformed into chemical energy.

The total of all energy transformations that occur in the body is called **metabolism**. When energy is used to build tissues—as when amino acids are combined to form proteins that make up muscle—the process is called *anabolism*. When energy is produced from the breakdown of foodstuffs and stored so that it is available to do work, the process is called *catabolism*.

Metabolism The total of all energy transformations that occur in the body.

It is catabolism that is of primary importance in exercise metabolism. Energy is needed to support muscle activity, whether a little or a lot of muscle mass is involved or the exercise is light or heavy, submaximal or maximal. In providing this needed energy, the human body is subject to the *First Law of Thermodynamics*, which states that energy is neither created nor destroyed but only changed in form. **Figure 2.1** depicts this law and the changes in form representing catabolism. Potential chemical energy—or fuel—is ingested as food. Carbohydrates, fats, and protein can all be used as fuels, although they are not used equally by the body in that capacity. The chemical energy produced from the food fuel is stored as **adenosine triphosphate (ATP)**. The ATP then transfers its energy to energy-requiring physiological functions, such as muscle contraction during exercise, in which some energy performs the work and some is converted into heat. Thus, ATP is stored chemical energy that links the energy-yielding and the energy-requiring functions within all cells. The aim of this chapter is to fully explain ATP and how it is produced from carbohydrate, fat, and protein food sources.

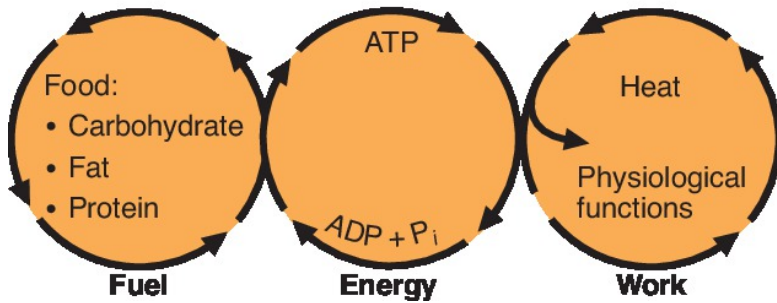


Figure 2.1 Generalized Scheme of Catabolic Energy Transformation in the Human Body.

Energy is the capacity to do work. Energy exists in six forms: chemical, mechanical, heat, light, electrical, and nuclear. Movement of the human body (work) represents mechanical energy that is supported by the chemical energy derived from food fuels. See animation, Catabolism, on

Lippincott Connect. 

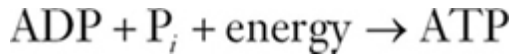
Adenosine Triphosphate (ATP) Stored chemical energy that links the energy-yielding and energy-requiring functions within all cells.

Adenosine Triphosphate

Structurally, ATP is composed of a carbon-nitrogen base called adenine, a five-carbon sugar called ribose, and three phosphates, symbolized by P_i (inorganic phosphate). Each phosphate group is linked by a chemical bond. When one phosphate is removed, the remaining compound is adenosine diphosphate (ADP). When two phosphates are removed, the remaining compound is adenosine monophosphate (AMP).

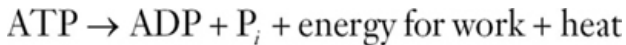
The ATP energy reaction is reversible. When ATP is synthesized from ADP and P_i, energy is required. The addition of P_i is known as **phosphorylation**.

Phosphorylation The addition of a phosphate (P_i).



When ATP is broken down, energy is released. **Hydrolysis** is a chemical process in which a substance is split into simpler compounds by the addition of water. ATP is split by hydrolysis.

Hydrolysis A chemical process in which a substance is split into simpler compounds by the addition of water.

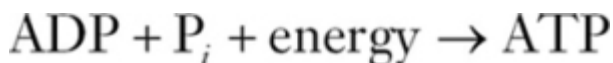


The energy-requiring and energy-releasing reactions involving ATP are **coupled reactions**. Coupled reactions are linked chemical processes in which a change in one substance is accompanied by a change in another; that is, one of these reactions does not occur without the other. As the chemical agent that links the energy-yielding and energy-requiring functions in the cell, ATP is also a universal agent. It is the immediate source of energy for virtually all reactions requiring energy in all cells.

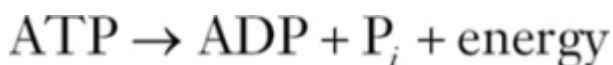
Coupled Reactions Linked chemical processes in which a change in one substance is accompanied by a change in another.

ATP is often referred to as cellular energy. Actually, ATP is a high-energy molecule. The term high-energy means that the probability is high that when a phosphate is removed, energy will be transferred (Brooks et al., 2004). To better understand how the breakdown of ATP releases energy, consider the analogy of a bow and arrow. The arrow can be considered analogous to P_i . It takes energy to draw back the arrow; this energy corresponds to the

energy involved in the energy-requiring reaction:



Once the arrow is loaded, the stretched string has potential energy, which is released when the arrow is fired. Firing corresponds to the energy-releasing reaction:

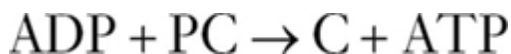


The ATP content of skeletal muscle at rest is about 6 mmol·kg⁻¹. If not replenished, this amount could supply energy for only about 3 seconds of maximal contraction. The total amount of ATP stored in the body at any given time is approximately 0.1 kg, which is enough energy for only a few minutes of physiological function. However, ATP is constantly being hydrolyzed and resynthesized. The average adult produces and breaks down (turns over) approximately 40 kg (88 lb) of ATP daily, and an athlete may turn over 70 kg (154 lb) a day. The rate of hydrolysis of ATP during maximal exercise may be as high as 0.5 kg·min⁻¹ (Mougiou, 2006).

ATP can be resynthesized from ADP in three ways:

1. By interaction of ADP with PC (phosphocreatine, which is sometimes designated as CP, or creatine phosphate)
2. By anaerobic respiration in the cell cytoplasm
3. By aerobic respiration in the cell mitochondria

Phosphocreatine is another high-energy compound stored in muscles. It transfers its phosphate—and, thus, its potential energy—to ADP to form ATP, leaving creatine:



Resting muscle contains more PC (~20 mmol·kg⁻¹) and C (~12 mmol·kg⁻¹) than ATP. The maximal rate of ATP resynthesis from PC is approximately 2.6 mmol·kg⁻¹·sec⁻¹ and

occurs within 1–2 seconds of the onset of maximal contraction. PC stores are used to regenerate ATP and in a working muscle will be depleted in 15–30 seconds. The rest of this chapter will concentrate on the more substantial production of ATP by the second and third techniques listed, namely, anaerobic and aerobic cellular respiration.

Cellular Respiration

The process by which cells transfer energy from food to ATP in a stepwise series of reactions is called **cellular respiration**. This term is used because, to produce energy, the cells rely heavily on the oxygen that the respiratory system provides. In addition, the by-product of energy production, carbon dioxide, is exhaled through the respiratory system. Cellular respiration can be either anaerobic or aerobic. **Anaerobic** respiration means it occurs in the absence of oxygen, does not require oxygen, or does not use oxygen. **Aerobic** means it occurs in the presence of oxygen, requires oxygen, or uses oxygen. Brain cells cannot produce energy anaerobically, and cardiac muscle cells have only a minimal capacity for anaerobic energy production. Skeletal muscle cells, however, can produce energy aerobically and/or anaerobically as the situation demands.

Cellular Respiration The process by which cells transfer energy from food to ATP in a stepwise series of reactions. It relies heavily on the use of oxygen.

Anaerobic In the absence of, not requiring, nor utilizing, oxygen.

Aerobic In the presence of, requiring, or utilizing, oxygen.

Figure 2.2 outlines the products and processes of cellular respiration, which are discussed in detail in the following sections. Following are some basic points about these processes:

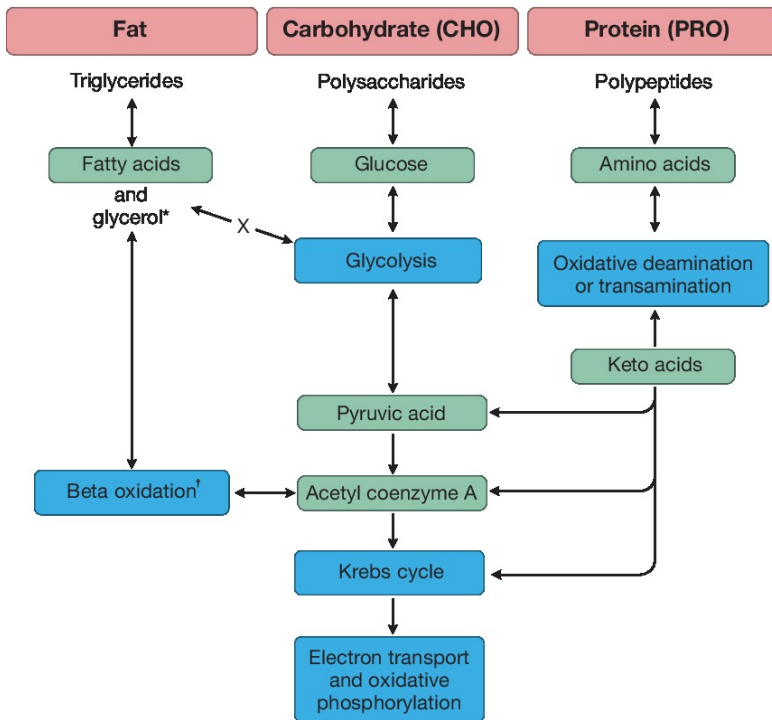


Figure 2.2 An Overview of Cellular Respiration.

*Glycerol can enter glycolysis in liver or fat cells but not in muscle cells. †Blue boxes represent the process; green boxes represent metabolic intermediates; arrows show the direction of flow.

1. All three major food nutrients, fats (FAT), carbohydrates (CHO), and proteins (PRO), can serve as fuel or **substrates**—the substances acted upon by enzymes—for the production of ATP.

Substrate Fuel substance acted on by an enzyme.

2. The most important immediate forms of the substrates utilized are glucose (GLU), free fatty acids (FFA), and amino acids (AA). Both FFA and glycerol are derived from the breakdown of triglycerides. Some cells can use glycerol directly in glycolysis, but muscle cells cannot.
3. Acetyl coenzyme A (acetyl CoA) is the central converting substance (usually called the universal or *common intermediate*) in the metabolism of FAT, CHO, and PRO. Although the process of glycolysis provides a small amount of ATP as well as acetyl CoA, both betaoxidation and oxidative deamination or transamination are simply preparatory steps by which FFA and AA are converted to acetyl CoA. That is, beta-oxidation and oxidative deamination or transamination are simply processes for converting FFA and AA, respectively, to a common substrate that allows the metabolic pathway to continue. The end result is that the primary metabolic pathways of the Krebs cycle, electron transport system (ETS), and oxidative phosphorylation (OP) are the same regardless of the type of food precursor. This is certainly more efficient than having totally different pathways for each food nutrient.
4. Each of the energy-producing processes or stages (glycolysis, formation of acetyl CoA, Krebs cycle, ETS/OP) consists of a series of steps. Each step represents a small chemical change to a substrate, resulting in a slightly different product in a precise, unvarying sequence with a designed first and last step. This is known as a **metabolic pathway**. That is, a metabolic pathway is a sequence of enzyme-mediated chemical reactions resulting in a specific product. Each stage may be made up of one or more metabolic pathways. These sequences of steps are important for the body because they allow energy to be released gradually. If all of the energy contained in food nutrients were released at one time, it would be predominantly released as heat and would destroy tissue.

Metabolic Pathway A sequence of enzyme-mediated chemical reactions resulting in a specified product.

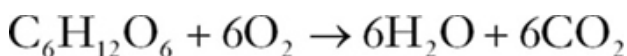
It is easy to be intimidated by or uninterested in so many steps, each with its own chemical structure, enzyme, and long, complicated name. There is a logic to these steps, however. It is on this logic and understanding (rather than the chemical structures) that the following discussion concentrates.

Carbohydrate Metabolism

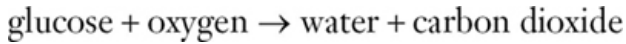
The discussion of ATP production begins with carbohydrate metabolism for several reasons. First, many of the energy requirements of the human body are met by carbohydrate metabolism. Second, carbohydrate is the only food nutrient that can be used to produce energy anaerobically. Energy for both rest and exercise is provided primarily by aerobic metabolism. However, exercise often requires anaerobic energy production, and then carbohydrate is essential. Third, carbohydrate is the preferred fuel of the body because carbohydrate requires less oxygen in order to be metabolized than does fat. Finally, once you understand carbohydrate metabolism, it is relatively simple to understand how fats and proteins are metabolized.

Carbohydrates are composed of carbon (C), oxygen (O), and hydrogen (H). The complete metabolism of carbohydrate requires oxygen, which is supplied by the respiratory system and is transported by the circulatory systems to the muscle cells. The metabolism of carbohydrate also produces carbon dioxide (CO₂), which is removed via the circulatory and respiratory systems, and water.

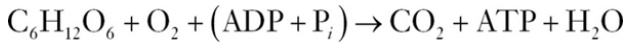
The form of carbohydrate that is exclusively metabolized is glucose, a six-carbon sugar arranged in a hexagonal formation and symbolized as C₆H₁₂O₆. Thus, all carbohydrates must be broken down into glucose in order to continue through the metabolic pathways. In its most simplistic form, the oxidation of glucose can be represented by the equation



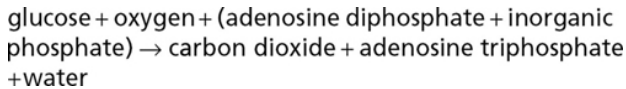
or



In the skeletal muscle, cell oxidation is tightly coupled with phosphorylation to produce energy in the form of ATP. Therefore, the equation becomes



or



When excess glucose is available to the cell, it can be stored as **glycogen**, which is a chain of glucose molecules chemically linked together, or it can be converted to and stored as fat. The formation of glycogen from glucose is called *glycogenesis*. Glycogen is stored predominantly in the liver and muscle cells, as shown in the electron micrograph in **Figure 2.3**. When additional glucose is needed, stored glycogen is broken down (hydrolyzed) to provide glucose, through a process called **glycogenolysis**. Because glycogen must first be broken down into glucose, the production of energy from glucose or glycogen is identical after that initial step. The complete breakdown of glucose and glycogen follows the four-stage process outlined in **Figure 2.4** and detailed in later figures.

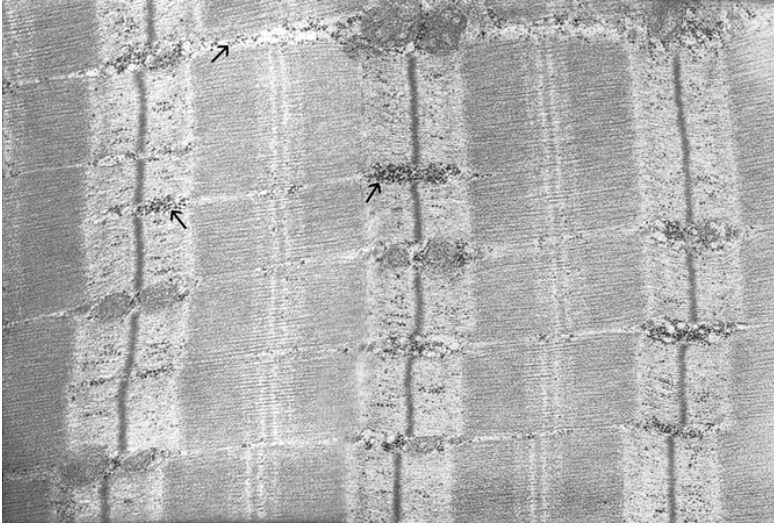


Figure 2.3 Glycogen Storage in Skeletal Muscle.

In this electron micrograph of skeletal muscle (EM: 25,000 \times), stored glycogen is visible as the small round *black dots* near the *arrows* and elsewhere. **Source** : Photo courtesy of Lori Bross, Northern Illinois University Electron Microscope Laboratory.

Embden-Meyerhof Pathway

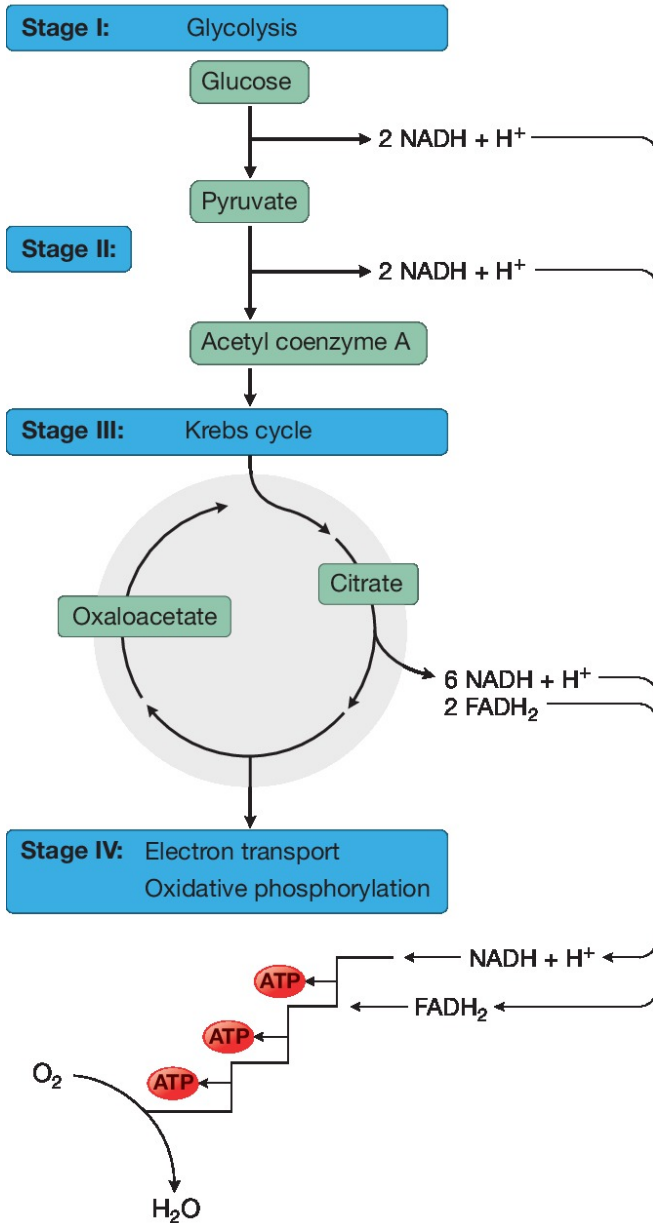


Figure 2.4 The Four Stages of Carbohydrate Cellular Respiration.

Glycogen Stored form of carbohydrate composed of chains of glucose molecules chemically linked together.

Glycogenolysis The process by which stored glycogen is broken down (hydrolyzed) to provide glucose.

Stage I: Glycolysis Overview

Glycolysis is important in part because it prepares glucose to enter the next stage of metabolism (see **Figure 2.4**) by converting glucose to pyruvate. Also very important is the fact that ATP is produced directly during glycolysis. This process is the only way ATP is produced in the absence of oxygen (anaerobically).

Glycolysis literally means the breakdown or dissolution of sugar. It is the energy pathway responsible for the initial catabolism of glucose in a 10- or 11-step process. Glycolysis begins with either glucose or glycogen and ends with the production of either pyruvate (pyruvic acid) by aerobic glycolysis or lactate (lactic acid) by anaerobic glycolysis (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983). (Note that names ending in *-ate* technically indicate the salts of their respective acids; that is lactate is the salt of lactic acid. However, the salt and acid forms are often used interchangeably in descriptions of the metabolic pathways.)

Glycolysis The energy pathway responsible for the initial catabolism of glucose in a 10- or 11-step process that begins with glucose or glycogen and ends with the production of pyruvate (aerobic glycolysis) or lactate (anaerobic glycolysis).

Each step is catalyzed by a specific enzyme. An **enzyme** is a protein that accelerates the speed of a chemical reaction without itself being changed by the reaction. It does not cause a reaction

that would not otherwise occur; it simply speeds up one that would occur anyway. Metabolic enzymes are easy to identify because their names usually end in the suffix *-ase*. Generally, their names are also related to the substrate or type of reaction (or both) they are catalyzing. For those reactions that are reversible, a specific single enzyme will catalyze the reaction in both directions.

Enzyme A protein that accelerates the speed of a chemical reaction without itself being changed by the reaction.

The activity of enzymes is affected by the concentrations of the substrate acted upon and of the enzyme itself, temperature, pH, and the presence or absence of other ions, poisons, or medications. The following discussions describe how enzymes act in a normal physiological environment. Only a few selected enzymes that are especially important or function in regulatory or rate-limiting roles are described here. *Regulatory or rate-limiting enzymes* are the enzymes that are critical in controlling the rate and direction of energy production along a metabolic pathway—just as a traffic light regulates the flow of vehicles along a road.

As a metabolic pathway, glycolysis begins with the absorption of glucose into the bloodstream from the small intestine or with the release of glucose into the bloodstream from the liver. Either step supplies the fuel. Glucose is then transported into the muscle cell. Transport occurs across the cell membrane via facilitated diffusion, utilizing a protein carrier and occurring down a concentration gradient. The carriers are called *glucose transporter carrier proteins*, or *GLUT*, and are differentiated by numbers, from 1 to 14 (Wilson-O'Brien et al., 2010; Zhao and Keating, 2007). Transport is a passive process that does not require the expenditure of energy.

The predominant transporters of glucose in human skeletal and cardiac muscle and adipose cells are GLUT-1 (non-insulin regulated) and GLUT-4 (insulin regulated) (Figure 2.5). GLUT-1 transporters are located in the sarcolemma or cell membrane. In resting muscles when blood glucose levels are relatively stable, most glucose enters by GLUT-1 transport. When glucose and

insulin levels are high, such as after a meal, or when insulin levels are low, such as during exercise, most glucose enters by GLUT-4 transport. The GLUT-4 transporters are thus activated both by insulin, through a second messenger system, and by muscle contraction. Calcium (Ca^{2+}) is probably one of the second messengers (Brooks et al., 2004; MacLean et al., 2000; Wilson-O'Brien et al., 2010; Zhao and Keating, 2007). Within skeletal muscle, the number of GLUT-4 transporters is highest in fast-twitch oxidative glycolytic (FOG, Type IIA) fibers, followed by slow-twitch oxidative (SO, Type I) fibers, and is lowest in fast-twitch glycolytic (FG, Type IIX fibers (Sato et al., 1996).

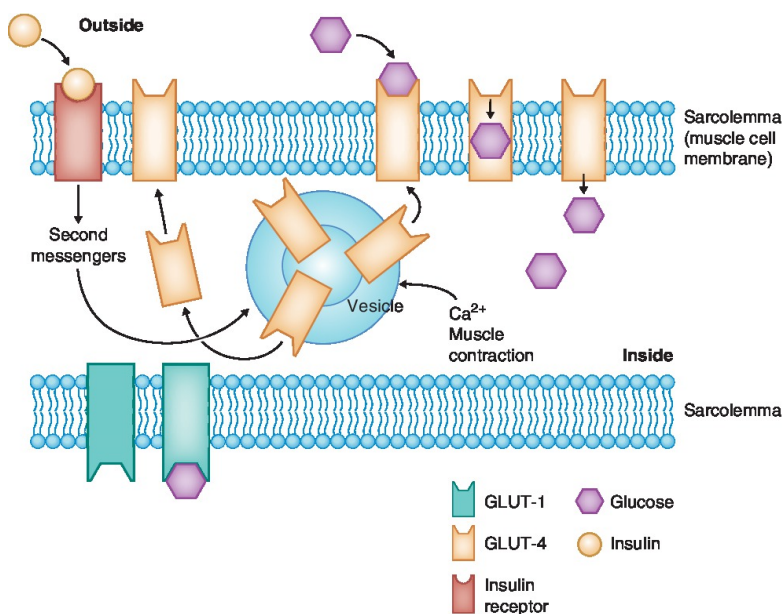


Figure 2.5 Glucose Transport into Muscle Cells.

In the resting muscle, when blood glucose levels are relatively stable, glucose enters by the non-insulin-regulated GLUT-1 transporters. Following a meal, insulin interacts with its receptors, and second messengers stimulate GLUT-4 to translocate from their storage vesicles to the sarcolemma and glucose is transported into the cell. During exercise and early recovery, muscle contraction involves Ca^{2+} release that stimulates the translocation of

GLUT-4.

GLUT-4 transporters exist intracellularly in small sacs or vesicles within the cytoplasm. When activated, they literally move to the cell surface (translocate) and serve as portals through which glucose enters the cell. The maximal rate of muscle glucose transport is determined by both the total number of GLUT-4 molecules and the proportion that are translocated to the cell membrane (Houston, 1995; Sato et al., 1996). The dual stimulation of GLUT-4 translocation by insulin and contraction is important because insulin secretion is suppressed during exercise. Thus, during exercise, the predominant activator of GLUT-4 transporters is the muscle contraction itself. When total work performed is equal, GLUT-4 increases are similar despite differences in exercise intensity and duration (Kraniou et al., 2006). The effect of muscle contraction persists into the early recovery period to help rebuild depleted glycogen stores (Houston, 1995).

FOCUS ON APPLICATION | *Clinically Relevant*

Diabetes Mellitus

As described in the text and shown in **Figure 2.5**, for glucose to enter muscle cells and be used as fuel, insulin and GLUT-4 translocation or muscle contraction and GLUT-4 translocation are required. Prolonged malfunction of this process can result in diabetes mellitus.

Type I diabetes is an autoimmune disease that involves the destruction of the β cells in the pancreas that normally secrete insulin. This leads to a deficiency of insulin, glucose intolerance (the inability to use carbohydrate effectively), and increased blood glucose levels (hyperglycemia).

Type II diabetes is characterized by a progression of steps indicating impaired regulation of glucose metabolism. The initial step is insulin resistance (the inability to achieve

normal rates of glucose uptake in response to insulin). Insulin resistance is indicated by glucose intolerance, sometimes called impaired fasting glucose (IFG). IFG may be diagnosed in an individual more than 10 years before the disease fully develops. In response to insulin resistance, the β cells increase insulin secretion (hyperinsulinemia), which may compensate for the insulin resistance for a while. However, at some point, the β cells will fail and hyperglycemia occurs. This signals that the clinical development of the disease, but not the consequences, is complete.

Research evidence indicates that the initial step of insulin resistance is due to dysfunction in the GLUT-4 translocator process. That is, insulin resistance results from a reduced ability to stimulate GLUT-4 cells to migrate to the cell surface. Precisely what in the insulin-signaling pathway is defective is unknown. The number of both GLUT-1 and GLUT-4 transporters is similar in those with and without type II diabetes.

Although there is a genetic component to type II diabetes, obesity and physical inactivity are strongly related to the expression of this predisposition. Conversely, exercise training increases GLUT-4 function in skeletal muscle that, in turn, improves insulin action on glucose metabolism and can be important in the prevention of, or delay in, the development of type II diabetes.



Sources: Alcazar et al. (2007); Dengel and Reynolds (2004).

Glycolysis takes place in the cytoplasm of the cell. The enzymes that catalyze each step float free in the cytoplasm. The substances acted upon by the enzymes and the resultant products are transferred by diffusion, which is the tendency of molecules to move from a region of high concentration to one of low concentration. Most of the intermediates (everything but glucose and the pyruvate or lactate) are phosphorylated compounds—that is, they contain phosphates. All of the phosphate intermediates, ADP, and ATP are unable to pass through the cell membrane. The cell membrane, however, is freely permeable to glucose and lactate.

Glycolysis, as depicted in **Figure 2.6**, involves both the utilization and production of ATP. One ATP molecule is used in the first step if the initial fuel is glucose, but not if the initial fuel is glycogen. One ATP is used in step 3 regardless of whether the initial fuel is glucose or glycogen. Thus, 1 ATP is used for activation if the initial fuel is glycogen, but 2 ATP are used if the initial fuel is glucose. ATP is produced from $\text{ADP} + \text{P}_i$ at steps 7 and 10 by a process known as substrate-level phosphorylation. **Substrate-level phosphorylation** is the transfer of P_i directly from the phosphorylated intermediates or substrates to ADP without any oxidation occurring. A net total of 3 ATP is gained if glycogen is the initial fuel, but only 2 ATP are gained if glucose is the initial fuel.

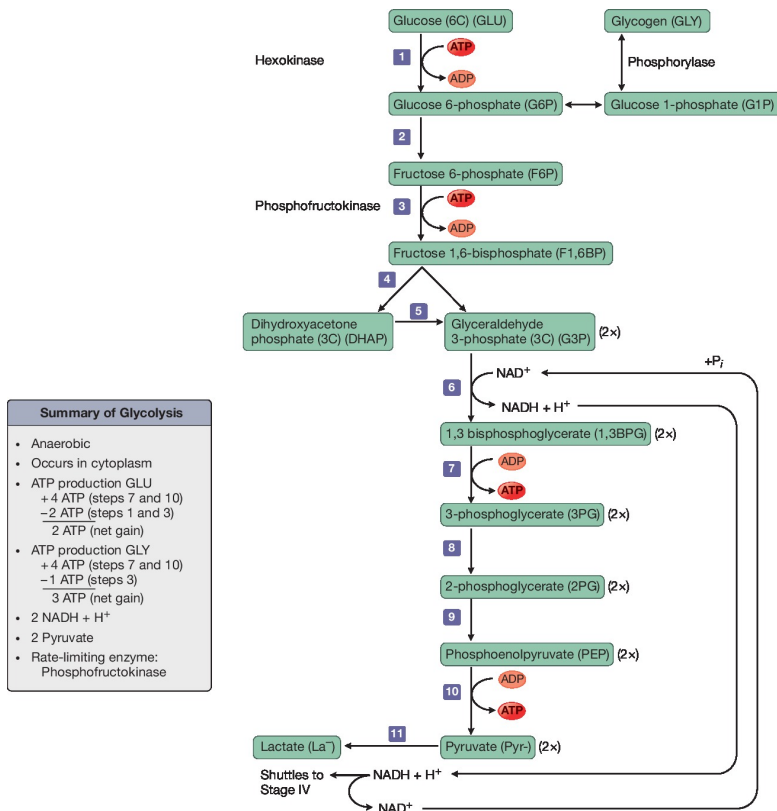


Figure 2.6 Stage I: Glycolysis.

Substrate-Level Phosphorylation The transfer of P_i directly from a phosphorylated intermediate or substrates to ADP without any oxidation occurring.

Exactly how glycolysis is accomplished is explained in the following step-by-step analysis. First, however, one should understand the processes of oxidation and reduction and the roles of nicotinamide adenine dinucleotide (NAD) and flavin adenine dinucleotide (FAD).

Oxidation-Reduction

There are three kinds of **oxidation**: a gain of oxygen (hence the name), a loss of hydrogen, or the direct loss of electrons by an atom or substance.

Oxidation A gain of oxygen, a loss of hydrogen, or the direct loss of electrons by an atom or substance.

The process whereby an atom or substance gains electrons is called **reduction**. The electron's negative charge reduces the molecule's overall charge. A good way to remember that electrons are gained through reduction, not oxidation, is to associate the "e" in reduction with the "e" in electron: reduction = $+e^-$. Reduction can also mean a loss of oxygen or a gain of hydrogen by an atom or a substance. Electron donors are known as reducing agents and electron acceptors as oxidizing agents. The major electron donors are organic fuels, for example, glucose. Oxygen is the final electron acceptor or oxidizing agent in cellular respiration.

Reduction A loss of oxygen, a gain of electrons, or a gain of hydrogen by an atom or substance.

When one substance is oxidized, another is simultaneously reduced. The substance that is oxidized also loses energy; the substance that is reduced also gains energy.

Oxidation in the form of hydrogen removal occurs in several of the intermediary steps in cellular respiration. When hydrogen atoms are removed, they must be transported elsewhere. The two most important hydrogen carriers in cellular respiration are **nicotinamide adenine dinucleotide (NAD)** and **flavin adenine dinucleotide (FAD)**. Both NAD and FAD can accept two electrons and two protons from two hydrogen atoms. Each does so in a slightly different manner, however. FAD (the oxidized form is FAD_{ox}) actually binds both protons and is written as FADH₂ (or FAD reduced, written FAD_{red}). NAD actually exists as NAD⁺ (or NAD oxidized, written NAD_{ox}); when bonded to

hydrogen, it is written as $\text{NADH} + \text{H}^+$ (or NAD reduced, written as NADred). The symbols NAD^+ and FAD will be used to indicate the oxidized form in this text, and $\text{NADH} + \text{H}^+$ and FADH_2 will be used to indicate the reduced form of these carriers, so that it is clear where the hydrogen atoms are. NAD^+ is by far the more important hydrogen carrier in human metabolism.

Nicotinamide Adenine Dinucleotide (NAD) A hydrogen carrier in cellular respiration.

Flavin Adenine Dinucleotide (FAD) A hydrogen carrier in cellular respiration.

A helpful analogy of the roles of FAD and NAD is an Uber or taxi. The purpose of NAD and FAD is to transport (serve as an Uber for) the hydrogen. NAD and FAD must pick up hydrogen passengers (be reduced), and they must drop them off (be oxidized) at another point without either the carrier (Uber) or the hydrogen (passengers) being permanently changed.

The Steps of Stage I

Refer to **Figure 2.6** as each step is explained in the text (Frisell, 1982; Lehninger, 1971; Mougios, 2006; Newsholme and Leech, 1983; Salway, 1994).

Step 1. The glucose molecule, which has six carbons indicated by the (6C), is phosphorylated (has a phosphate attached) through the transfer of one phosphate group from ATP to the location of the sixth carbon on the glucose hexagon, producing glucose-6-phosphate. Hexokinase is the enzyme that catalyzes this process. This phosphorylation effectively traps the glucose in that particular cell, because the electrical charge of the phosphate group prohibits glucose from crossing the membrane, and the enzyme that can break this phosphate bond is not present in muscle cells.

The same is true for the glycogen in the cells; that is, it is

trapped in the muscle where it is stored. On the other hand, the liver has enzymes to break down glycogen and release glucose into the bloodstream.

These facts are important because during high-intensity, long-term exercise, glycogen stored in specific muscles must be used there. Furthermore, glycogen that is stored in muscles but not used in an activity (e.g., the upper body during a running event) cannot be transported from the inactive muscles to the active ones. Therefore, high levels of glycogen need to be stored in the muscles that will be used.

Step 2. The atoms that make up the glucose-6-phosphate are simply rearranged to form fructose-6-phosphate.

Step 3. Another molecule of ATP is broken down and the phosphate added at the first carbon. This addition places a phosphate group at each end of the molecule and results in the product fructose-1,6-bisphosphate. The prefixes *di-* and *bis-* both mean “two,” but *di-* indicates two groups attached to the same spot and *bis-* at different positions. Two phosphates are now part of the molecule. This step is catalyzed by the enzyme phosphofructokinase (PFK), which is the rate-limiting enzyme in glycolysis.

Step 4. This is the step from which glycolysis, meaning sugar breaking or splitting, gets its name, for here the 6-carbon sugar is split into two 3-carbon sugars indicated by the (3C). The two sugars are identical in terms of their component atoms but have different names (dihydroxyacetone phosphate, or DHAP, and glyceraldehyde-3-phosphate, or G3P) because the component atoms are arranged differently.

Step 5. The atoms of dihydroxyacetone phosphate are rearranged to form glyceraldehyde 3-phosphate, with the phosphate group at the third carbon. From this point on, each step occurs twice (indicated by $2\times$ in **Figure 2.6**), once for each of the three carbon subunits.

Step 6. Two reactions that are coupled occur in this step. In the first, a pair of hydrogen atoms is transferred from the G3P to the hydrogen carrier NAD^+ , reducing this to $\text{NADH} + \text{H}^+$. The fate of this $\text{NADH} + \text{H}^+$ will be dealt with later. This reaction releases enough energy to perform the second reaction, which adds a phosphate from the P_i always present in the cytoplasm to

the first carbon, so that the product becomes 1,3-bisphosphoglycerate.

Step 7. ATP is finally produced from ADP in this step when the phosphate from the first carbon is transferred to ADP, storing energy. Since this step is also doubled, two ATP molecules are formed. The resulting intermediate is called 3-phosphoglycerate (3PG).

Step 8. This is simply another rearrangement step where the phosphate is moved from the number 3 to the number 2 carbon and the name becomes 2-phosphoglycerate (2PG).

Step 9. A water molecule is removed, which weakens the bond between the remaining phosphate group and the rest of the atoms forming phosphoenolpyruvate (PEP).

Step 10. The remaining phosphate is transferred from PEP to ADP, forming ATP and pyruvate. The enzyme that catalyzes this step is pyruvate kinase. Again, since the reaction happens twice, two ATP molecules are produced.

Step 11. If the hydrogen atoms carried by $\text{NADH} + \text{H}^+$ are unable to enter the electron transport chain (as described in Stage IV), they are transferred instead to pyruvate (pyruvic acid), forming lactate (lactic acid), which regenerates the NAD^+ . The enzyme catalyzing this reaction is lactic dehydrogenase.

The formula for pyruvate is $\text{C}_3\text{H}_3\text{O}_3$, whereas the formula for lactate is $\text{C}_3\text{H}_5\text{O}_3$. The formula for pyruvic acid is $\text{C}_3\text{H}_4\text{O}_3$, and the formula for lactic acid is $\text{C}_3\text{H}_6\text{O}_3$. Both the number of carbon atoms and the number of oxygen atoms are the same; however, there are four fewer hydrogen atoms in two molecules of pyruvate/pyruvic acid than lactate/lactic acid. These are the hydrogen atoms that are carried by the NAD^+ from step 6 and used to produce the lactate ($2\text{C}_3\text{H}_5\text{O}_3$)/lactic acid ($2\text{C}_3\text{H}_6\text{O}_3$) from pyruvate/pyruvic acid. If lactate/lactic acid is not formed, the hydrogen atoms from step 6 may be carried further down the metabolic chain. Currently, there is some unresolved controversy as to whether skeletal muscle actually produces lactate or lactic acid. What has just been presented represents the accepted classic view and the interchangeable use of the salt and acid forms in the metabolic pathways, and discussions will be maintained throughout this textbook (Brooks, 2010, 2018, 2020; Ferguson et al., 2018; Marcinek et al., 2010; Robergs et al., 2004, 2018).

When the end product of glycolysis is pyruvate/pyruvic acid, the process is called *aerobic glycolysis* (or *slow glycolysis*). When the end product of glycolysis is lactate/lactic acid, the process is called *anaerobic glycolysis* (or *fast glycolysis*). Glucose predominates as the fuel for slow glycolysis, and glycogen predominates as the fuel for fast glycolysis.

Mitochondria

Mitochondria (the plural form of the singular mitochondrion) are subcellular organelles that are often called the powerhouses of the cell. The formation of acetyl CoA, Krebs cycle, electron transport, and oxidative phosphorylation (OP) all take place in the mitochondria.

Mitochondria Cell organelles in which the formation of acetyl CoA, Krebs cycle, electron transport, and oxidative phosphorylation take place.

Figure 2.7A presents a diagram and an electron micrograph of a mitochondrion: a three-dimensional, discretely encapsulated, bean-shaped structure. In actual living tissue, mitochondria exist in many different shapes—and, indeed, they constantly change shapes.

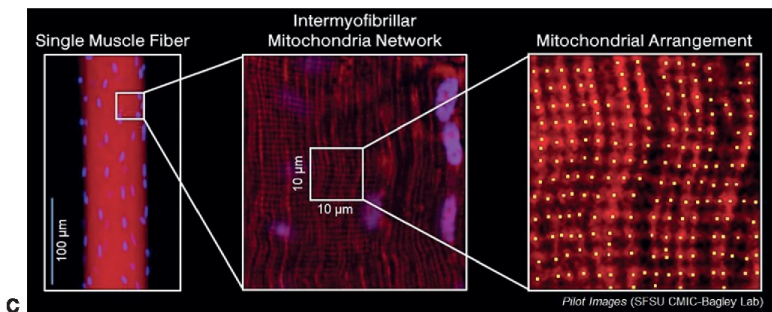
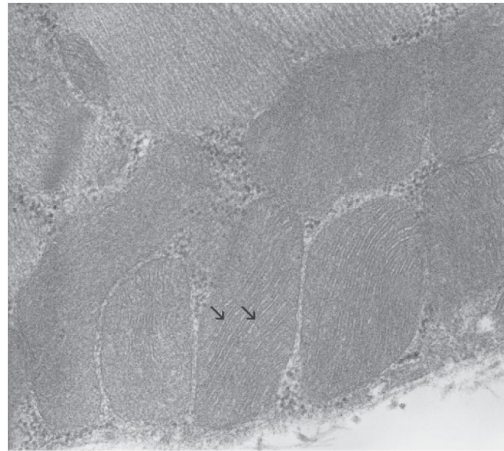
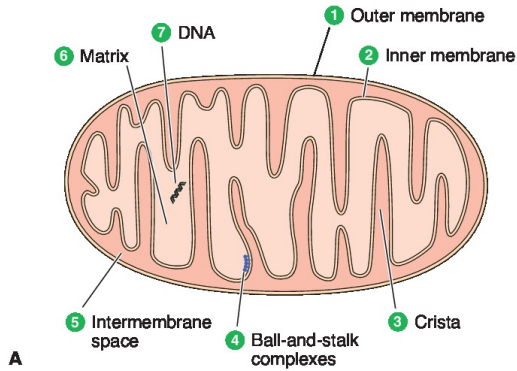


Figure 2.7 Mitochondrion.

A. Labeled schematic diagram of a mitochondrion. **B.** An electron micrograph of skeletal muscle mitochondria (EM: 40,000 \times) in which the *arrows* point to crista. **C.** Fluorescently labeled mitochondria in a human skeletal

muscle fiber. **Source:** Electron micrograph provided by Lori Bross, Northern Illinois University Electron Microscope Laboratory. Fluorescently labeled mitochondria provided by Dr. Jimmy Bagley, San Francisco State University Muscle Physiology Laboratory.

As shown in **Figure 2.7A and B**, mitochondria have seven distinct components. The first component is the outer membrane (labeled 1 in panel A). As is usual, this membrane serves as a barrier; however, it contains many channels through which solutes can pass and so is permeable to many ions and molecules. The second component (2) is an inner membrane. The inner mitochondrial membrane is impermeable to most ions and molecules unless each has a specific carrier. It is, however, permeable to water and oxygen. The inner membrane is parallel to the outer membrane in spots, but it also consists of a series of folds or convolutions called crista (3) (the plural is cristae—identified by the arrows in panel B). Extending through and protruding from the inner membrane are a series of protein-enzyme complexes called ball-and-stalk ATP synthase complexes (4) that look like a golf ball on a tee. Although the inner membrane has transport functions, the crista portion and especially the ball-and-stalk apparatus are specialized as the locations where ATP synthesis actually takes place.

The area between the two membranes is known simply as the intermembrane space (5). The center portion of the mitochondria is known as the matrix (6). The matrix is filled with a gel-like substance composed of water and proteins. Metabolic enzymes and DNA (7) for organelle replication are stored here.

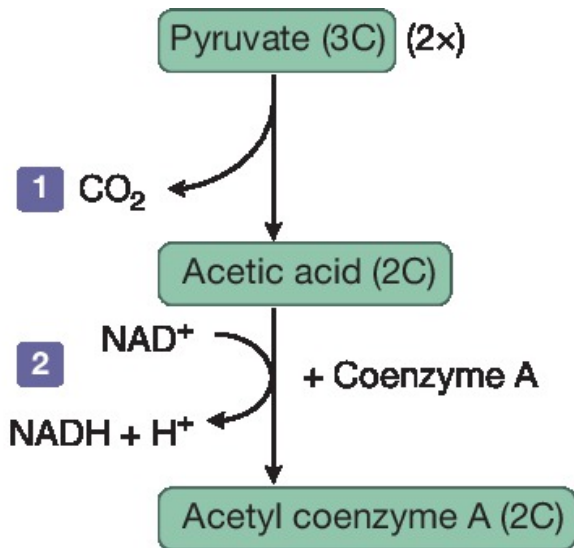
The fact that mitochondria contain their own DNA means that they are self-replicating. When a need for more ATP arises, mitochondria simply split in half and then grow to their former size. This property, discussed later, has specific implications for how an individual adapts to exercise training. It also explains why some cells have only a few mitochondria but others have thousands. Red blood cells are unique in that they contain no mitochondria.

Mitochondria tend to be located (**Figure 2.7C**) where they are

needed. Within muscle cells, they lie directly beneath the cell membrane (called sarcolemmal or subsarcolemmal mitochondria) and among the contractile elements (called interfibrillar mitochondria).

Stage II: Formation of Acetyl Coenzyme A

Stage II (**Figure 2.8**) is a very short metabolic pathway consisting solely of the conversion of pyruvate to acetyl CoA. No ATP is either used or produced directly. However, a pair of hydrogen atoms (because all reactions still happen twice) are removed and picked up by NAD^+ to be transferred to the electron transport chain.



Summary of the Formation of Acetyl CoA

- Does not directly utilize O₂ but must be aerobic
- Occurs in mitochondrial matrix
- No ATP produced
- 2 NADH + H⁺
- 2 CO₂
- 2 Acetyl CoA

Figure 2.8 Stage II: The Formation of Acetyl Coenzyme A.

The conversion of pyruvate to acetyl CoA takes place within the mitochondrial matrix (**Figure 2.7**). This conversion requires that the pyruvate be transported across the mitochondrial membranes via a specific carrier. No oxygen is used directly in

this stage, but these steps occur only in the presence of oxygen. As usual, enzymes catalyze each reaction.

The Steps of Stage II

Refer to the diagram in **Figure 2.8** for the two steps of Stage II (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983; Salway, 1994).

Step 1. Pyruvate is converted to acetic acid. In the process, one molecule of CO₂ is removed. This CO₂, as well as the CO₂ that will be formed in Stage III, diffuses into the bloodstream and is ultimately exhaled via the lungs.

Step 2. Acetic acid is combined with coenzyme A to form acetyl CoA. A **coenzyme** is a nonprotein substance derived from a vitamin that activates an enzyme. The conversion of pyruvate to acetyl CoA commits the pyruvate to Stages III and IV since there is no biochemical means of reconvertng acetyl CoA back to pyruvate.

Coenzyme A nonprotein substance derived from a vitamin that activates an enzyme.

Stage III: Krebs Cycle

The **Krebs cycle** is a cycle because it begins and ends with the same substance, called oxaloacetate (or oxaloacetic acid, abbreviated as OAA). The cycle is an eightstep process (see **Figure 2.9**) that actually constitutes two metabolic pathways: one pathway for steps 1–3 and the second pathway for steps 4–8.

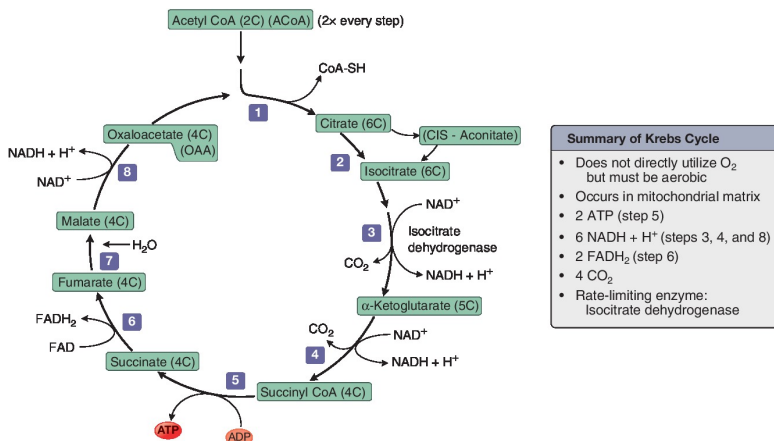


Figure 2.9 Stage III: The Krebs Cycle.

Krebs Cycle A series of eight chemical reactions that begins and ends with the same substance; energy is liberated for direct substrate-level phosphorylation of ATP from ADP and Pi; carbon dioxide is formed and hydrogen atoms removed and carried by NAD⁺ and FAD to the electron transport system; does not directly utilize oxygen but requires its presence.

No ATP is used in the Krebs cycle, and only one step (5) results in the substrate-level phosphorylation production of ATP. However, four steps (3, 4, 6, and 8) result in the removal of hydrogen atoms, which are picked up by either NAD⁺ or FAD. This result is critically important, because these are the hydrogen atoms that will provide the electrons for the electron transport system. Carbon dioxide is produced at steps 3 and 4. As with every step since the 6-carbon glucose molecule was split into two 3-carbon units in Stage I (step 4, **Figure 2.6**), every reaction and product is doubled.

All of the enzymes and hence all of the reactions for the Krebs cycle occur within the mitochondrial matrix, with one exception. The enzyme succinate dehydrogenase that catalyzes step 6 is located in the inner mitochondrial membrane.

The intermediate molecules as well as the preliminary

molecules, pyruvic acid and acetyl CoA, are keto acids. Although no oxygen is used directly, this stage requires the presence of oxygen.

The Steps of Stage III

The steps for the Krebs cycle (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983; Salway, 1994) are presented in **Figure 2.9**, which you should refer to while reading the discussion.

Step 1. The 2-carbon acetyl CoA combines with the 4-carbon molecule oxaloacetate (OAA) to form citrate or citric acid. This cycle is thus also known as the citric acid cycle. In the reaction, the coenzyme A (CoA-SH) is removed from acetyl CoA and is free to convert more pyruvate to acetyl CoA or to be used later in the cycle.

Step 2. The atoms of citric acid (citrate) are rearranged to become isocitrate. This rearrangement sometimes involves an intermediary substrate, cis-aconitate, being produced between citrate and isocitrate (shown in parenthesis is **Figure 2.9**). However, this step is not obligatory, and a single enzyme, aconitase, catalyzes both reactions (Newsholme and Leech, 1983).

Step 3. Two reactions occur. In the first, hydrogen atoms are removed and accepted by the carrier NAD^+ , forming $\text{NADH} + \text{H}^+$. In the second reaction, a CO_2 is removed, leaving the 5-carbon α -ketoglutarate.

Step 4. Step 4 is basically a repetition of step 3 in that a pair of hydrogen atoms are removed and picked up by NAD^+ and a CO_2 is also removed. In addition, the remaining structure is attached to coenzyme A (CoASH). The resultant succinyl CoA has four carbons, which will remain intact throughout the rest of the cycle.

Step 5. In this step, coenzyme A is displaced by a phosphate group, which in turn is transferred to a substance called guanosine diphosphate (GDP), resulting in guanosine triphosphate (GTP). GTP is equivalent to ATP in terms of energy and is labeled and counted as ATP in this book. This is the only step in the Krebs cycle that produces and stores energy directly.

As in glycolysis, this ATP is produced by substrate-level phosphorylation. The resulting intermediate is succinate.

Step 6. More hydrogen atoms are removed, but this time the carrier substance is FAD, forming FADH₂. Fumarate results.

Step 7. Water is added, converting fumarate to malate.

Step 8. A pair of hydrogen atoms is removed and accepted by NAD⁺. The remaining atoms once more make up oxaloacetate, and the cycle is ready to begin again.

The ATP produced in Stages I and III can be used immediately to provide energy for the cell. The CO₂ produced in Stage II diffuses into the bloodstream, is transported to the lungs, and is exhaled. Now, we will describe what happens to all of the hydrogen atoms that are carried as NADH + H⁺ and FADH₂.

Stage IV: Electron Transport and Oxidative Phosphorylation

The **electron transport system (ETS)** or respiratory chain is the final metabolic pathway in the production of ATP. It proceeds as a series of chemical reactions in the mitochondria that transfer electrons from the hydrogen atom carriers NAD⁺ and FAD to oxygen. Water is formed as a by-product, and the electrochemical energy released by the hydrogen ions is coupled to the formation of ATP from ADP and P_i. The chain itself consists of a series of electron (e⁻) carriers and proton pumps embedded in the inner membrane of the mitochondria. Most carriers are proteins or proteins combined with metal ions (such as iron, Fe) that attract e⁻. The major carriers are indicated in **Figure 2.10**. Four of these carriers are stationary: Complexes I, II, III, and IV. Three (I, III, and IV) are embedded into the inner membrane of the mitochondria, while Complex II is attached to the inner membrane on the matrix side. Cytochromes *b* and *c*₁ are part of Complex III, and cytochromes *a* and *a*₃ are part of Complex IV. Coenzyme Q and cytochrome *c* are mobile and diffuse through the inner membrane carrying electrons. Coenzyme Q moves from Complexes I and II to Complex III, and cytochrome *c* moves from Complex III to IV.

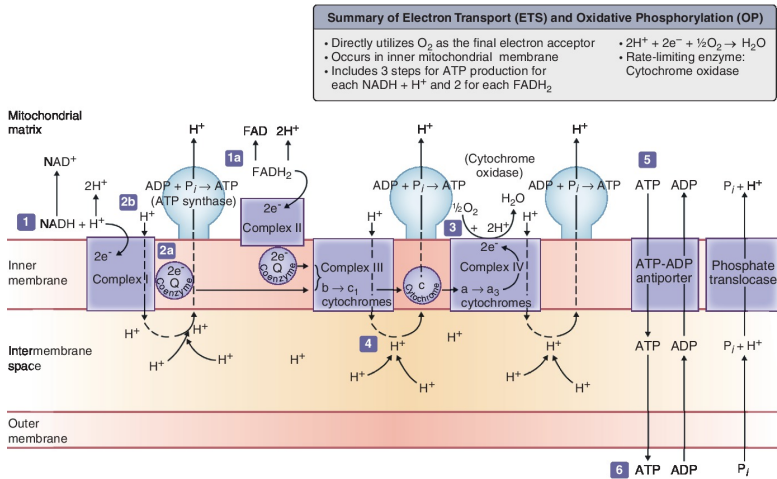


Figure 2.10 Stage IV: Electron Transport and Oxidative Phosphorylation.

The numbered steps are indicated in the *solid boxes*.

Electron Transport System (ETS) The final metabolic pathway, which proceeds as a series of chemical reactions in the mitochondria that transfer electrons from the hydrogen atom carriers NAD and FAD to oxygen; water is formed as a by-product; the electrochemical energy released by the hydrogen ions is coupled to the formation of ATP from ADP and P_i.

The H⁺ and e⁻ come from the breakdown of the hydrogen atoms released in Stages I, II, and III and transported to the electron transport chain by NAD⁺ and FAD:



The H⁺ are deposited first into the mitochondrial matrix and then move via the proton pumps into the intermembrane space. The e⁻ are shuttled along from one electron acceptor to the next (see **Figure 2.10**). The e⁻ move along in a series of oxidation-reduction reactions, because each successive carrier in the sequence has a greater affinity (force of chemical attraction) for

them than the preceding one. Oxygen has the greatest affinity of all for e^- and acts as the final e^- acceptor. The additional electrons on the oxygen give it a negative charge and attract the positively charged H^+ , thus forming water (**Figure 2.11**). Other H^+ move back from the intermembrane space to the matrix through the ball-and-stalk apparatus known as ATP synthase. The H^+ produced from the ETS bind to one component of the ATP synthase enzyme and then are released into the mitochondrial matrix. The process of binding and releasing H^+ causes the ATP synthase structure to turn. It is this turning motion of ATP synthase that allows for phosphorylation of ADP to ATP (Jonckheere et al., 2012).

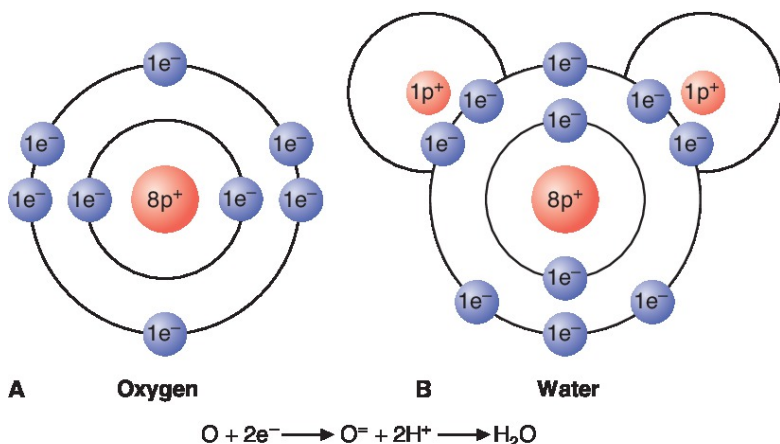


Figure 2.11 Oxygen as the Final Electron Acceptor.

A. The outer shell of oxygen has room for eight electrons but contains only six. Thus, it can accept two electrons at the end of electron transport. **B.** When oxygen accepts these two electrons, it then has a double negative charge (O^{2-}). Two hydrogen ions (H^+) are thus attracted, and water (H_2O) is formed.

Thus, the formation of ATP from ADP and P_i is coupled to the movement of H^+ and e^- through and along the electron transport chain. The movement of those ions releases energy that is harnessed when ADP is phosphorylated to ATP. Because

phosphate is added (phosphorylation) and the $\text{NADH} + \text{H}^+$ and FADH_2 are oxidized (electrons removed), the term oxidative phosphorylation (OP) is used to denote this formation of ATP. **Oxidative phosphorylation** is the process in which $\text{NADH} + \text{H}^+$ and FADH_2 are oxidized in the electron transport system and the energy released is used to synthesize ATP from ADP and P_i . Remember that the process of producing ATP directly in glycolysis and the Krebs cycle was called substrate-level phosphorylation because the P_i was transferred directly from phosphorylated intermediates to ADP without any oxidation occurring.

Oxidative Phosphorylation (OP) The process in which $\text{NADH} + \text{H}^+$ and FADH_2 are oxidized in the electron transport system and the energy released is used to synthesize ATP from ADP and P_i .

The Steps of Stage IV

Again, the series of steps (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983) presented in **Figure 2.10** are necessary so that the energy is preserved rather than released all at once and lost as heat, which would destroy tissue.

Step 1. $\text{NADH} + \text{H}^+$ arrives at Complex I, transfers the electrons (e^-) to the complex, and deposits the protons (H^+) into the mitochondrial matrix.

Step 1a. This step is really a variation and not a sequential step. If the original hydrogen carrier was FAD instead of NAD^+ , the transfer of electrons and protons occurs at Complex II instead of Complex I.

Step 2a. The electrons shuttle down the cytochromes, alternately causing the cytochromes to gain (become reduced) and lose (become oxidized) the electrons.

Step 2b. In this process, electrons also move across the width of the inner membrane, driving the proton pumps that shuttle H^+ from the matrix to the inner membrane space.

Step 3. Oxygen accepts the electrons. This increase in negative charge attracts hydrogen ions and causes the formation of water. **Figure 2.11** depicts this step graphically.

Step 4. The H^+ that have been transported into the intermembrane space create an electrochemical gradient. Since the drive is to equalize that gradient, the H^+ move back into the mitochondrial matrix through the ball-and-stalk complexes that are the enzyme ATP synthase. The H^+ bind to ATP synthase and then are released into the mitochondrial matrix. This binding and releasing of H^+ causes ATP synthase to turn and, along with the electrical current, is used to synthesize ATP from $ADP + P_i$, both of which are present in the matrix. This occurs three times if the carrier is $NADH + H^+$ and twice if the carrier is $FADH_2$.

Step 5. ATP moves into the intermembrane space through the ATP-ADP antiporter protein. For every ATP molecule exported, an ADP molecule is imported. P_i is moved into the mitochondrial matrix via the phosphate translocase protein along with one H^+ .

Step 6. In the final step, the ATP moves out of the mitochondria in exchange for the inward movement of ADP needed for the continual production of energy.

Figure 2.12 summarizes the process of cellular respiration just described and shows the interrelationships in a cell. As you study the figure, refer to the individual stage explanations as needed.

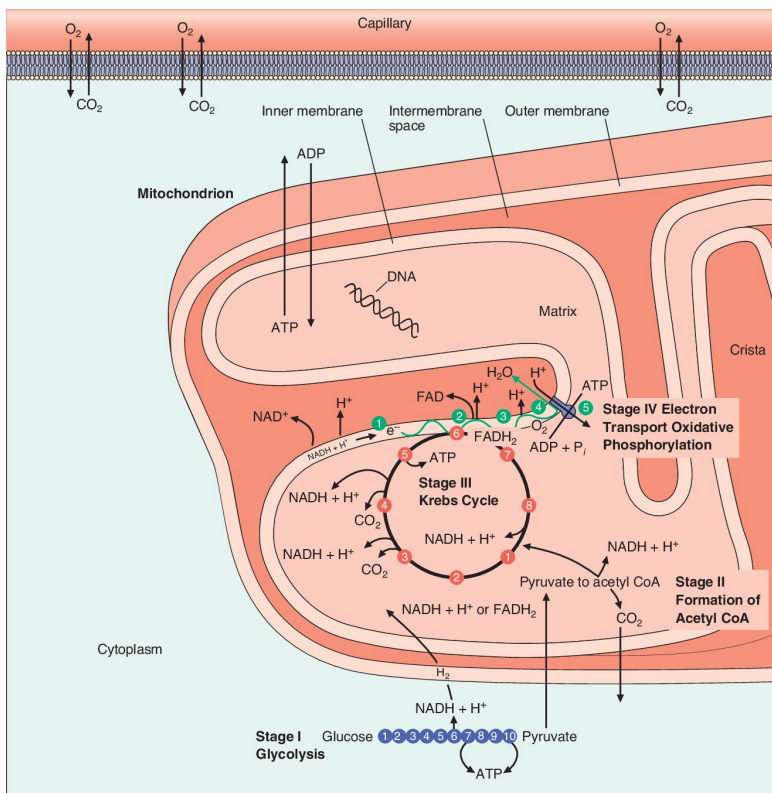


Figure 2.12 Cellular Respiration.

ATP Production from Carbohydrate

The number of ATP produced directly by substrate-level phosphorylation and the number of hydrogen atoms being carried by NAD^+ and FAD were described above for each stage. These numbers are summarized in **Table 2.1**. From this summary, we can compute the total yield of ATP from one molecule of glucose or glycogen.

TABLE 2.1 ATP Production from Carbohydrate

| Metabolic Process Stage | ATP | | | | Hydrogen Atoms and Carrier | |
|---------------------------|--------------|-----------------|-----------------|-----------------|---|---|
| | Heart Muscle | | Skeletal Muscle | | Heart Muscle | Skeletal Muscle |
| I. Glycolysis | +4 | +4 | +4 | +4 | 2 NADH + H ⁺ | 2 NADH + H ⁺ → 2 FADH ₂ |
| Glucose | -2 | -1 [†] | -2 | -1 [†] | | |
| Glycogen [†] | 2 | 3 | 2 | 3 | | |
| II. Pyruvate → acetyl CoA | 0 | 0 | 0 | 0 | 2 NADH + H ⁺ | 2 NADH + H ⁺ |
| III. Krebs cycle | 2 | 2 | 2 | 2 | 6 NADH + H ⁺ 2 FADH ₂ | 6 NADH + H ⁺ 2 FADH ₂ |
| IV. ETS/OP: | | | | | | |
| Hydrogen atoms from | | | | | | |
| Stage I | 5 | 5 | 3 [*] | 3 [*] | | |
| Stage II | 5 | 5 | 5 | 5 | | |
| Stage III | 18 | 18 | 18 | 18 | | |
| Total | | | | | | |
| Glucose | 32 | | 30 | | | |
| Glycogen | | 33 | | 31 | | |

*Owing to shuttle differences in crossing into mitochondrial membrane, these hydrogen atoms are actually carried by FAD in the mitochondria, reducing the ATP production.

†If glycogen, not glucose, is fuel.

Stage I (glycolysis) yields a gross production of 4 ATP but uses 2 ATP, yielding a net gain of 2 ATP from direct substrate-level phosphorylation, if the substrate is glucose. If the substrate is glycogen, only 1 ATP is used and the net gain is 3 ATP.

Two molecules of NADH + H⁺ are also produced. These hydrogen atoms are transported from the cytoplasm, where glycolysis has taken place, into the mitochondria, if the level of pyruvate is not too high and if there is enough oxygen to accept the electrons at the end of the ETS. However, the inner mitochondrial membrane is impermeable to NADH + H⁺, meaning that the membrane does not allow it to pass through. Therefore, a shuttle system must be employed.

Actually, two shuttle systems appear to operate—one in cardiac muscle and the other in skeletal muscle. The cardiac muscle shuttle system is called the *malate-aspartate shuttle*. It operates by the NADH + H⁺ in the cytoplasm giving up the hydrogens to malate, which carries them across the inner mitochondrial membrane. Here, mitochondrial NAD⁺ picks up the H⁺ and enters the ETS ([Newsholme and Leech, 1983](#)).

The skeletal muscle shuttle system is called the *glycerol-phosphate shuttle*. It operates by the NADH + H⁺ in the cytoplasm giving up the hydrogens to glycerol phosphate, which carries them into the inner mitochondrial membrane. Here, FAD picks up the H⁺ and enters the ETS. This shuttle predominates in

skeletal muscle (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983), although there is some evidence that the malate-aspartate shuttle may operate in Type I slow-twitch oxidative skeletal muscle (Houston, 1995).

Because of these different shuttle systems, in heart muscle, the ATP yield from the glycolytic hydrogens is higher than in skeletal muscle. In heart muscle, the shuttle releases H^+ to $NADH^+$, whereas in skeletal muscle, the shuttle releases H^+ to FAD. As detailed in step 4 above, the amount of ATP produced in ETS/OP depends on where the carrier enters the ETS, with $NADH + H^+$ resulting in more ATP produced than $FADH_2$. No differences between cardiac and skeletal muscle exist in the ATP production count for any of the remaining stages.

The counts for ATP produced during the oxidative phosphorylation are a little different from those in substrate-level phosphorylation. Theoretically and historically, because the H^+ moves through ATP synthase ball-and-stalk apparatus in three locations if deposited by $NADH + H^+$ and two locations if deposited by $FADH_2$, it was assumed that 3 ATP molecules were produced for each $NADH + H^+$ and 2 ATP molecules were produced for each $FADH_2$. The actual number is probably less. Biochemists are currently uncertain (Mougios, 2006) because the numbers of H^+ moving are not necessarily constant but may depend on the energy state of the cell. The best available estimate is that it takes 3 H^+ moving through ATP synthase to form 1 ATP. However, this actually involves 4 H^+ to compensate for the 1 H^+ that enters the mitochondrial matrix with P_i through the phosphate translocase (**Figure 2.10**). Thus, the movement of 4 H^+ results in 1 ATP. The number of H^+ expelled by the three complexes (I, III, and IV) is thought to be 4 at Complex I, 2 at Complex III, and 4 at Complex IV for a total of 10 H^+ per $NADH + H^+$. Dividing 10 H^+ by 4 H^+ for each ATP = 2.5 ATP per $NADH + H^+$. $FADH_2$ bypasses Complex I. Therefore, the total is only 2 H^+ at Complex III and 4 H^+ at Complex IV for a total of 6 H^+ . Dividing 6 H^+ by 4 H^+ per ATP = 1.5 ATP per $FADH_2$.

Stage II (the conversion of pyruvate to acetyl CoA) yields no substrate-level ATP, but it does produce 2 $NADH + H^+$. Since these molecules are already in the mitochondria, they directly

enter the ETS. This results in a yield of 5 ATP ($2 \text{ NADH} + \text{H}^+ \times 2.5$).

Stage III (the Krebs cycle) produces 2 ATP directly by substrate-level phosphorylation, $\text{NADH} + \text{H}^+$ at three steps, and FADH_2 at one step. Thus, ($3 \text{ NADH} + \text{H}^+ \times 2.5 = 7.5 \text{ ATP}$) + ($1 \text{ FADH}_2 \times 1.5 = 1.5 \text{ ATP}$). Since each step occurs twice for each 6-carbon glucose molecule, this yield must be doubled: $7.5 \text{ ATP} + 1.5 \text{ ATP} = 9 \text{ ATP} \times 2 = 18 \text{ ATP}$.

If we add all of these results for skeletal muscle when glucose is the fuel, we get:

| | |
|--------|--|
| 2 ATP | (substrate-level phosphorylation, glycolysis) |
| 3 ATP | ($2 \text{ NADH} + \text{H}^+ \rightarrow 2 \text{ FADH}_2 \times 1.5$, glycolysis) |
| 5 ATP | ($2 \text{ NADH} + \text{H}^+ \times 2.5$, Stage II) |
| 2 ATP | (substrate-level phosphorylation, Krebs Cycle) |
| 18 ATP | ($2 \text{ FADH}_2 \times 1.5 + 6 \text{ NADH} + \text{H}^+ \times 2.5$, Krebs Cycle EPS/OP) |
| 30 ATP | (for a total of 30 ATP for the aerobic oxidation of one molecule of glucose by skeletal muscle). |

Complete the [Check Your Comprehension 1](#).

CHECK YOUR COMPREHENSION 1

Determine the count of total ATP produced when the aerobic oxidation takes place in heart muscle and the initial fuel is glucose. Check your answer against the value given in **Table 2.1**. Next, do the same computations assuming that glycogen, not glucose, is the energy substrate. Again, check your answer against the value given in **Table 2.1**.

Fat Metabolism

Although the body may prefer to use carbohydrate as fuel from the standpoint of oxygen cost, the importance of fat as an energy

source should not be underestimated. Fat is found in many common foods. Fat, in the form of triglyceride (sometimes known as triacylglycerol), is the major storage form of energy in humans. Some triglyceride is stored within muscle cells (**Figure 2.13**), but the vast majority is deposited in adipose cells (**Figure 2.14**), which constitute at least 10–15% of the body weight of average young males and 20–25% of the body weight of average young females ([Malina and Bouchard, 1991](#)). Roughly half of this adipocyte storage occurs subcutaneously (under the skin). The remaining stores surround the major organs of the abdominothoracic cavity as support and protection. Triglycerides are turned over constantly in the body. Because body fat is turned over completely about every 3–4 weeks, no one is literally still carrying their “baby fat” ([Marieb and Hoehn, 2018](#)).

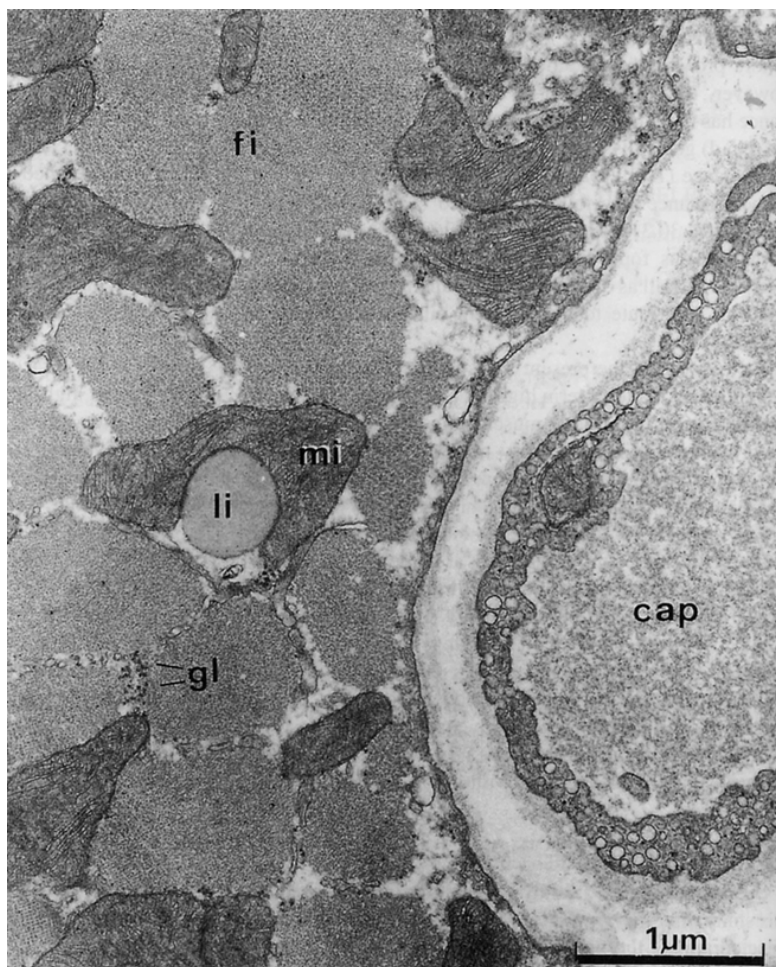


Figure 2.13 Electron Micrograph of Cross-Sectional Muscle Area (EM: 28,500 \times).

A lipid droplet is labeled as “li.” **Source:** Reprinted with permission from Vogt, M., A. Puntschart, H. Howald, B. Mueller, C. Mannhart, L. Gfeller-Tuescher, P. Mullis, & H. Hoppeler: Effects of dietary fat on muscle substrates, metabolism, and performance in athletes. *Medicine & Science in Sports & Exercise*. 35(6):952–960 (2003). Copyright ©2003 The American College of Sports Medicine.

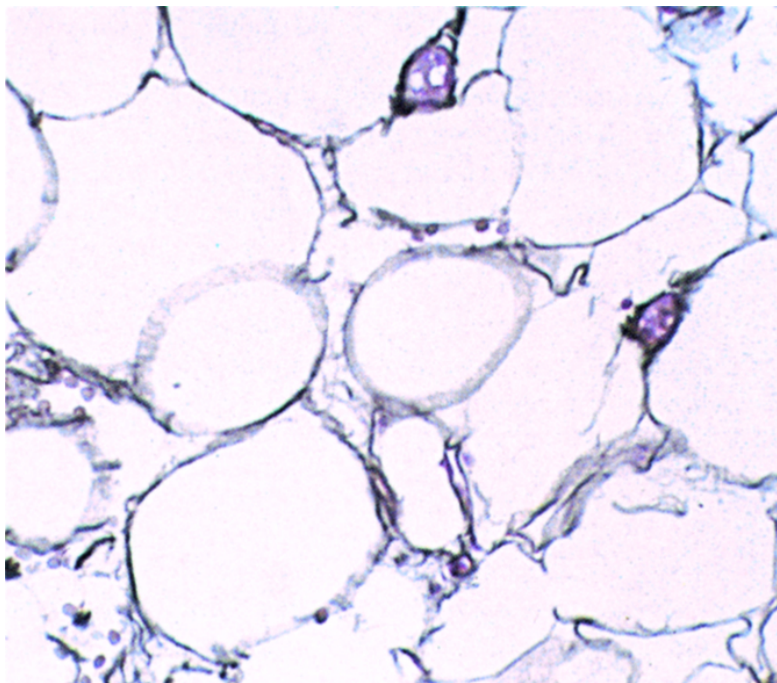


Figure 2.14 Triglyceride Stored in Adipose Tissue.

Source: Reprinted with permission from Mills, S. E.: *Histology for Pathologists* (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins (2007).

Fat is an excellent storage fuel for several reasons. First, fat is an energy-dense fuel yielding $9.13 \text{ kcal}\cdot\text{g}^{-1}$; both carbohydrate and protein yield slightly less than $4 \text{ kcal}\cdot\text{g}^{-1}$. The difference is due to the chemical structure of the substrates—specifically, the amount of oxidizable carbon and hydrogen. It is easy to appreciate the difference by looking at the chemical composition of the free fatty acid palmitate, which is $\text{C}_{16}\text{H}_{32}\text{O}_2$. This fatty acid has almost three times as much C and H, but only a third as much O as glucose ($\text{C}_6\text{H}_{12}\text{O}_6$). Remember that it is H atoms that donate the electrons (e^-) and protons (H^+) used during electron transport and oxidative phosphorylation.

Second, glycogen stores are relatively small in comparison to fat stores. A person can deplete stored glycogen in as little as 2 hours of heavy exercise or 1 day of bed rest, whereas fat supplies

can last for weeks, even with moderate activity. It is estimated that stored carbohydrates might provide 2,000 kcals of energy, whereas stored fat (even when lean) may provide 20,000–100,000 kcals of energy (Acheson et al., 1988). Although many North Americans and Europeans are concerned about having too much body fat, this storage capacity is undoubtedly important for survival of the species when food is not readily available.

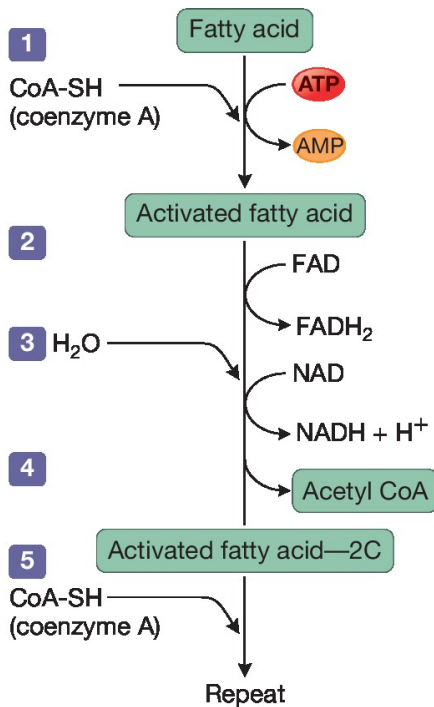
The triglycerides stored in adipose tissue must first be broken down into glycerol and free fatty acids before they can be used as fuel (see earlier **Figure 2.2**). One glycerol and three fatty acids make up a triglyceride. Seven fatty acids predominate in the body, but since three fatty acids combine with a glycerol to make up a triglyceride, $343 (7 \times 7 \times 7)$ different combinations are possible (Péronnet et al., 1987). Some common fatty acids include oleic acid, palmitic acid, stearic acid, linoleic acid, and palmitoleic acid.

Fatty acids may be saturated, unsaturated, or polyunsaturated. A saturated fatty acid has a chemical bonding arrangement that allows it to hold as many hydrogen atoms as possible. Thus, the term “saturated” means “saturated with hydrogen.” Unsaturated fatty acids have a chemical bonding arrangement with a reduced-hydrogen binding potential and therefore are unsaturated with respect to hydrogen atoms. Polyunsaturated means several bonds are without hydrogen.

The breakdown of triglycerides into glycerol and fatty acids is catalyzed by two enzymes: adipose triacylglycerol lipase (ATGL) and hormone-sensitive lipase (HSL) (Spriet, 2012). The glycerol is soluble in blood, but the free fatty acids (FFAs) are not. Glycerol can enter glycolysis in the cytoplasm (as 3-phosphoglycerate, the product of step 7 in **Figure 2.6**), but it is not typically utilized by muscle cells in this way (Newsholme and Leech, 1983; Péronnet et al., 1987). The direct role of glycerol as a fuel in the muscle cells during exercise is so minor that it need not be considered. However, glycerol can be converted to glucose by the liver but not in a fat cell because fat cells lack the enzyme glycerol kinase.

FFA must be transported in the blood bound to albumin. Specific receptor sites on the muscle cell membrane take up the FFA into the cell where movement occurs via specific transport proteins (Spriet, 2012). The FFA must then be translocated or

transported from the cytoplasm into the mitochondria. Once in the mitochondrial matrix, the FFA undergoes the process of beta-oxidation (**Figure 2.15**).



Summary of Beta Oxidation

- Does not directly utilize O₂, but must be aerobic
- Occurs in mitochondrial matrix
- 1 ATP used for activation, but since it is hydrolyzed to AMP, this is equal to 2 ATP being used
- No ATP produced directly
- 1 FADH₂ + 1 NADH + H⁺ produced for each pair of carbon atoms split off (which yields 4 ATP in ETS/OP)
- 1 Acetyl CoA (which yields 3 NADH + H⁺ + 1 FADH₂ + 1 ATP directly for a total of 10 ATP) produced for each pair of carbon atoms split off

Figure 2.15 Beta-Oxidation.

Beta-Oxidation

Beta-oxidation is a cyclic series of steps that breaks off successive pairs of carbon atoms from FFA, which are then used to form acetyl CoA. Remember that acetyl CoA is the common intermediate by which all foodstuffs enter the Krebs cycle and ETS/OP stage. The number of cycles depends upon the number of carbon atoms; most fatty acids have 14–24 carbons.

Beta-oxidation A cyclic series of steps that breaks off successive pairs of carbon atoms from FFA, which are then used to form acetyl CoA.

When there is an adequate supply of oxaloacetate to combine with, the fat-derived acetyl CoA enters the Krebs cycle and proceeds through electron transport and oxidative phosphorylation. **Figure 2.15** shows the steps of beta-oxidation, which are explained next.

As with glycolysis, ATP is used for activation; but unlike glycolysis, beta-oxidation produces no ATP directly by substrate-level phosphorylation.

The Steps of Beta-Oxidation

Step 1. The fatty acid molecule is activated by the breakdown of 1 ATP to AMP, releasing the energy equivalent of 2 ATP if broken down as it is normally to ADP. Concurrently, coenzyme A is added.

Step 2. FAD is reduced to FADH₂. The FADH₂ enters the electron transport chain and produces 1.5 ATP.

Step 3. A molecule of water is added, and NAD⁺ is reduced to NADH + H⁺. The NADH + H⁺ will enter the electron transport chain and produce 2.5 ATP. Steps 2 and 3, with the removal of the hydrogen atoms, account for the oxidation portion of the name for this process.

Step 4. The bond between the alpha (α) carbon (C2) and the beta (β) carbon (C3) is broken, resulting in the removal of two carbon atoms (C1 and C2), which are then used to form acetyl CoA. The cleavage of carbons at the site of the beta carbon explains why the process is called beta-oxidation.

Step 5. Steps 1–4 are repeated for each pair of carbons except the last, since the last unit formed is acetyl CoA itself. Therefore, the number of cycles that must be completed to oxidize the fat can be computed using the formula $n/2 - 1$, where n is the number of carbons. The acetyl CoA can enter the Krebs cycle and electron transport system (see earlier **Figures 2.2, 2.9, and 2.10**).

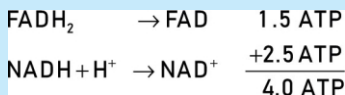
ATP Production from Fatty Acids

The number of ATP produced from the breakdown of fat depends on which fatty acid is utilized. The following example shows a calculation of ATP production for palmitate.

Example

Calculate the number of ATP produced from the breakdown of palmitate (palmitic acid). The steps in the calculation follow:

1. Palmitate is a 16-carbon fatty acid. Therefore, as noted in the previous step 5, it cycles through beta-oxidation $n/2 - 1 = 16/2 - 1 = 7$ times.
2. Each cycle produces 1 FADH₂ and 1 NADH + H⁺, as follows:



This reaction happens 7 times: $7 \times 4 = 28$ ATP.

3. Each cycle (7) plus the last step (1) produces acetyl

CoA, for a total of 8. Each acetyl CoA yields 1 ATP, 3 NADH + H⁺, and 1 FADH₂ in the Krebs cycle. The 3 NADH + H⁺ will produce 7.5 ATP, and the FADH₂ will produce 1.5 ATP in the electron transport system. Thus, 10 ATP are produced for each acetyl CoA, for a total of 8 acetyl CoA \times 10 ATP = 80 ATP.

4. Add the results of steps 2 and 3: 80 ATP + 28 ATP = 108 ATP produced.
5. 1 ATP was utilized in step 1 of beta-oxidation to activate the fatty acid. However, this ATP was broken down to AMP, not ADP; thus the equivalent of 2 ATP was used.
6. Subtracting the ATP used from the total ATP produced, we get 108 ATP - 2 ATP = 106 net ATP from palmitate.

Recall that three acids plus one glycerol make up a triglyceride. These calculations apply to a single specific fatty acid. Complete the [Check Your Comprehension 2](#).

CHECK YOUR COMPREHENSION 2

To check your understanding of the ATP yield from fatty acids, calculate the count for ATP produced from an 18-carbon fatty acid, such as stearate. Check your answer in [Appendix C](#).

Ketone Bodies and Ketosis

As mentioned earlier, in order for the acetyl CoA produced by beta-oxidation to enter the Krebs cycle, a sufficient amount of oxaloacetate is necessary. When carbohydrate supplies are sufficient, this is no problem, and fat is said to burn in the flame of carbohydrate. However, when carbohydrates are inadequate (perhaps as a result of fasting, prolonged exercise, diabetes mellitus, or a specific nutrition plan), oxaloacetate is converted to glucose. The production of glucose from noncarbohydrate sources under these conditions is necessary because some tissue, such as

the brain and nervous system, rely predominantly on glucose for fuel (Marieb and Hoehn, 2018).

When oxaloacetate is converted to glucose and is therefore not available to combine with acetyl CoA to form citrate, the liver converts the acetyl CoA derived from the fatty acids into metabolites called *ketones* or *ketone bodies*. Despite the similarity in the names, do not confuse ketones with keto acids (pyruvic acid and the Krebs cycle intermediates) (Figure 2.9). There are three ketones: acetoacetic acid, beta-hydroxybutyric acid, and acetone. All are strong acids. Acetone, at high concentrations, can give the breath a characteristic fruity smell.

Ketones themselves can be used as fuel by muscles, nerves, and the brain. If the ketones accumulate, fat is primarily being used as a fuel, and a condition called *ketosis* occurs. The high acidity of ketosis, sometimes called keto-acidosis, in some extreme circumstances (i.e., insulin insufficiency) can disrupt normal physiological functioning, especially acid-base balance, but not likely during nutritional ketosis (i.e., low carbohydrate diet). Ketosis is more likely to result from a purposeful low carbohydrate diet, an inadequate diet (as in anorexia nervosa), or diabetes than from prolonged exercise, since the muscles will use the ketones as fuel. During exercise, aerobically trained individuals can utilize ketones more effectively than can untrained individuals.

Protein Metabolism

Proteins are present in many food sources. Proteins are large molecules consisting of varying combinations of amino acids linked together. Approximately 20 amino acids occur naturally (Kapit et al., 1987). Because there are so many ways these amino acids can combine, the number of possible proteins is almost infinite. Like carbohydrates and fats, amino acids contain atoms of carbon, oxygen, and hydrogen. In addition, they may include sulfur, phosphorus, and iron. All amino acids have in common an amino group containing nitrogen (NH_2).

Proteins are extremely important in the structure and function of the body. Among other things, they are components of

hemoglobin, contractile elements of the muscle, hormones, fibrin for clotting, tendons, ligaments, and portions of all cell membranes. Because proteins are so important in the body, the constituent amino acids are used predominantly as building blocks, not as a source of energy.

However, amino acids can be, and in certain instances are, used as a fuel source. When amino acids are used as a fuel source, muscles appear to preferentially, but not exclusively, utilize the group of amino acids known as branched-chain amino acids (BCAA), that is, leucine, isoleucine, and valine. In this situation, as with carbohydrate and fat metabolism, the final common pathways of the Krebs cycle, electron transport, and oxidative phosphorylation are utilized. The site of entry into the metabolic pathways varies, as shown in **Figure 2.2**, with the specific amino acid.

Six amino acids can enter metabolism at the level of pyruvic acid, 8 at acetyl CoA, 4 at alpha-ketoglutarate, 4 at succinate, 2 at fumarate, and 2 at oxaloacetate. All of these intermediates except acetyl CoA are, in turn, converted to pyruvate before being used to produce energy. The acetyl CoA is used directly in the Krebs cycle and electron transport, as previously described.

Transamination and Oxidative Deamination

Before amino acids can be used as a fuel and enter the pathways at any place, the nitrogen-containing amino group (the NH_2) must be removed. It is removed by the process of transamination and sometimes by oxidative deamination (Frisell, 1982; Lehninger, 1971; Marieb and Hoehn, 2018; Mougios, 2006; Newsholme and Leech, 1983). These processes are summarized in **Table 2.2**.

TABLE 2.2 Transamination and Oxidative Deamination

| Transamination | Oxidative Deamination |
|--|--|
| Generalized | Generalized |
| Amino acid (1) + keto acid (1) \leftrightarrow amino acid (2) + keto acid (2) The NH_2 group from amino acid (1) is transferred to keto acid (1), forming a different amino acid (2) and a different keto acid (2) | Amino acid \leftrightarrow keto acid + NH_3 The NH_2 group is removed from an amino acid, forming a keto acid and ammonia |
| Most common | Most common |
| Any 1 of 12 different amino acids + α -ketoglutarate \leftrightarrow glutamate + keto acid | Glutamate + H_2O + NAD^+ \leftrightarrow NH_3 + α -ketoglutarate + $\text{NADH} + \text{H}^+$ |
| Specific example | Fate of products |
| Glutamate + pyruvate \leftrightarrow alanine + α -ketoglutarate | <ol style="list-style-type: none"> 1. $\text{NADH} + \text{H}^+$ enters the electron transport system 2. α-Ketoglutarate is a Krebs cycle intermediate 3. NH_3 (ammonia) is removed in urine. The urea cycle is $2\text{NH}_3 + \text{CO}_2 \rightarrow \text{NH}_2\text{CONH}_2 \text{ (urea)} + \text{H}_2\text{O}$ |

All but two amino acids appear to be able to undergo transamination. **Transamination** involves the transfer of the NH_2 amino group from an amino acid to a keto acid. Remember that keto acids include pyruvic acid, acetyl CoA, and the Krebs cycle intermediates. This process occurs in both the cytoplasm and the mitochondria, predominantly in muscle and liver cells. Transamination results in the formation of a new amino acid and a different keto acid. The most frequent keto acid acceptor of NH_2 is α -ketoglutarate, with glutamate being the amino acid formed (Marieb and Hoehn, 2018; Mougios, 2006).

Transamination The transfer of the NH_2 amino group from an amino acid to a keto acid.

Two fates for glutamate are shown in **Table 2.2**. In the first, glutamate is transaminated to alanine, another amino acid. Alanine, in turn, can be converted to glucose in a process called gluconeogenesis (Frisell, 1982; Marieb and Hoehn, 2018). **Gluconeogenesis** is the creation of glucose in the liver from noncarbohydrate sources, particularly glycerol, lactate or pyruvate, and alanine.

Gluconeogenesis The creation of glucose in the liver from noncarbohydrate sources, particularly glycerol, lactate or pyruvate, and alanine.

In the second process, glutamate undergoes oxidative deamination. In *oxidative deamination*, the NH_2 amino group is removed from the amino acid forming a keto acid and ammonia. That is, the oxidized form of NAD (NAD^+) is reduced ($\text{NADH} + \text{H}^+$), the amino group (NH_2) is removed, and NH_3 is formed. NH_3 is ammonia, which in high concentrations in the body is extremely toxic. The dominant pathway for NH_3 removal is by conversion to urea in the liver (via the urea cycle) and excretion in urine by the kidneys. Oxidative deamination is used much less frequently than transamination.

ATP Production from Amino Acids

Because the amino acid derivatives are ultimately utilized as pyruvate or acetyl CoA, the ATP production count from the amino acids is the same as for glucose from that point on, except that it is not doubled (Péronnet et al., 1987). Refer to **Figure 2.6** to remind yourself why the double count occurs with glucose and to **Figure 2.2** to see amino acids entering the pathways denoted next.

| | | |
|------------------------------------|-----------------------|-----------------|
| Pyruvate \rightarrow acetylCoA = | 1 NADH + H^+ | 2.5 ATP |
| Krebscycle | | 1 ATP |
| ETS/OP | 3 NADH + H^+ | 7.5 ATP |
| | 1 FADH_2 | 1.5 ATP |
| | | <u>12.5 ATP</u> |

This is the first time we have seen a total indicating a fraction of ATP. Interpret this as an average, not literally. ATP molecules do not exist in halves. Acetyl CoA would produce 10 ATP, because the $\text{NADH} + \text{H}^+$ production from pyruvate to acetyl CoA would be the only step missing.

The Regulation of Cellular Respiration and ATP Production

Intracellular Regulation

Intracellularly, the production of ATP—and, hence, the flow of substrates through the various metabolic pathways—is regulated predominantly by feedback mechanisms. Again, each step in each metabolic pathway is catalyzed by a specific enzyme. At least one of these enzymes in each pathway can be acted on directly by other chemicals in the cell and, as a result, increases or decreases its activity. Such an enzyme is called a *rate-limiting enzyme*, and the other factors that influence it are called modulators. When the rate-limiting enzyme is inhibited, every step in the metabolic pathway beyond that point is also inhibited. When the rate-limiting enzyme is stimulated, every step in the metabolic pathway beyond that point is also stimulated.

The primary rate-limiting enzyme in glycolysis is phosphofructokinase (PFK), the enzyme that catalyzes step 3. PFK is stimulated, and subsequently, the rate of glycolysis increased, by modulators such as ADP, AMP, P_i , and a rise in pH. ADP, AMP, and P_i modulators are the result of the breakdown of ATP. This is an example of a positive-feedback system in which the by-product of the utilization of a substance stimulates a greater production of that original substance. Conversely, PFK is inhibited, and subsequently, the rate of glycolysis decreased, by the modulators ATP, CP, citrate (a Krebs cycle intermediate), FFA, and a drop in pH. Each of these modulators signals that sufficient substances exist to supply ATP. This is an example of a negative-feedback system in which the formation of a product or other similarly acting product inhibits further production of the product (Newsholme and Leech, 1983).

The primary rate-limiting enzyme in the Krebs cycle is isocitrate dehydrogenase (ICD), which catalyzes step 3. ICD is stimulated by ADP, P_i , and calcium (positive feedback), and is inhibited by ATP (negative feedback) (Newsholme and Leech, 1983).

Cytochrome oxidase—which catalyzes the transfer of electrons to molecular oxygen, resulting in the formation of water—is the rate-limiting enzyme for the electron transport system. It is stimulated by ADP and P_i (positive feedback) and is inhibited by ATP (negative feedback) (Newsholme and Leech, 1983).

A pattern in the modulators is readily evident, with ATP, ADP, and P_i being universally important. When ATP is present in

sufficient amounts to satisfy the needs of the cell and to provide some reserve in storage, there is no need to increase its production. Thus, key enzymes in the metabolic pathways are inhibited. However, when muscle activity begins and ATP is broken down into ADP and P_i , these by-products stimulate all the metabolic pathways to produce more ATP so that the muscle contractions can continue.

Extracellular Regulation

During exercise, metabolic processes must provide ATP for energy and maintain blood glucose levels at near-resting values for the proper functioning of the entire organism. This is because the brain and nervous tissue must have glucose as a fuel. One of the ways of maintaining glucose levels is by the process of gluconeogenesis.

Gluconeogenesis

As defined earlier, gluconeogenesis is the creation (–genesis) of new (–neo) glucose (gluco-) in the liver from noncarbohydrate sources. The primary fuel sources for gluconeogenesis are glycerol, lactate or pyruvate, and alanine. Glycerol is released into the bloodstream when triglycerides are broken down (**Figure 2.16A**).

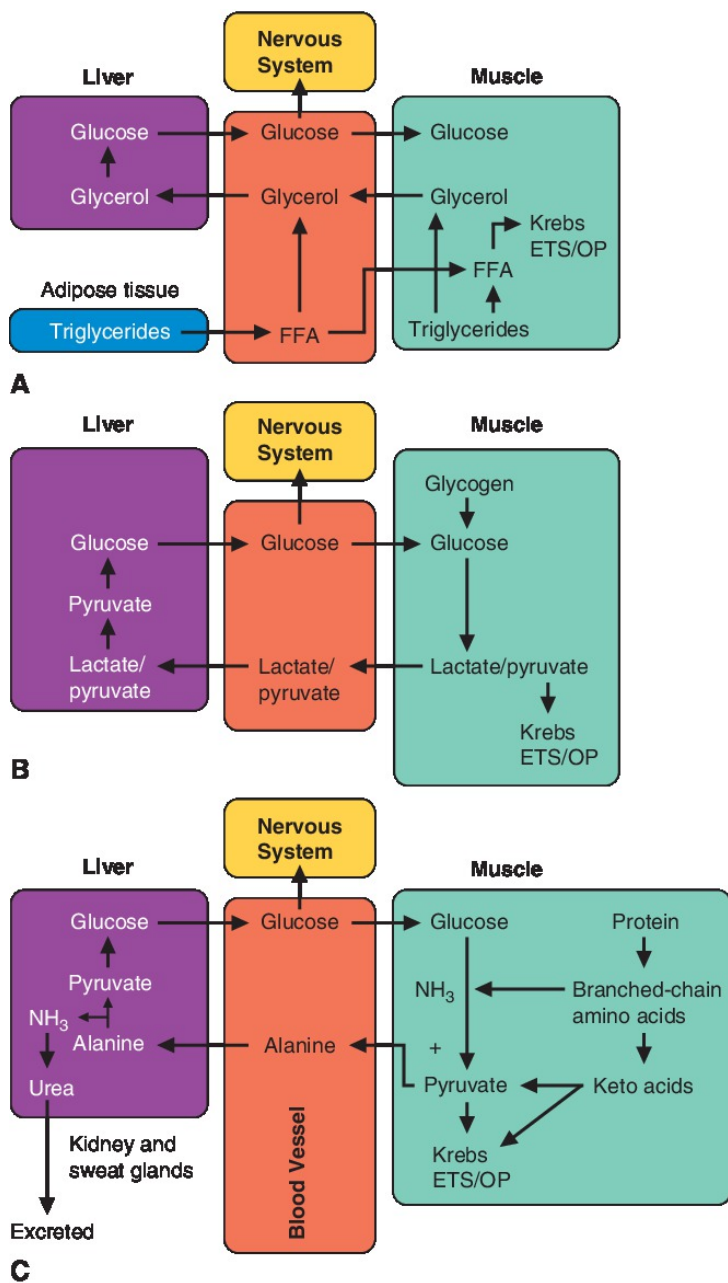


Figure 2.16 Gluconeogenesis.

A. Glycerol-glucose cycle. **B.** Cori cycle. **C.** Felig cycle.

Pyruvate is the end product of glycolysis. The majority of the pyruvate is converted to acetyl CoA and enters the Krebs cycle and electron transport system. However, a small portion diffuses out of the muscle cell and into the bloodstream (Péronnet et al., 1987). Still another portion of the pyruvate is converted to lactate, which also diffuses into the bloodstream. About 10 times as much lactate as pyruvate diffuses out of the muscle cells (Figure 2.16B).

Alanine is formed by transamination when the amino group from one amino acid (preferentially, the BCAA or an amino acid derived from glutamate) is transferred to pyruvate (Figure 2.16C). Note that in the liver, alanine is first reconverted to pyruvate (freeing the NH₃ to enter the urea cycle and be excreted) before being converted to glucose.

In all cases, the conversion to glucose takes place in the liver. For each gram of glucose produced, 1.02 g of glycerol, 1.43 g of pyruvate, 1.23 g of lactate, or 1.45 g of alanine is utilized (Péronnet et al., 1987).

The glycerol-glucose cycle has no special name, but the pyruvate/lactate-glucose cycle is known as the *Cori cycle*, and the alanine-glucose cycle is called the *Felig cycle*. The Cori and Felig cycles help maintain normal blood glucose levels so that the brain, nerves, and kidneys as well as the muscles may draw from this supply.

Neurohormonal Coordination

The regulation of blood glucose levels, including gluconeogenesis, is governed jointly by the autonomic nervous system (particularly the sympathetic division) and the endocrine system, which function in a coordinated fashion. Figure 2.17 shows this integration of the two control systems for the regulation of energy production in response to exercise. Chapter 21 provides a comprehensive explanation of the functioning of the neurohormonal system.

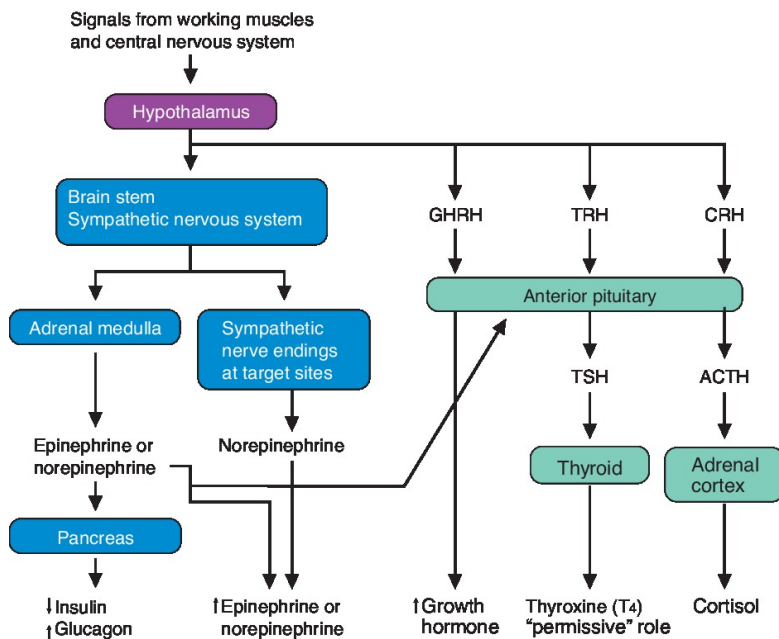


Figure 2.17 Extracellular Neurohormonal Regulation of Metabolism.

TRH, thyroid-releasing hormone; ACTH, adrenocorticotrophic hormone; GHRH, growth hormone–releasing hormone; CRH, corticotrophin-releasing hormone; TSH, thyroid-stimulating hormone; ↓, decrease; ↑, increase.

Sources: Bunt (1986); Marieb and Hoehn (2018); Van de Graaff and Fox (1989).

When exercise begins, signals from the working muscles and motor centers in the central nervous system bring about a neurohormonal response mediated through the hypothalamus. The hypothalamus stimulates both the anterior pituitary and the sympathetic nervous system. The neurotransmitter norepinephrine is released directly from sympathetic nerve endings, and the hormones epinephrine and norepinephrine are released from the adrenal medulla. Epinephrine and norepinephrine both act directly on fuel sites and stimulate the anterior pituitary and pancreas. As a result of the dual stimulation of the anterior pituitary, hormones are released—

namely, growth hormone, cortisol, and thyroxine. The release of insulin is inhibited from the pancreas, but glucagon release is stimulated (Bunt, 1986; Galbo, 1983). In general, there are three results (see also Table 2.3):

| TABLE 2.3 Hormonal Regulation of Metabolism during Exercise | | | | |
|---|----------|--------------------------------|-----------------|----------|
| Effect | Hormone | | | |
| | Glucagon | Epinephrine and Norepinephrine | Growth Hormone* | Cortisol |
| Glucose uptake and utilization | ↓† | | ↓† | ↓† |
| Glycogen breakdown (glycogenolysis) | ↑ | ↑ | ↑ | ↑ |
| Glycogen formation (glycogenesis) | ↓ | ↓ | ↑ | ↑ |
| Gluconeogenesis | ↑ | ↑ | ↑ | ↑ |
| FFA storage (lipogenesis) | ↓ | | ↓ | ↓ |
| FFA mobilization | ↑ | ↑ | ↑ | ↑ |
| Amino acid transport and uptake | | | ↑ | ↑ |
| Protein breakdown | | | | ↑ |

*Heavy exercise.

†Nonactive cells.

↑, Increases; ↓, Decreases.

1. An increase in the mobilization and utilization of free fatty acid stores from extramuscular (adipose tissue) and intramuscular stores
2. An increase in the breakdown of extramuscular (liver) and intramuscular stores of glycogen and the formation of glucose from noncarbohydrate sources (gluconeogenesis) in the liver
3. A decrease in the uptake of glucose into the nonworking cells

A two-tier system of hormonal involvement appears to operate on the basis of either a fast or slow time of response. The catecholamines (epinephrine and norepinephrine) and glucagon react to an increase in muscular activity in a matter of seconds to minutes. Both assist in all three metabolic functions listed above, although not equally. Epinephrine’s influence on fat metabolism is far greater than its effect on glucose metabolism (Hall and Hall, 2021).

The actions of the catecholamines and glucagon are backed up

by growth hormone and cortisol, respectively. The response of growth hormone and cortisol may take hours to become maximal. Thus, these latter two hormones are probably most important for carbohydrate conservation during long-duration activity. This explains why growth hormone and cortisol can bring about glycogen synthesis, while glucagon and the catecholamines cannot (Hall and Hall, 2021).

Insulin secretion is suppressed during exercise. This decrease in insulin and the increase in glucagon, growth hormone, and cortisol cause a decrease in the glucose uptake into cells other than the working muscles. The available glucose is thus spared for the working muscles and the nervous system.

During exercise, the working muscles become highly permeable to glucose despite the lack of insulin. As previously noted, the muscle contraction itself stimulates GLUT-4 translocation to the cell surface for the movement of glucose into the working muscle (Houston, 1995; Sato et al., 1996). The contractile process also helps to break down the intramuscular stores of glycogen and triglycerides (Galbo, 1983; Hall and Hall, 2021).

The increases in protein breakdown and in amino acid transport caused by growth hormone and cortisol are important in providing protein precursors for gluconeogenesis. The fact that these hormones are slow responders also explains why protein is not used in significant amounts except in long-duration activity.

Because the mobilization of free fatty acids involves the breakdown of triglycerides, glycerol is also released and made available for gluconeogenesis. Note that an accumulation of the other gluconeogenic precursor, lactate, inhibits free fatty acid release from adipose tissue. Therefore, gluconeogenesis serves a dual purpose in helping to provide glucose and reducing the levels of lactate so that it does not interfere as much with fat utilization.

Thyroxine itself has a number of metabolic functions. However, its role in exercise metabolism appears to be one of potentiation or permissiveness. That is, it helps to create an environment in which the other metabolic hormones can function more effectively, rather than having any major effect itself.

FOCUS ON APPLICATION

Metabolic Pathways: The Vitamin Connection

Many individuals mistakenly believe that vitamins are a source of energy for the body. Although this is not true, vitamins do participate in many of the chemical reactions described in this chapter that convert the potential energy of food sources into ATP. Your body cannot produce energy without vitamins. In general, the ingestion of a well-balanced diet (the fuel source) provides the needed vitamins. Conversely, the ingestion of excessive amounts of vitamins does not increase the activity of the enzymes for which the vitamins act as coenzymes. Listed in the table are the six vitamins that are particularly important for energy metabolism, their actions, and selected dietary sources. Check to see that you are getting all of these in your diet.

Source: VanderBeek (1985).

| Vitamin | Function in Metabolism | Selected Dietary Sources |
|---|---|--|
| B ₁ (thiamine) | Coenzyme involved in the conversion of pyruvate to acetyl CoA during carbohydrate metabolism | Lean meats, eggs, whole grains, leafy green vegetables, legumes |
| B ₂ (riboflavin) | Basis of coenzyme FAD, which serves as hydrogen acceptor in the mitochondria | Milk, eggs, lean meat, green vegetables |
| B ₃ (niacin or nicotinamide) | Basis of coenzyme NAD, which is the primary hydrogen carrier in both the cytoplasm and mitochondria | Lean meat, fish, legumes, whole grains, peanuts |
| B ₆ (pyridoxine) | Functions as coenzyme in amino acid transamination reactions; required coenzyme for glycogenolysis action of the enzyme phosphorylase | Meat, poultry, fish, white and sweet potatoes, tomatoes, spinach |
| Pantothenic acid | Functions as coenzyme A in the formation of acetyl CoA from pyruvate | Meat, legumes, whole grains, eggs |
| Biotin | Essential as coenzyme for a number of enzymes in the Krebs cycle and gluconeogenesis | Eggs, legumes, nuts |

Source: [VanderBeek \(1985\)](#).

The level of response and reaction to all of these hormones depends on the following:

1. The type, duration, and intensity of the exercise; relative intensity appears to be more important than the absolute workload.

2. The nutritional status, health, training status, and fiber-type composition of the exerciser.
3. The size of the fuel depots, the state of the hormone receptors, and the capacity of the involved enzymes (Galbo, 1983).

Fuel Utilization at Rest and during Exercise

Preceding sections explain how each of the major fuel sources—fats, carbohydrates, and proteins—are converted to ATP energy for the human body. At rest, it has been estimated that fats contribute 41–67%, carbohydrates 33–42%, and proteins from just a trace to 17% of the total daily energy requirements of the human body (Lemon and Nagel, 1981).

During exercise, various forms of each fuel are utilized to supply working muscles with the additional ATP energy needed to sustain movement. To understand which energy sources are used during exercise, it is first necessary to know approximately how much of each energy substrate is available. **Table 2.4** shows the tissue fuel stores in an average adult male who weighs 65 kg, has 8.45 kg (13%) body fat, and 30 kg of muscle. Protein stores are omitted because the major tissue storage of protein is in the muscles, and complete degradation (breakdown) of muscle to sustain energy metabolism is neither realistic nor desirable under normal circumstances.

TABLE 2.4 Tissue Fuel Stores in Average Adult Males (Body Weight = 65 kg, Percent Body Fat = 13)

| Tissue Fuel | Total Energy ^a | | | Estimated Period Fuel Would Supply Energy ^a | | |
|--|---------------------------|---------------------|---------------------|--|-----------------------|------------------------|
| | g | kJ | kcal | Basal ^b (d) | Walk ^b (d) | Run ^b (min) |
| Triglycerides ^c (fatty acids) | 8,450 | 322,789 | 77,150 | 47.46 | 11.25 | 5,223 |
| Liver glycogen ^d | 80 | 1,275 | 305 | 0.19 | 0.05 | 20.7 |
| Muscle glycogen (28.25 kg muscle ^e) | 425 | 7,384 ^{**} | 1,743 ^{**} | 1.07 | 0.25 | 118 |
| Circulating glucose (blood + extracellular) | 20 | 319 | 76 | 0.05 | 0.01 | 5.2 |

*Conversion factors utilized: glucose, $3.81 \text{ kcal}\cdot\text{g}^{-1}$; fatty acid, $9.13 \text{ kcal}\cdot\text{g}^{-1}$; $\text{kcal} \times 4.184 = \text{kJ}$ (Bursztein et al., 1989; Péronnet et al., 1987).

†Calculations assume that each fuel is the only fuel utilized. In reality, the fuels are utilized with mixtures that depend on the type, intensity, and duration of the activity and training, fiber-type proportions, and nutritional status of the exerciser.

‡Kilocalorie cost of exercise: basal $0.174 \text{ kcal}\cdot\text{kg}^{-1}\cdot 10 \text{ min}^{-1}$; walking at $3.5 \text{ mi}\cdot\text{hr}^{-1} = 0.733 \text{ kcal}\cdot\text{kg}^{-1}\cdot 10 \text{ min}^{-1}$; running at $8.7 \text{ mi}\cdot\text{hr}^{-1}$ (6:54 mi) = $2.273 \text{ kcal}\cdot 10 \text{ min}^{-1}$ (Consolazio et al., 1963).

§65 kg body weight $\times 0.13$ (fraction of body fat) = 8.45 kg fat.

||Liver equals approximately 2.5% body weight. Normal glycogen content is $50 \text{ g}\cdot\text{kg}^{-1}$; extracellular glucose includes the portion in liver available to circulation (Péronnet et al., 1987).

¶Weight of the muscle is approximately equal to half the lean body mass.

**Muscle fatty acid values would equal this value because 1 kg of muscle contains approximately 13.5 g fatty acid and 1.5 g glycerol. The triglyceride value includes the muscle store (Péronnet et al., 1987).

Sources: Based on Kapit et al. (2000); Newsholme and Leech (1983).

Several important points should be noted in **Table 2.4**. First, triglycerides provide the greatest source of potential energy (77,150 kcal is enough energy to sustain life for ~47 days or to walk ~771 miles or 1,240 km!). Second, comparatively, very little carbohydrate is actually stored. In fact, the human body's stores of carbohydrate are only enough to support life for $1\frac{1}{4}$ days, if all three sources (muscle glycogen, liver glycogen, and circulating glucose) are added together. Thus, carbohydrate replenishment on a regular basis is essential. Third, exercise has a major impact on how long each fuel can supply energy.

Table 2.4 assumes that each fuel is acting independently, which in fact never happens. **Table 2.5 and 2.6** provide more realistic views of what actually happens during exercise. **Table**

2.5 depicts the relative utilization of each substrate source based on the type, intensity, and duration of the activity. Very-short-duration, very-high-intensity dynamic activity and static contractions are special cases that rely predominantly on energy substrates stored in the muscle fibers—that is, ATP-PC and glycogen (which can quickly be broken down into ATP to provide energy).

TABLE 2.5 Relative Degree of Fuel Utilization in Muscle for Various Types of Exercise

| Fuel | Rest | Exercise Condition | | | | |
|-------------------------------|------------|--|---|---|---|--|
| | | Very-High-Intensity, Very-Short-Duration (<3 min), and Static Contractions | High-Intensity (80–85% max), Short-Duration (<40 min) | High-Intensity (70–80% max), Moderate-Duration (40–150 min) | Moderate-Intensity (60–70% max), Long-Duration (>150 min) | Low-Intensity (<50% max), Long-Duration (>150 min) |
| Muscle glycogen | Negligible | High | High | High | Moderate | Low |
| Liver glycogen/ blood glucose | Moderate | Negligible | High | High | Moderate | Moderate |
| Free fatty acid (FFA) | Moderate | Negligible | Low | Moderate | High | High |
| Amino acid | Low | Negligible | Negligible | Low | Low | Low |

Sources: Based on Felig and Wahren (1975); Pernow and Saltin (1971).

TABLE 2.6 Fuel Utilization by 70-kg Male Runner at Different Distances and Levels of Performance

| | 10 km (6.2 mi) | | 42.2 km (26.2 mi) | |
|---------------------------------------|----------------|---------|-------------------|---------|
| | Fast | Slow | Fast | Slow |
| Performance (h:min:sec) | 0:30:00 | 1:00:00 | 2:21:00 | 5:12:00 |
| Circulating glucose (g) | 9.7 | 13.4 | 120.5 | 217.8 |
| Muscle glycogen (g) | 149.0 | 134.0 | 403.0 | 69.0 |
| Liver glycogen (g) | 20.1 | 10.9 | 73.7 | 103.9 |
| Fatty acids (g) | 7.0 | 12.0 | 104.0 | 229.0 |
| Branched-chain amino acids (g) (BCAA) | 0.7 | 0.9 | 15.7 | 48.5 |

Sources: Péronnet et al. (1987).

The other exercises listed in **Table 2.5** are assumed to be predominantly dynamic and to a large extent aerobic (utilizing oxygen). In general, the lower the intensity, the more important fat is as a fuel; the higher the intensity, the more important carbohydrates are as fuel. At higher exercise intensities, fat

utilization is compromised because of decreased blood flow to adipose tissue, decreased release of FFA into the blood stream to be delivered to skeletal muscle, decreased breakdown of intramuscular triglycerides, and decreased transport of FFA into skeletal muscle mitochondria, in part because of reductions in muscle pH ([Holloway and Spriet, 2012](#)). Duration has a similar effect: the shorter the duration, the more important carbohydrates are as a fuel, with fat being utilized more and more as the duration lengthens. Fats come into play over the long term because the glycogen stores can and will be depleted. Indeed, long-duration activities often exhibit a three-part sequence in which muscle glycogen, blood-borne glucose (including glucose that has been broken down from liver glycogen and glucose that has been created by gluconeogenesis), and fatty acids successively predominate as the major fuel source. Protein may account for 5–15% of the total energy supply in activities lasting more than an hour ([Felig and Wahren, 1975](#); [Lemon and Nagel, 1981](#); [Pernow and Saltin, 1971](#)).

FOCUS ON RESEARCH

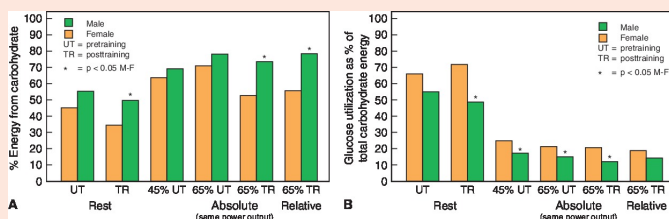
Sex Differences in Substrate Utilization

The data presented in [Table 2.5 and 2.6](#) show variations not only in the reliance on carbohydrate, fat, and protein substrates based on exercise intensity but also among the various components of carbohydrate used as a fuel (glucose, and muscle and liver glycogen). Data from this study by [Friedlander et al. \(1998\)](#) demonstrate that there might also be meaningful variations among fuel substrates between the sexes. Substrate utilization in young adult males and females

was determined at rest and at 45% $\dot{V}O_2$ peak and 65% $\dot{V}O_2$ peak exercise intensities on a cycle ergometer. Although only those comparisons marked with an asterisk (*) in the accompanying graphs attained statistical significance, the overall pattern seen in panel (A) is that carbohydrates provided a higher percentage of the total energy supply for

males than for females. Conversely, the pattern in panel (B) shows that the females used relatively more glucose than other carbohydrate sources (presumably glycogen and lactate) than did the males. Both of these trends were true at rest before (UT) and after (TR) training, at the two submaximal power outputs pretraining (45% UT and 65% UT), at the same absolute power output pretraining and posttraining (65% UT and 65% TR), and at the new relative power output posttraining (65% TR).

The mechanism that explains these differences is unknown. However, the authors speculate that hormonal differences may have been the cause. Factors that may influence these differences include the amount of circulating epinephrine (although these values did not differ between the males and females in this study), receptor availability and affinity for epinephrine (which was not tested), or the interaction of other circulating hormones, especially estrogen and progesterone (higher, of course, in the females) with epinephrine. It is also possible, because many of the differences were significant only after training that the sympathetic nervous system in males and females may adapt differently to exercise training.



Source: Friedlander, A. L., G. A. Casazza, M. A. Horning, M. J. Huie, M. F. Piacentini, J. K. Trimmer, & G. A. Brooks: Training-induced alterations of carbohydrate metabolism in women: Women respond differently from men. *Journal of Applied Physiology*. 85(3):1175–1186 (1998).

Table 2.6 provides an estimation of how fuel utilization varies by race distance (10 km [6.2 mi] and 42.2 km [26.2 mi]), or

marathon distance) and performance (a “fast” and a “slow” time for each distance). Compare the muscle glycogen utilization in the fast and the slow conditions. At the short distance (10 km), there is not much difference, but during the marathon, almost six times as much muscle glycogen is used by the faster runner. Conversely, the greatest amount of fat is utilized by the slow marathoner. Circulating glucose use is much higher in marathoners because the longer duration and lower intensity permit the liver to generate (by gluconeogenesis) and release glucose into the bloodstream. The 10-km runner has sufficient muscle glycogen stores and does not need to rely on external glucose production. Very little protein (BCAA) is used by either of the 10-km runners, but a small amount provides some energy for the marathoners.

A complex interaction exists between exercise energy needs and the fuel sources that provide this energy within the human organism.

Summary

1. The process by which ATP is formed from food is called cellular respiration.
2. Carbohydrate metabolism consists of four stages that convert glucose or glycogen into carbon dioxide, water, and ATP energy.
3. Stage I of carbohydrate metabolism is known as glycolysis. Glycolysis consists of a series of 10 or 11 steps; it occurs in the cytoplasm of cells and is anaerobic. It begins with glucose or glycogen and ends with pyruvate (pyruvic acid) or lactate (lactic acid). In the process, a net gain of 2 ATP molecules is achieved by substrate-level phosphorylation if the fuel was glucose and 3 ATP if the fuel was glycogen. A pair of $\text{NADH} + \text{H}^+$ also results.
4. Stage II of carbohydrate metabolism has no identifying name, but it results in the formation of acetyl CoA from pyruvate. These two steps occur in the mitochondrial matrix; although no oxygen is used directly, the process must be

aerobic. No ATP is produced, but two pairs of hydrogen atoms are released and picked up by NAD^+ , forming $\text{NADH} + \text{H}^+$.

5. Stage III of carbohydrate metabolism is the Krebs cycle. This stage consists of eight steps and also occurs in the mitochondrial matrix; again, no oxygen is used directly, but it also must be aerobic. Two ATP are produced by substrate-level phosphorylation and pairs of hydrogen atoms are removed at four separate steps. In three cases, the hydrogen ions are picked up by NAD^+ and in the fourth by FAD.
6. Stage IV of carbohydrate metabolism is known as electron transport and oxidative phosphorylation. Electron transport takes place in the inner mitochondrial membrane and consists of relaying electrons from the hydrogen atoms from one protein carrier to another and transporting the hydrogen ions into the intermembrane space. In the process, an electrical current is created. This electrical energy is then used to synthesize ATP from ADP by the addition of a phosphate as the H^+ move through the ball-and-stalk apparatus (ATP synthase) into the mitochondrial matrix. For each hydrogen carried to the ETS by NAD^+ , 2.5 ATP molecules are formed. For each hydrogen carried by FAD to the electron transport chain, 1.5 ATP molecules are formed, lower than the previously thought theoretical values of 3 and 2, respectively.
7. A total of 30 ATP molecules are produced if the fuel substrate is glucose and the muscle is skeletal. A total of 31 ATP are produced if the fuel substrate is glycogen and the muscle is skeletal. A total of 32 ATP are produced if the fuel substrate is glucose and the muscle is cardiac. A total of 33 ATP are produced if the fuel substrate is glycogen and the muscle is cardiac.
8. Triglycerides stored in adipose cells or stored intramuscularly are the major storage form of energy in humans. Triglycerides are composed of fatty acids and glycerol. Muscle cells can only use fatty acids as a fuel.
9. Fatty acid can only be utilized as a fuel source aerobically within the mitochondria. Fatty acids must undergo the process of beta-oxidation—which involves the removal of

hydrogen atoms (oxidation) and the removal of pairs of carbons (at the beta carbon location) to form acetyl coenzyme A—before entering the Krebs cycle and electron transport.

10. The ATP produced from each fatty acid depends upon the number of carbon pairs.
11. When glucose supplies are inadequate and oxaloacetate must be converted to glucose, the acetyl coenzyme A derived from fatty acids is converted into three ketones: acetoacetic acid, beta-hydroxybutyric acid, and acetone.
12. Amino acids are used primarily in anabolic processes in human cells, building proteins such as hemoglobin and the contractile elements of muscle. However, amino acids—and especially the branched chain amino acids (valine, leucine, and isoleucine)—can be used as a fuel source and may contribute as much as 5–15% of the energy supply in long-term, dynamic endurance activity.
13. All amino acids contain an amino group (NH_2). Before an amino acid can be used as a fuel, this amino group must be removed. Removal of the amino group is accomplished by two processes: oxidative deamination or transamination (most common). Entry into the Krebs cycle and electron transport then occurs at several locations, but ultimately all of the intermediates except acetyl CoA are converted to pyruvate before being used to produce energy.
14. Twelve-and-a-half ATP are derived from each of the amino acid derivatives utilized as pyruvate, and 10 ATP are produced from each derivative utilized as acetyl CoA.
15. ATP energy production has both intracellular and extracellular regulation. Intracellular regulation operates primarily by the feedback stimulation (by ADP, AMP, P_i , and a rise in pH) or inhibition (by ATP, CP, citrate, and a drop in pH) of the rate-limiting enzyme. Extracellular regulation is achieved by the coordinated action of the sympathetic nervous system (via norepinephrine) and the hormonal system (norepinephrine, epinephrine, glucagon, growth hormone, and cortisol).
16. The goal of metabolism during exercise is threefold:

- a. To increase the mobilization and utilization of free fatty acid from adipose tissue and intramuscular stores
 - b. To decrease the uptake of glucose into nonworking muscle cells while at the same time providing glucose for nerve and brain cells
 - c. To increase the breakdown of liver and muscle stores of glycogen and create glucose from noncarbohydrate sources in the liver
17. The creation of glucose from noncarbohydrate sources is called gluconeogenesis. The primary fuel sources for gluconeogenesis are glycerol, lactate or pyruvate, and alanine.
18. Fatty acids constitute the largest fuel supply. Other fuels, in descending order, are total muscle glycogen, liver glycogen, and circulating glucose.
19. Which energy substrate is utilized and in what amounts depends upon the complex interaction of the type, duration, and intensity of exercise; the proportion of muscle fiber types involved; and the training status, long-term nutritional state, and shortterm dietary status of the exerciser.
20. In general, very-short-duration, very-high-intensity dynamic activity, and static contractions are special cases that rely predominantly on the ATP-PC and glycogen stored in the muscle fibers.
21. In dynamic aerobic activity, the higher the intensity, the more important carbohydrates (glucose and glycogen) are as a fuel; the lower the intensity, the more important fats are as a fuel. Likewise, the shorter the duration, the more important carbohydrates are as a fuel; the longer the duration, the more that fat is utilized. In activities lasting more than an hour, protein makes a small but important contribution to the energy supply.

Review Questions

1. Distinguish between anabolism and catabolism. Is cellular

respiration anabolic or catabolic?

2. Name and briefly summarize the four steps of carbohydrate metabolism.
3. Explain how a count of 30 ATP is achieved by cellular respiration if the fuel substrate is glucose in skeletal muscle. Why is the count often 31, 32, and 33?
4. Why is beta-oxidation necessary before fat can be used as an energy substrate? Describe what occurs during beta-oxidation.
5. State how the calculation is completed to determine the number of ATP produced from fatty acids. Complete an example using a fat having 24 carbons.
6. Name and describe the process that amino acids must undergo before being used as a fuel substrate. Why is this process necessary?
7. Identify the locations in the metabolic pathways where amino acids may enter. How does the ATP count differ between these locations?
8. Why is acetyl CoA called the universal common intermediate?
9. Why might the breath of someone suffering from anorexia nervosa smell sweet?
10. Describe the role of enzymes in the metabolic pathways. Identify the rate-limiting enzymes in Stages I, III, and IV of carbohydrate metabolism. How are enzymes regulated?
11. What are the goals of metabolic regulation during exercise? How are these goals achieved by the interaction of the sympathetic nervous system and the hormonal system?
12. Compare the relative availability and use of carbohydrate, fat, and protein fuel substrates on the basis of the intensity and duration of exercise.

Literature Search

We discussed glycolysis and glycogenolysis in this chapter. These concepts are important to understand to when considering how energy is produced to fuel exercise. To explore this topic further,

do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search glycogen storage, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Glycogen storage and muscle
 - ii. Glycogen storage and training status
 - iii. Glycogen storage and caffeine
 - iv. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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3 Anaerobic Metabolism during Exercise



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OBJECTIVES

After studying the chapter, you should be able to:

- Describe the energy continuum as it relates to varying durations of maximal maintainable exercise.
- Provide examples of sports or events within sports in which the ATP-PC, lactic, or oxygen system predominates.
- List the major variables that are typically measured to describe

the anaerobic response to exercise and, where appropriate, the actual exercise test itself.

- Explain the physiological reasons why lactate may accumulate in the muscles and blood.
- Distinguish among the ATP-PC, lactic, and oxygen systems in terms of power and capacity.
- Identify oxygen deficit and excess postexercise oxygen consumption and explain the causes of each.
- Describe the changes in ATP and PC that occur during constant-load, heavy exercise lasting 3 minutes or less.
- Describe the changes in lactate accumulation that occur during short-term, highintensity, anaerobic exercise lasting 3 minutes or less; short-term, light to moderate, and moderate to heavy submaximal aerobic exercise; long-term, moderate to heavy, submaximal aerobic exercise; incremental exercise to maximum; and dynamic resistance exercise.
- Differentiate among the terms anaerobic threshold, ventilatory threshold, and lactate threshold and explain why anaerobic threshold is a misnomer.
- Describe the benefits of lactate as a fuel source and signaling molecule.
- Discuss why the accumulation of lactate is a physiological and performance problem.
- Explain the fate of lactate during exercise and recovery.
- Compare anaerobic metabolic characteristics for males versus females; children and adolescents versus young and middle-aged adults; and the elderly versus young and middle-aged adults and cite possible reasons for these differences.

Introduction

Chapter 2 explained how ATP, the ultimate energy source for all human work, is produced by the metabolic pathways. The emphasis there was on the energy substrate (carbohydrate, fat, or protein) utilized as fuel. This chapter and the next look at ATP production and utilization on the basis of the need for oxygen. Anaerobic metabolism does not require oxygen to produce ATP, whereas aerobic metabolism does. Critical to understanding

anaerobic and aerobic exercise metabolism is the fact that these processes are not mutually exclusive; that is, anaerobic metabolism and aerobic metabolism are not either/or situations in terms of how ATP is provided. Both systems start simultaneously and work concurrently when additional energy is needed. When describing muscular exercise, the terms aerobic or anaerobic refer to the system predominating at the time.

The Energy Continuum

Figure 3.1 reviews the three sources of ATP production introduced in [Chapter 2](#). **Figure 3.1A** describes *alactic anaerobic metabolism*, sometimes called the *phosphagen* or *ATP-PC system*. Once produced, ATP is stored in the muscle. This amount is relatively small and can provide energy for only 2–3 seconds of maximal effort ([Mougios, 2006](#)). However, another high-energy compound, phosphocreatine (PC), also known as creatine phosphate (CP), can be used to resynthesize ATP from ADP almost instantaneously. The amount of PC stored in muscle is about three to four times that of ATP ([Bogdanis et al., 1995](#)). Muscles differ in the amount of stored PC by fiber type. Muscle fiber types are fully described in [Chapter 17](#), but briefly, fibers that produce energy predominantly by anaerobic glycolysis are called glycolytic; those that produce energy predominantly aerobically are called oxidative. In terms of contraction speed, muscle fibers are either fast twitch or slow twitch. When contractile and metabolic characteristics are combined, three fiber types are generally described: fast-twitch glycolytic (FG also known as type IIX), fast-twitch oxidative glycolytic (FOG also known as type IIA), and slow-twitch oxidative (SO also known as type I). Fast-twitch fibers have proportionally more PC than ATP compared to SO fibers. Any time the energy demand is increased—whether the activity is simply turning a page of this book, coming out of the blocks for a sprint, or starting out on a long bicycle ride—at least part of the immediate need for energy is supplied by these stored forms (ATP, PC), which must ultimately be replenished. These sources are also used preferentially in high-intensity, very-short-duration activity such as the 50- and 100-m

(yd) dashes and snowboard half-pipe. Most resynthesis of ATP from PC takes place in the first 10 seconds of maximal muscle contraction; little, if any, occurs after 20 seconds of maximal activity ([Gastin, 2001](#)). This ATP-PC system neither uses oxygen nor produces lactate and is thus said to be *alactic anaerobic*.

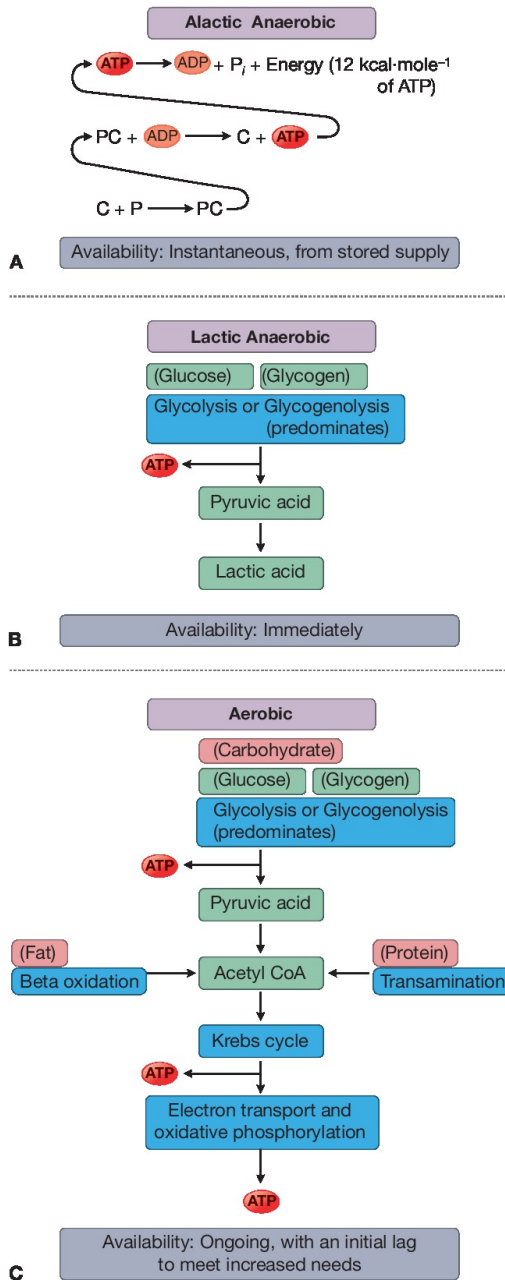


Figure 3.1 Anaerobic and Aerobic Sources of ATP.

A. Describes alactic anaerobic metabolism. **B.** Represents lactic anaerobic metabolism. **C.** Depicts aerobic metabolism.

Figure 3.1B represents *anaerobic glycolysis*, also called the *lactic acid (LA) system*. When the demand for ATP exceeds the capacity of the phosphagen system and the aerobic system (at the initiation of any activity or during high-intensity, short-duration exercise), anaerobic (*fast*) glycolysis is used to produce ATP. This is rather like calling in the reserves, for glycolysis can provide the supplemental energy quickly. The rate of ATP production from glycolysis reaches its maximum about 5 seconds after initiation of contraction and is maintained at this rate for several seconds ([Gastin, 2001](#)). This system predominates in activities such as a 1,500-m speed skating event. Other sport activities with a heavy reliance on the LA system include middle distances (e.g., track 200–800 m, swimming 100 m, slalom, and downhill skiing), gymnastic floor exercise, parallel bars, a round of boxing, and a period of wrestling. The ability to perform the events with speed and power is the benefit. The cost is that the production of lactate often exceeds clearance, and lactate accumulates. Because this system does not use oxygen but does result in the production of lactate, it is said to be *lactic anaerobic*.

Figure 3.1C shows *aerobic metabolism*, also termed the *O₂ system*. The generation of ATP from *slow* (aerobic) glycolysis, the Krebs cycle, and electron transport–oxidative phosphorylation is constantly in operation at some level. In resting conditions, this system provides basically all of the energy needed. When activity begins or occurs at moderate levels of intensity, oxidation increases quickly and proceeds at a rate that supplies the needed ATP. If the workload is continuously increased, aerobic metabolism proceeds at a correspondingly higher rate until its maximal limit is reached. The highest amount of oxygen the body can consume during heavy dynamic exercise for the aerobic production of ATP is called maximal oxygen uptake, or $\dot{V}O_{2\max}$. Because $\dot{V}O_{2\max}$ is primarily an index of cardiorespiratory power, and as such is used as a measure of cardiovascular-respiratory fitness, it is discussed in depth in that unit. However, because $\dot{V}O_{2\max}$ reflects the amount of oxygen available for the aerobic production of ATP, it is also an important metabolic measure. Both aerobic and anaerobic exercises are often described in terms of a given percentage of $\dot{V}O_{2\max}$ (either <

or $>100\% \dot{V}O_{2\max}$), that is submaximal, maximal, and supramaximal. Anaerobic metabolic processes are important at the onset of all aerobic exercise, contribute significantly at submaximal levels, and increase their contribution as the exercise intensity gets progressively higher. Depending on an individual's fitness level, lactic anaerobic metabolism begins to make a significant contribution to dynamic activity at approximately 40–60% $\dot{V}O_{2\max}$. However, even then the ability to use oxygen is most important. By between 1 and 2 minutes of maximal exercise, the relative contributions of ATP production from the aerobic and anaerobic energy systems are approximately equal (Gastin, 2001). Because the aerobic system involves the use of oxygen and proceeds completely to oxidative phosphorylation, it is said to be *aerobic* or *oxidative*. Sport activities that rely predominantly on the O_2 system include long-distance events, such as the 5,000 and 10,000 m in track; marathons; swimming 1,500 m; long-distance triathlon; cross-country running, skiing, and orienteering; field hockey, soccer, and lacrosse; and race walking.

These three sources of ATP production—the phosphagen system (ATP-PC system), the glycolytic system (LA system), and the oxidative system (O_2 system)—contribute to maximal exercise of different durations in a pattern called the *time-energy system continuum*. This continuum assumes that the individual is working at a maximal maintainable intensity for a continuous duration. This means that it is assumed that an individual can go all out for 5 minutes or less or can work at $100\% \dot{V}O_{2\max}$ for 10 minutes, at $95\% \dot{V}O_{2\max}$ for 30 minutes, at $85\% \dot{V}O_{2\max}$ for 60 minutes, and at $80\% \dot{V}O_{2\max}$ for 120 minutes. Of course, there are individual differences, but these assumptions are reasonable in general.

Figure 3.2 differentiates the anaerobic systems into the ATP-PC system and LA system as well as indicates that the O_2 system participates quickly in all-out maximal exercise lasting 300 seconds (5 minutes) or less (Gastin, 2001). Note that both anaerobic systems respond immediately but neither can sustain the high level of ATP production needed. The LA system contributes more than does the ATP-PC system by approximately 10 seconds. Conversely, the aerobic energy system (O_2 system) is

incapable of meeting the increased energy demands immediately but contributes to a meaningful degree quickly.

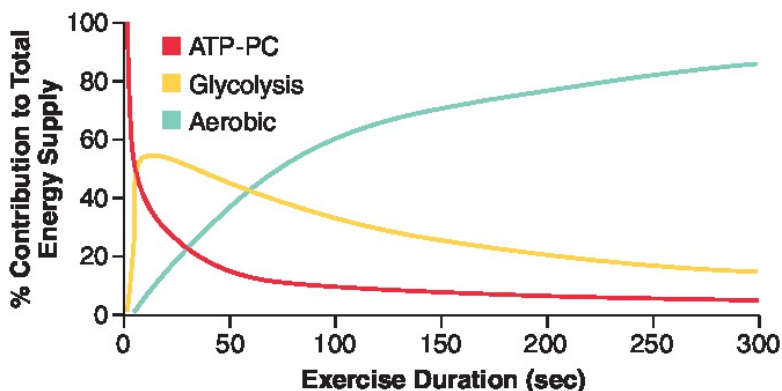


Figure 3.2 Relative Contributions of the ATP-PC, LA, and O₂ Energy Systems to Maximal Exercise.

Source: Reprinted by permission from Springer Gastin, P. B.: Energy system interaction and relative contribution during maximal exercise. *Sports Medicine*. 31(10):725–741 (2001). Copyright © 2012 Springer Nature.

Figure 3.3 shows the best estimates available of the relative contributions of the combined anaerobic (ATP-PC plus LA) systems and the aerobic energy system to the total energy requirement for exercise durations up to 120 minutes. Again, initially the anaerobic systems predominate, but by 30 seconds more than 20% of the ATP is being supplied by oxidative phosphorylation. The point at which the aerobic and anaerobic contribution becomes approximately equal is 75 seconds (**Figure 3.2**). From then on, the aerobic system dominates.

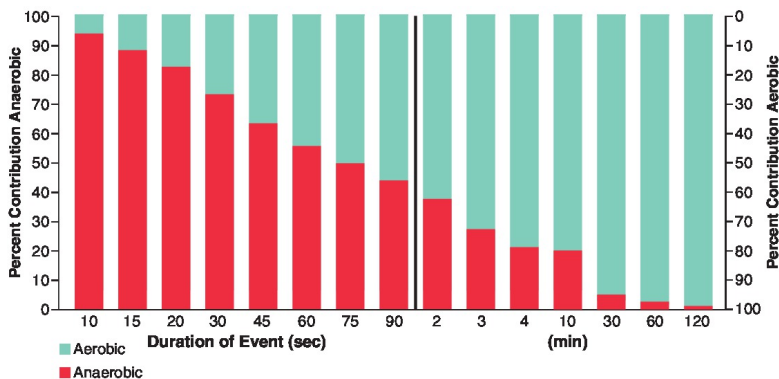


Figure 3.3 Time-Energy System Continuum.

Approximate relative contributions of aerobic and anaerobic energy production at maximal maintainable intensity for varying durations. **Note:** The graphs assume 100% $\dot{V}O_{2\max}$ at 10 minutes; 95% $\dot{V}O_{2\max}$ at 30 minutes; 85% $\dot{V}O_{2\max}$ at 60 minutes; and 80% $\dot{V}O_{2\max}$ at 120 minutes. ATP-PC \leq 10 seconds.

Four basic patterns can be discerned from this continuum. Understanding these patterns are helpful when developing exercise or sport-specific training programs.

1. All three energy systems (ATP-PC, LA, O₂) are involved in providing energy for all durations of exercise.
2. The ATP-PC system predominates in activities lasting 10 seconds or less. Since the ATP-PC system is involved primarily at the onset of longer activities, it becomes a smaller portion of the total energy supply as duration gets longer.
3. Anaerobic metabolism (ATP-PC and LA) predominates in supplying energy for exercises lasting between 1 and 2 minutes. The equal contribution point for anaerobic and aerobic energy contribution to maximal exercise is probably close to 75 seconds. However, even exercises lasting as long as 10 minutes use at least 15% anaerobic sources. Within the anaerobic component the longer the duration, the greater the relative importance of the LA system in comparison to the

ATP-PC system.

4. By 2 minutes of exercise, the O₂ system clearly dominates. The longer the duration, the more important it becomes.

The rest of this chapter concentrates on the anaerobic contribution to energy metabolism. [Chapter 4](#) then focuses on the aerobic contribution to energy metabolism, although again it must be emphasized that the two systems work together, with one or the other predominating based primarily on the duration and intensity of the activity.

Anaerobic Energy Production

Alactic Anaerobic PC Production

As previously described, alactic anaerobic production of ATP involves the use of phosphocreatine (PC), which is simply creatine bound to inorganic phosphate. An adult human has a creatine concentration of approximately 120–140 mmol·kg⁻¹ of dry muscle, although there is considerable individual variation from a low of 90–100 mmol·kg⁻¹ to a high of about 150–160 mmol·kg⁻¹. Each day approximately 2 g of creatine are degraded in a nonreversible reaction to form creatinine. This creatinine is ultimately excreted by the kidneys in urine. The loss is counterbalanced under normal dietary conditions by the ingestion of about 1 g of creatine from meat, poultry, and/or fish and the synthesis of another gram in the liver from three amino acids: arginine, glycine, and methionine. Close to 95% of the creatine in the body is stored in skeletal muscle. Thirty to forty percent is stored as free creatine and the rest as phosphocreatine.

Figure 3.4 shows the breakdown of ATP and PC during heavy exercise. It also shows the restoration of ATP from energy substrate sources and the restoration of PC from the regenerated ATP. The process continues until the resting levels of both PC and ATP are regained. Specifically, when ATP is hydrolyzed by the contractile proteins in muscle (1) (large inner circle), the resulting ADP is rephosphorylated in the cytoplasm by the PC available there (2) (small inner circle). In turn, the now free creatine is rephosphorylated at the inner mitochondrial

membrane from the breakdown of ATP produced at that site (3) (large outside oval). The remnant (4) ADP is then free, in turn, to be phosphorylated again by oxidative phosphorylation using energy substrates. In addition to providing ATP rapidly, this mechanism, called the creatine phosphate shuttle, is one way in which electron transport and oxidative phosphorylation are regulated (Brooks et al., 1999).

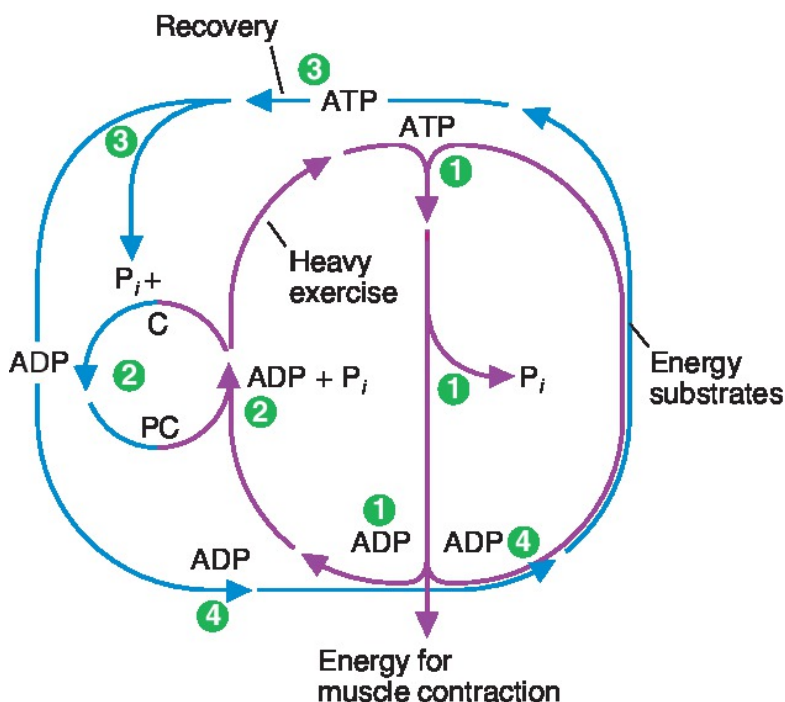


Figure 3.4 Use and Regeneration of ATP-PC.

1. $ATP \rightarrow ADP + P_i + \text{energy for muscle contraction}$ (large inner circle).
2. During heavy exercise, the ADP is regenerated to ATP by the breakdown of PC ($PC + ADP \rightarrow C + ATP$) (small inner circle).
3. Only in recovery will the nearly depleted PC stores be restored from the breakdown of ATP resulting from aerobic metabolic production (large outside oval).
4. $ADP \rightarrow ATP$ from energy substrates.

See the Focus on Application box on the use of creatine as an

ergogenic aid.

FOCUS ON APPLICATION

Creatine as an Ergogenic Aid

Creatine (C) (primarily in the form of creatine monohydrate [CM]) continues to enjoy unprecedented popularity as an ergogenic aid. Oral C supplementation can increase muscle C content by approximately 10–40%, depending on an individual's starting muscular concentration. If an individual has the upper limit of C already stored in their muscle (~ 160 mmol·kg⁻¹ of dry muscle), supplementation with C will not further increase storage, and excess ingested C will be excreted in the urine ([American College of Sports Medicine \[ACSM\] Roundtable, 2000](#); [Buford et al., 2007](#)). This may explain why one individual responds to CM supplementation and another might be a “nonresponder.” Although these individuals may not have increases in C storage, there are other benefits of C supplementation that a “nonresponder” could exhibit that will be discussed later in this section. The relative percentage of different muscle fiber types can also impact the response to C ingestion such that individuals with higher percentages of type II fibers have a higher response potential ([Cooper et al., 2012](#)). Training status may impact C uptake during a loading phase because intramuscular levels may already be elevated ([Bemben and Lamont, 2005](#)).

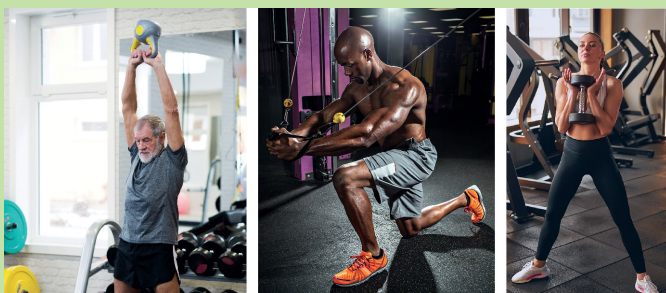
Metabolically, there are three primary mechanisms to explain how increased levels of C in muscle could impact anaerobic energy production: (1) increased C means increased levels of PC for increased rephosphorylation of ADP to ATP; (2) stimulation of phosphofructokinase (PFK), the rate-limiting enzyme of glycolysis, occurs when PC levels decline as explained in the previous point ([Bemben and Lamont, 2005](#)); and (3) increased muscle glycogen when combined with a high-carbohydrate diet ([Cooper et al., 2012](#); [Rawson and Persky, 2007](#)). Additionally, molecular

adaptation through an increased gene expression of growth factors, and reduced muscle damage also occur (Poortmans et al., 2010). These adaptations allow an individual to train harder and recover faster (Buford et al., 2007). A number of studies have also indicated that C supplementation, along with heavy resistance training, enhances the normal training adaptation (ACSM Roundtable, 2000; Cooper et al., 2012). Creatine alone does not increase skeletal muscle protein synthesis (Poortmans et al., 2010).

Numerous studies have concluded that CM supplementation is beneficial as an ergogenic aid and a lifestyle supplement. On the whole, of the several hundred peerreviewed published research studies, nearly 70% have reported a significant improvement in exercise capacity (ACSM Roundtable, 2000; Buford et al., 2007; Cooper et al., 2012; Jäger et al., 2011; Luckose et al., 2015). The efficacy of C depends in large part on the exercise category. The greatest improvement in performance seems to occur in the later bouts of a repetitive series of exercises in which the high power output lasts only a matter of seconds separated by rest periods of 20–60 seconds (ACSM Roundtable, 2000; Sahlin, 2014). A 2005 analysis by Bembien and Lamont concurred that the use of CM is effective for short-term high-intensity activities, especially when the activity involves repeated bouts. They also concluded benefits from CM supplementation depended on the type of muscle contraction in which dynamic and isotonic observed the greatest improvements. No studies have reported an impairment of exercise performance. The International Society of Sports Nutrition position stand—Safety and Efficacy of Creatine Supplementation in Exercise, Sport, and Medicine (2017)—concluded that “creatine monohydrate is the most effective ergogenic nutritional supplement currently available to athletes with the intent of increasing high-intensity exercise capacity and lean body mass during training.” The addition of carbohydrate or carbohydrate and protein combined with C appears to increase power output, intramuscular glycogen stores, and muscular retention of C; however, the effect on performance currently does not appear to be any greater than using CM alone (Buford et al., 2007; Tomcik et al., 2018).

There is no evidence that any of the newer forms of C are more effective and/or safer than CM whether ingested alone or in combination with other ingredients (Jäger et al., 2011).

Many studies have also highlighted advantageous results of CM in other aspects of life, including, but not limited to: aging, menopause, vascular health, mental health, cognition, and brain injuries (Clarke et al., 2020; Dickinson et al., 2014). Furthermore, C has numerous positive effects on aging populations by managing age-related deficiencies in skeletal muscle and bone, as well as improving mental cognition (Chilibeck et al., 2015; Gualano et al., 2016). CM has many known benefits on cognition and brain health (Bakian et al., 2020; Sullivan et al., 2000). C is widely accepted as a vastly beneficial supplement for people in all stages of life to take (Kreider et al., 2017).



As with any supplement, concerns have been expressed regarding the safety of C ingestion. The most commonly reported side effect is weight gain, which is usually a desired outcome for many taking CM (Kreider et al., 2017). Creatine loading is associated with an increase in body weight of approximately 2%; thus, in individuals with otherwise stable weight, an increase in weight can be used as a rough marker that C is actually being loaded (Sahlin, 2014). Despite some anecdotal claims and case reports of gastrointestinal disturbances (nausea, vomiting, diarrhea), musculoskeletal injuries, dehydration and cramping, numerous credible well-controlled clinical studies, and reviews have concluded that there is no scientific evidence that the short- or long-term use of CM has any detrimental effects in healthy or clinical

populations of any age (Buford et al., 2007; Jäger et al., 2011; Kreider et al., 2017; Luckose et al., 2015). Importantly, out of extreme caution, individuals with medically documented preexisting kidney problems are advised to consult with their physician prior to using CM; however, there is no convincing evidence to suggest C negatively affects renal function (Kreider et al., 2017). With regard to thermoregulation, C appears to actually enhance performance in hot/humid conditions by maintaining hematocrit, aiding thermoregulation, reducing exercising heart rate and sweat rate, and maintaining plasma volume (Dalbo et al., 2008). The evidence of safety is strong at intakes up to 5 g·d⁻¹ for chronic supplementation for up to 5 years (Cooper et al., 2012; Shao and Hathcock, 2006; Tarnopolsky, 2010).

Recent literature suggests that there are systematic sex differences with C supplementation as males and females store and metabolize C in different manners. Literature suggests that hormones cause changes in women across their reproductive life regarding endogenous creatine synthesis, transport, and expression allude to changes in the cellular energy circuit (Ellery et al., 2016). While fewer studies have been conducted using children or adolescent participants, and these have primarily been therapeutic interventions, none have shown CM to have adverse effects in this population. Thus, the International Society of Sports Nutrition (2017) recommends that adolescents supplementing with CM should be involved in competitive supervised training; consuming a well-balanced, performance-enhancing diet; and knowledgeable about appropriate use of creatine. A typical C protocol consists of a loading phase of 20 g of CM split into four daily intakes of 5 g each or 0.3 g·kg⁻¹·d⁻¹ of CM for 5–7 days followed by a maintenance phase of 3–5 g·d⁻¹ (or 0.03 g·kg⁻¹·d⁻¹) thereafter.

CM products are readily available as a dietary supplement and are regulated by the U.S. Food and Drug Administration as a dietary ingredient. The legal and regulatory status of other forms is somewhat ambiguous. CM is approved as a natural health product in Canada by the Natural Health Products Directorate (Jäger et al., 2011). Creatine supplementation is not currently banned by any athletic

organization, although the National Collegiate Athletic Association (NCAA) does not allow institutions to provide CM supplements for athletes. If an NCAA athlete wishes to supplement with CM, the supplement must be tested by a third-party organization. Thirdparty testing guarantees that the contents and doses of a supplement are true to the product label. The International Olympic Committee (IOC) ruled that there was no need to ban C since it is naturally found in food and there is no valid noninvasive test to determine supplementation (Buford et al., 2007). These rulings, of course, can change at any time, so the wise athlete will check before supplementing.

Sources: ACSM Roundtable (2000); Bakian et al. (2020); Bemben and Lamont (2005); Branch (2003); Buford et al. (2007); Chilibeck et al. (2015); Clarke et al. (2020); Cooper et al. (2012); Dalbo et al. (2008); Dickinson et al. (2014); Ellery et al. (2016); Gualano et al. (2016); Jäger et al. (2011); Lopez et al. (2009); Luckose et al. (2015); Misic and Kelley (2002); Rawson and Persky (2007); Sahlin (2014); Shao and Hathcock (2006); Sullivan et al. (2000); Tarnopolsky (2010).

Lactic Acid/Lactate Production

Lactic acid is produced in muscle cells when the $\text{NADH} + \text{H}^+$ formed in glycolysis (step 6; see [Figure 2.6](#) in [Chapter 2](#)) is oxidized to NAD^+ by a transfer of the hydrogen ions to pyruvic acid ($\text{C}_3\text{H}_4\text{O}_3$), which in turn is reduced to lactic acid ($\text{C}_3\text{H}_6\text{O}_3$) (Brooks, 1985; Brooks, 2010; Newsholme and Leech, 1983). In muscle tissue, lactic acid is produced in amounts that are in equilibrium with pyruvic acid under normal resting conditions. As stated in [Chapter 2](#), there is some unresolved controversy as to whether skeletal muscle actually produces lactate or lactic acid although most recently, lactate is the preferred term (Brooks, 2018, 2020; Marcinek et al., 2010; Robergs et al., 2018) because even if lactic acid is the substance actually produced, at physiological pH 99% of the lactic acid is dissociated immediately to H^+ and La^- (lactate) (Brooks, 1985; Gladden, 2004). In addition, lactate is always produced by red blood cells,

portions of the kidneys, and certain tissues within the eye. Both resting and exercise blood lactate values depend on the balance between lactate *production* (*appearance*) and *removal* (*disappearance* or *clearance*). This balance of appearance and clearance is called *turnover*. When production exceeds removal, lactate is said to accumulate. The issues, then, especially during exercise, focus on what conditions result in lactate production and what processes lead to lactate removal. When blood lactate concentrations are measured, they reflect the balance of lactate production and removal. The major contributors to lactate production and removal are summarized in **Figure 3.5**.

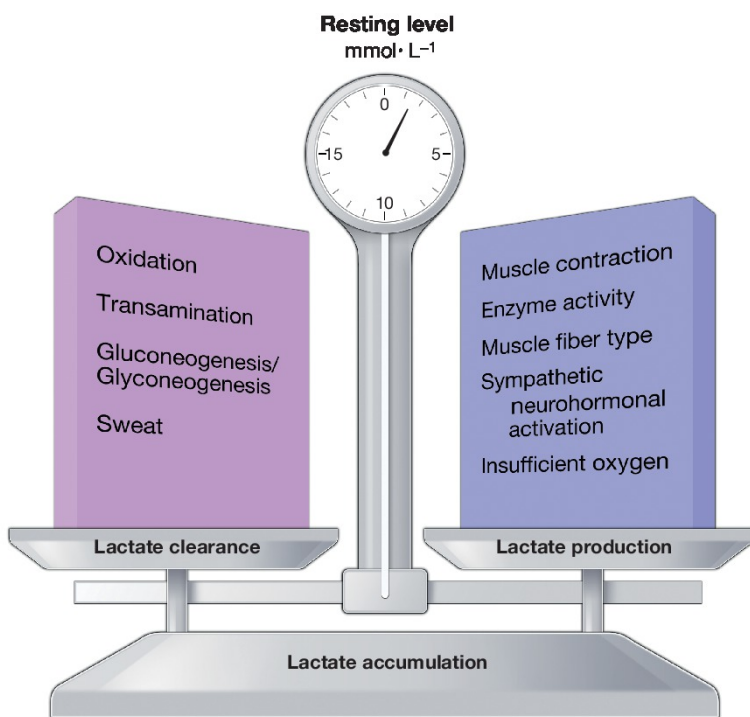


Figure 3.5 The Balance between Lactate Production and Clearance.

Lactate accumulation in the blood is the result of the balance between lactate production and lactate clearance. Normal resting blood values are usually $\leq 2.0 \text{ mmol} \cdot \text{L}^{-1}$.

Factors in Lactic Acid/Lactate Production

In general, the relative rates of glycolytic activity (stage I of carbohydrate metabolism) and oxidative activity (stages II, III, and IV) determine the production of lactate. Specifically, five factors play important roles: muscle contraction, enzyme activity, muscle fiber type, sympathetic neurohormonal activation, and insufficient oxygen (anaerobiosis, the onset of anaerobic metabolism).

1. **Muscle contraction.** During exercise, muscle activity obviously increases. Muscle contraction involves the release of calcium (Ca^{2+}) from the sarcoplasmic reticulum. In addition to its role in the coupling process of actin and myosin, Ca^{2+} also causes glycogenolysis by activating the enzyme glycogen phosphorylase. Glycogen is processed by fast glycolysis and results in the production of lactate regardless of whether oxygen levels are sufficient or not (Brooks, 2018, 2020; Ferguson et al., 2018).
2. **Enzyme activity.** The conversion of pyruvate and $\text{NADH} + \text{H}^+$ to lactate and NAD^+ is catalyzed by specific isozymes of the enzyme lactate dehydrogenase (LDH), whereas the conversion of pyruvate to acetyl CoA (stage II) prior to entry into the Krebs cycle is catalyzed by the enzyme pyruvate dehydrogenase (PDH). LDH has the highest rate of functioning of any of the glycolytic enzymes and is much more active than the enzymes that provide alternate pathways for pyruvate metabolism, including PDH and the rate-limiting enzymes in the Krebs cycle. Any increase in pyruvate and $\text{NADH} + \text{H}^+$ further increases the activity of LDH and results in the production of lactate (Spriet et al., 2000). Therefore, lactate production is an inevitable consequence of glycolysis (Brooks, 1986, 2010, 2020; Gaesser and Brooks, 1975; Spriet et al., 2000). The more pyruvate provided, the more lactate produced. The shifting of the hydrogen atoms from $\text{NADH} + \text{H}^+$ to pyruvate-forming lactate serves to maintain the redox potential of the cell. The redox, or oxidation-reduction, potential is the ratio of $\text{NADH} + \text{H}^+$ to NAD^+ . There is a finite amount of NAD^+ available in the cytoplasm to accept hydrogen atoms in step

6 and keep glycolysis going. To maintain this supply, $\text{NADH} + \text{H}^+$ must either transfer the hydrogen atoms into the mitochondria (via hydrogen ion shuttles) to the electron transport chain or give them up to pyruvate.

3. Muscle fiber type. During high-intensity, short-duration activities, FG muscle fibers are preferentially recruited. These fast-contracting glycolytic fibers produce lactate when they contract, regardless of whether oxygen is present in sufficient amounts or not. This response appears to be a function of the specific LDH isozyme and the low mitochondrial density found in these fibers. Different LDH isozymes are found in different fibers ([Green, 1986](#)). LDH isozymes 4 and 5 predominate in fast-twitch fibers and facilitate the conversion of pyruvate to lactate. Conversely, LDH isozymes 1 and 2 predominate in cardiac, slow twitch oxidative (SO), and fast-twitch mitochondria and catalyze the conversion of lactate back into pyruvate ([Ferguson et al., 2018](#); [Houston, 1995](#)).
4. Sympathetic neurohormonal activation. During heavy exercise, activity of the sympathetic nervous system stimulates the release of epinephrine and glucagon and suppression of insulin (see [Figure 2.17](#) in [Chapter 2](#)). The result is the breakdown of glycogen, leading ultimately to high levels of glucose-6-phosphate (G6P). (Refresh your memory by referring to [Figure 2.6](#), if necessary, to locate G6P in glycolysis.) High levels of G6P increase the rate of glycolysis and thus the production of pyruvate (sometimes referred to as pyruvic acid) ([Brooks, 1986](#)). As previously described, any increase in pyruvate and $\text{NADH} + \text{H}^+$ ultimately results in an increase in lactate. In addition, late in prolonged endurance activity, epinephrine-mediated glycogen breakdown may bring about the release of lactate from resting muscle when an increased lactate release is no longer occurring in contracting muscles ([Stallknecht et al., 1998](#)).
5. Insufficient oxygen (anaerobiosis, onset of anaerobic metabolism). Finally, during high-intensity, short-duration or near-maximal exercise or during static contractions when blood flow is impeded ([Stallknecht et al., 1998](#)), the delivery

of oxygen to the mitochondria—and thus the availability of oxygen as the final electron acceptor at the end of the respiratory chain—can become deficient. In these circumstances, glycolysis proceeds at a rate that produces larger quantities of $\text{NADH} + \text{H}^+$ than the mitochondria have oxygen to accept. Again, “something” has to be done with the hydrogen atoms so that the NAD^+ can be regenerated. That “something” is the transfer of the hydrogen atoms to pyruvic acid and the formation of lactic acid, which dissociates quickly to lactate.

Thus, although lactate is associated with high-intensity, short-duration exercise, this is not the only exercise condition that results in the production of lactate. Furthermore, although insufficient oxygen can contribute to the production of lactate, the presence of lactate does not absolutely indicate a lack of oxygen. The presence of lactate simply reflects the use of the anaerobic glycolytic pathway for ATP production and the balance between glycolytic and mitochondrial activity. Furthermore, rather than lactate being a “waste product,” lactate provides a means of coordinating carbohydrate metabolism in diverse tissues (Brooks, 2020; Brooks et al., 1999). The formation, distribution, and utilization of lactate is a way for glycogen reserves to be mobilized and used within either the working muscle cell or other cells. In the process, blood glucose is spared for use by other tissues (Brooks, 2002; Gladden, 2004). The use or reconversion of lactate, or both, constitute lactate clearance.

Lactate Clearance

Blood lactate levels reflect the balance between lactate production (appearance) and clearance (removal). Lactate clearance (**Figure 3.5**) occurs primarily by three processes: oxidation (50–75%), gluconeogenesis/glyconeogenesis (10–25%), and transamination (5–10%). All three processes can involve the movement of lactate, either within or between cells. A small amount is excreted in sweat.

As stated previously, although produced as lactic acid, at physiological pH more than 99% quickly dissociates into lactate (La^-) anions and protons (H^+) (Gladden, 2004). Lactate moves

readily between cytoplasm and mitochondria, muscle and blood, blood and muscle, active and inactive muscle, glycolytic and oxidative muscle, blood and heart, blood and liver, and blood and skin (Brooks, 2000). Lactate moves between lactate-producing and lactate-consuming sites through intracellular and extracellular lactate shuttles (Brooks, 2002). Transport across cellular and mitochondrial membranes occurs by facilitated exchange down concentration and hydrogen ion (pH) gradients using lactate transport proteins known as monocarboxylate transporters (MCTs) (Brooks, 2018, 2020; Ferguson et al., 2018; Gladden, 2004; Robergs, 2018).

To date, 14 monocarboxylate transporters had been reported in the literature. Two of these, MCT1 and MCT4, are important in skeletal muscles. MCT1 is abundant in oxidative skeletal and cardiac muscle fibers and mitochondrial membranes. Its primary role is to take up lactate from the circulation. It also functions to shuttle lactate between and within muscle fibers. MCT4 is most prevalent in the cell membranes of glycolytic skeletal fibers and is especially suited for lactate/lactic acid export from those cells. MCT1 and MCT4 transport lactate and protons, thus playing important roles in regulating intracellular pH since they facilitate most of the H^+ efflux from working muscles. Acute exercise produces an increase in both MCT1 and MCT4 with the change being greater in MCT1 than MCT4 (Thomas et al., 2012).

The *intracellular lactate shuttle* (Figure 3.6A) involves the movement of lactate from where it is produced in the cytoplasm (under the influence of LDH 4 or 5), into the mitochondria through pores in the outer membrane (Brooks, 2018, 2020; Ferguson et al., 2018). Within the inner mitochondrial membrane, a mitochondrial lactate oxidation complex (mLOC) exists. It consists of MCT1 interacting with its chaperone protein CD147 associated with Complex IV of the electron transport chain as well as LDH1 on the outside surface. Lactate is oxidized to pyruvate via this mitochondrial lactate oxidation complex. NAD^+ is reduced to $NADH + H^+$. The pyruvate proceeds through stages II, III, and IV of aerobic metabolism, while the $NADH + H^+$ is reoxidized by the malate-aspartate shuttle and enters stage IV at Complex I. Thus, muscle cells can both produce and consume lactate at the same time (Gladden, 2008a; Hashimoto and Brooks, 2008; Kane, 2014; Rasmussen et al., 2002; Sahlin et al., 2002;

Thomas et al., 2012).

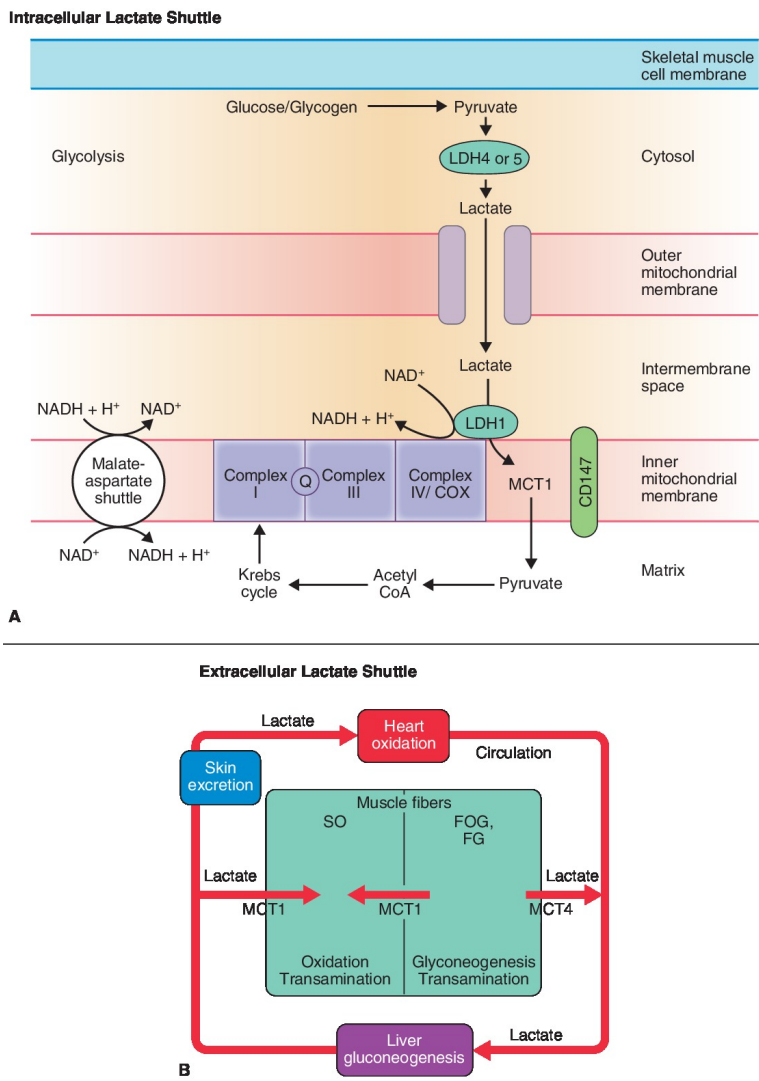


Figure 3.6 Intracellular and Extracellular Lactate Shuttles.

A. Lactate, which is always produced in the cytosol of skeletal muscle cells because of the characteristics and abundance of LDH 4 or 5, enters the mitochondrial intermembrane space through a passive diffusion pore in

the outer mitochondrial membrane. Here, LDH1 reduces NAD^+ to $\text{NADH} + \text{H}^+$. Pyruvate crosses the inner mitochondrial membrane via MCT1 stabilized by CD147. The $\text{NADH} + \text{H}^+$ is reoxidized by the malate-aspartate shuttle and transported into the mitochondrial matrix and enters the ETS at Complex I. The pyruvate is oxidized to acetyl CoA, which enters the Krebs cycle and finally the ETS. **B.** The extracellular lactate shuttle moves lactate directly between lactic acid-producing FG fibers (FOG and FG) and lactate-consuming SO fibers. It also transports lactate through the circulation to the liver, skin, and heart, where it is cleared by oxidation, transamination, gluconeogenesis, or excretion. Most lactate remaining in FG fibers is reconverted to glycogen by glycogenesis.

Sources: ([Brooks, 2018, 2020](#); [Ferguson et al., 2018](#)).

Extracellular lactate shuttles act to move lactate between tissues (**Figure 3.6B**). Muscle cell membrane lactate proteins (MCT1 and MCT4) move the lactate both out of and into tissues. Intermuscularly, most lactate moves out of active fast-twitch glycolytic skeletal muscle cells (both fast-twitch oxidative glycolytic [FOG] and fast-twitch glycolytic [FG]) and into active slow-twitch oxidative (SO) skeletal muscle cells. This can occur either by a direct shuttle between the skeletal muscle cells or through the circulation. Once lactate is in the bloodstream, it can also circulate to cardiac cells. During heavy exercise, lactate becomes the preferred fuel of the heart. In addition, when transported to the brain, lactate is involved in metabolism, signaling, and executive functions. In this manner, glycogenolysis in one cell can supply fuel for another cell. In each of these cases, the ultimate fate of the lactate is oxidation to ATP, CO_2 , and H_2O by aerobic metabolism ([Brooks, 1986, 2000, 2018, 2020](#); [Chatham, 2002](#); [Emhoff et al., 2013](#)).

Lactate circulating in the bloodstream can also be transported to the liver where, as described in [Chapter 2](#), it is reconverted into glucose by the process of gluconeogenesis. Indeed, the liver appears to preferentially make glycogen from lactate rather than glucose. In human glycolytic muscle fibers (both fast-twitch

oxidative glycolytic and fast-twitch glycolytic), some of the lactate produced during high-intensity exercise is retained, and in the postexercise recovery period, it is reconverted to glycogen in that muscle cell. This process is called glyconeogenesis (Brooks et al., 1999; Donovan and Pagliassotti, 2000; Emhoff et al., 2013; Gladden, 2000).

Both oxidative and glycolytic fibers can also clear lactate by transamination. Transamination forms keto acids (Krebs cycle intermediates) and amino acids. The predominant amino acid produced is alanine. In turn, alanine can undergo gluconeogenesis in the liver (Brooks, 1986; Emhoff et al., 2013; Gaesser and Brooks, 1975).

A small amount of lactate in the circulation moves from the blood to the skin and exits the body in sweat. Finally, some lactate remains as lactate circulating in the blood. This constitutes the resting lactate concentration.

Oxidation is by far the predominant process of lactate clearance both during and after exercise. The accumulation of lactate in the blood depends on the relative rate of appearance (production) and disappearance (clearance), which in turn is directly related to the intensity and duration of the exercise being done.

Measurement of Anaerobic Metabolism

Laboratory Procedures

Unfortunately, there is no generally accepted means by which to directly measure the contribution of anaerobic energy to exercise. Two general approaches are used, however, to describe the anaerobic exercise response. One approach describes changes in the chemical substances either used in alactic anaerobic metabolism (specifically, ATP and PC levels) or produced as a result of lactic anaerobic metabolism (lactate). The second approach quantifies the amount of work performed or the power generated during short-duration, high-intensity activity. The assumption is that such activity could not be done without anaerobic energy; therefore, measuring such work or power

indirectly measures anaerobic energy utilization and provides an indication of anaerobic capacity.

ATP-PC and Lactate

Muscle ATP, PC, and lactate can be measured by chemical analysis of muscle biopsy specimens. Lactate is the most frequently measured variable, in part because it can also be measured from blood samples. The blood sample may be obtained by venipuncture or by finger or ear lobe prick, all of which are less invasive than a muscle biopsy. Another reason for the popularity of lactate analysis is the availability of user-friendly, fast, accurate, and relatively inexpensive analyzers. **Figure 3.7** shows an analyzer that requires a minimal blood sample (a 25- μ L capillary tube obtained by a finger prick); it is portable and takes less than 5 minutes for each sample analysis. Even smaller, less expensive, handheld analyzers are available (**Figure 3.7C**).



A



B



C

Figure 3.7 Lactate Analysis.

A. Acquiring a lactate sample by finger prick. **B.** Inserting the sample into a laboratory style analyzer. **C.** Using a

handheld lactate analyzer.

Lactate is a small molecule that moves easily from the muscles to the blood and most other fluid compartments. However, much of the lactate that is produced does not get into the bloodstream. It takes time for the portion that does get into the bloodstream to reach equilibrium between muscle and blood. This equilibrium and the achievement of peak blood values may take as long as 5–10 minutes. Until equilibrium occurs, muscle lactate values will be higher than blood lactate values. This also means that the highest blood lactate values are typically seen after several minutes of recovery, not during high-intensity work (Gollnick and Hermansen, 1973).

Lactate levels are reported using a variety of units. The two most common are millimoles per liter ($\text{mmol}\cdot\text{L}^{-1}$, sometimes designated as mM) or milligrams per 100 mL of blood ($\text{mg}\cdot 100\text{ mL}^{-1}$, sometimes designated as mg% or $\text{mg}\cdot\text{dL}^{-1}$). One $\text{mmol}\cdot\text{L}^{-1}$ is equal to $9\text{ mg}\cdot 100\text{ mL}^{-1}$. Resting levels of lactate of $1\text{--}2\text{ mmol}\cdot\text{L}^{-1}$ or $9\text{--}18\text{ mg}\cdot 100\text{ mL}^{-1}$ are typical. A value of $8\text{ mmol}\cdot\text{L}^{-1}$ or $72\text{ mg}\cdot 100\text{ mL}^{-1}$ is usually taken to indicate that an individual has worked maximally (Åstrand et al., 2003). Peak values as high as $32\text{ mmol}\cdot\text{L}^{-1}$ or $288\text{ mg}\cdot 100\text{ mL}^{-1}$ have been reported.

Tests of Anaerobic Power and Capacity

Energy system capacity is the total amount of energy that can be produced by an energy system. **Energy system power** is the maximal amount of energy that can be produced per unit of time. **Table 3.1** clearly shows that the phosphagen (ATP-PC) system is predominantly a power system with very little capacity. The lactic anaerobic glycolytic system has almost equal power and capacity, just slightly favoring capacity. The information on the aerobic (O_2) system is included here just to show how truly high in power and low in capacity the anaerobic systems are.

Energy System Capacity The total amount of energy that can be produced by an energy system.

Energy System Power The maximal amount of energy that can be produced per unit of time.

TABLE 3.1 Estimated Maximal Power and Capacity in Untrained Males

| Energy System | Power | | Time | Capacity | |
|--|------------------------|----------------------|----------|-----------|-------------|
| | kcal·min ⁻¹ | kJ·min ⁻¹ | | kcal | kJ |
| ATP-PC (phosphagen) | 72 | 300 | :09–:10 | 11 | 45 |
| LA (anaerobic glycolysis) | 36 | 150 | 1:20 | 48 | 200 |
| O ₂ (aerobic glycolysis + Krebs cycle + ETS/OP; fuel = CHO) | 7.2–19.1 | 30–80 | 2:21:00* | 359–1,268 | 1,500–5,300 |

*When all fuels are considered, the time is unlimited.

Sources: Bouchard et al. (1982, 1991).

Although the ATP-PC system can put out energy at the rate of 72 kcal·min⁻¹, it can sustain that value when working maximally only for 9–10 seconds, for a total output of only 11 kcal (72 kcal·min⁻¹/60 sec·min⁻¹ = 1.2 kcal·sec⁻¹; 11 total kcal/1.2 kcal·sec⁻¹ = 9.17 seconds). The LA system has a lower power (36 kcal·min⁻¹) but can sustain it for almost 1 minute and 20 seconds (48 kcal/36 kcal·min⁻¹ = 1.33 minutes = 1:20). By comparison, if the O₂ system worked at a power output of 9 kcal·min⁻¹, exercise could be sustained for more than 2 hours (1,268 kcal/9 kcal·min⁻¹ = 141 minutes = 2:21) just using carbohydrate fuel sources. In fact, when all fuel supplies are included within a healthy, well-fed body, the capacity of the aerobic system is, for all intents and purposes, unlimited.

For measuring the anaerobic systems, an ideal test would distinctly evaluate alactic anaerobic power, alactic anaerobic capacity, lactic anaerobic power, and lactic anaerobic capacity. Because no such test exists, attempts have been made to get this information indirectly by measuring (1) the total mechanical power generated during high-intensity, short-duration work; (2) the amount of mechanical work done in a specific period of time; or (3) the time required to perform a given amount of presumably anaerobic work (Green, 1995). Two such tests are commonly used in laboratory settings: the Wingate Anaerobic Test (WAnT) and the Margaria-Kalamen Stair Climb (Bouchard et al., 1982).

THE WINGATE ANAEROBIC TEST The Wingate Anaerobic Test (WAnT) depicted in **Figure 3.8** is probably the most well-known of several bicycle ergometer tests used to measure anaerobic power and capacity and is considered to be the “gold standard” or criterion test. The test traditionally begins with pedaling against little or no resistance and then is an all-out ride for 30 seconds against a resistance based on body weight. Both arm and leg versions are available, although the leg test is most frequently used and will be the only version discussed here. Resistance values of $0.075 \text{ kg} \cdot \text{kg}^{-1}$ body weight for children, $0.085 \text{ kg} \cdot \text{kg}^{-1}$ body weight for adult females, and $0.095 \text{ kg} \cdot \text{kg}^{-1}$ body weight for adult males appear to be optimal although research continues in this area (Jaafar et al., 2014). Athletes may need values as high as $0.10 \text{ kg} \cdot \text{kg}^{-1}$ of body weight, but the most common value used (as in the example that follows) is $0.075 \text{ kg} \cdot \text{kg}^{-1}$ body weight (Vandewalle et al., 1987). The revolutions (rev) of the flywheel are counted per second during the test, and from the available information, three variables are determined.



Figure 3.8 Wingate Anaerobic Test.

This individual is performing the WAnT. Once she is pedaling as fast as she can, the tester releases the weight, providing a resistance based on the subject’s body weight. The individual then continues to pedal as quickly as possible for 30 seconds. Data from sensors attached to the flywheel are relayed to the computer for calculation of peak power, mean power, and fatigue index.

Computer-generated results from a typical test with a resistance of 4.4 kg are given in **Table 3.2**. The subject was a 129-lb female physical education major, but not an athlete. The data can be used to calculate three variables: peak power, mean power, and fatigue index. **Peak power (PP)** is the maximal mechanical power (force times distance divided by time) exerted during very short duration (5 seconds or less) work. **Mean power (MP)** is the average mechanical power (force times distance divided by time) exerted during short duration (typically 30 seconds) work. The **fatigue index (FI)** is the percentage of peak power (PP) drop-off during high-intensity, short-duration work. Each of these variables requires the use of different time periods in the calculations.

TABLE 3.2 Results for the Wingate Leg Test

Body weight is 129 lb = 58.50 kg; resistance for legs is
 $58.50 \text{ kg} \times 0.075 \text{ kg} \cdot \text{kg}^{-1} \text{ body weight} = 4.4 \text{ kg}$

| Inclusive Time (sec) | 5-sec Total Revolutions | $\text{kgm} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ | $\text{W} \cdot \text{kg}^{-1}$ |
|-------------------------|----------------------------|---|---------------------------------|
| 1–5 | 10.00 | 54.15 | 8.85 |
| 2–6 | 10.25 | 55.50 | 9.07 |
| 3–7 | 9.75 | 52.80 | 8.63 |
| 4–8 | 9.50 | 51.44 | 8.41 |
| 5–9 | 9.25 | 50.09 | 8.18 |
| 6–10 | 9.00 | 48.74 | 7.96 |
| 7–11 | 8.25 | 44.67 | 7.30 |
| 8–12 | 8.00 | 43.32 | 7.08 |
| 9–13 | 7.75 | 41.97 | 6.86 |
| 10–14 | 7.50 | 40.61 | 6.64 |
| 11–15 | 7.50 | 40.61 | 6.64 |
| 12–16 | 7.50 | 40.61 | 6.64 |
| 13–17 | 7.25 | 39.26 | 6.41 |
| 14–18 | 7.25 | 39.26 | 6.41 |
| 15–19 | 7.00 | 37.91 | 6.19 |
| 16–20 | 7.00 | 37.91 | 6.19 |
| 17–21 | 6.75 | 36.55 | 5.97 |
| 18–22 | 6.75 | 36.55 | 5.97 |
| 19–23 | 6.50 | 35.20 | 5.75 |
| 20–24 | 6.50 | 35.20 | 5.75 |
| 21–25 | 6.25 | 33.84 | 5.53 |
| 22–26 | 6.25 | 33.84 | 5.53 |
| 23–27 | 6.00 | 32.49 | 5.31 |
| 24–28 | 6.00 | 32.49 | 5.31 |
| 25–29 | 5.75 | 31.14 | 5.09 |
| 26–30 | 5.50 | 29.78 | 4.87 |

Total pedal revolutions = 45.25.

Highest 5-sec absolute peak power = 3,247.20 $\text{kgm} \cdot \text{min}^{-1}$.

Highest 5-sec relative peak power = $55.50 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{kg}^{-1} =$

$9.07 \text{ W}\cdot\text{kg}^{-1}$.

Mean absolute power = $2,389.20 \text{ kgm}\cdot\text{min}^{-1}$.

Mean relative power = $40.84 \text{ kgm}\cdot\text{min}^{-1}\cdot\text{kg}^{-1} = 6.67 \text{ W}\cdot\text{kg}^{-1}$.

Fatigue index = $100 - (\text{lowest 5 sec divided by highest 5 sec}) \times 100 = 46.34\%$.

Peak Power (PP) The maximum power (force times distance divided by time) exerted during very short duration (5 seconds or less) work.

Mean Power (MP) The average power (force times distance divided by time) exerted during short duration (typically 30 seconds) work.

Fatigue Index (FI) Percentage of peak power drop-off during high-intensity, short-duration work.

The first variable computed is peak power (PP), the maximum power exerted during the highest 5-second period. This usually occurs early in the activity and, in the example given, is the time between 2 and 6 seconds when 10.25 rev were completed. Peak power can be expressed in absolute terms as $\text{kgm}\cdot 5 \text{ sec}^{-1}$ or prorated to a full minute as $\text{kgm}\cdot\text{min}^{-1}$ or watts (W). Peak power can also be expressed relative to body weight as $\text{kgm}\cdot 5 \text{ sec}^{-1}\cdot\text{kg}^{-1}$, $\text{kgm}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$, or $\text{W}\cdot\text{kg}^{-1}$. The formula for peak power is

peak power ($\text{kgm}\cdot 5 \text{ sec}^{-1}$) = maximal revolutions
in 5 seconds \times distance that the flywheel travels
per revolution (m) \times force setting (kg)

or

$$3.1 \quad \text{PP} = \text{rev (max) in 5 seconds} \times D \cdot \text{rev}^{-1} \times F$$

Example

Thus, for this example, the calculation is

$$PP = 10.25 \text{ rev} \times 6 \text{ m} \cdot \text{rev}^{-1} \times 4.4 \text{ kg} = 270.6 \text{ kgm} \cdot 5 \text{ sec}^{-1}$$

Prorated to a full minute, PP is

$$\begin{aligned} PP &= 270.6 \text{ kgm} \cdot 5 \text{ sec}^{-1} \times 12 \\ &\quad (60 \text{ sec} \cdot \text{min}^{-1} \div 5 \text{ sec} = 12) \\ &= 3,247.2 \text{ kgm} \cdot \text{min}^{-1} \end{aligned}$$

Relative to body weight PP is

$$\begin{aligned} PP &= 3,247.2 \text{ kgm} \cdot \text{min}^{-1} \div 58.5 \text{ kg} \\ &= 55.50 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{kg}^{-1} \end{aligned}$$

When converted to watts ($1 \text{ W} = 6.12 \text{ kgm} \cdot \text{min}^{-1}$), the relative value is

$$\begin{aligned} PP &= 55.5 \text{ kgm} \cdot \text{min}^{-1} \div 6.12 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{W}^{-1} \\ &= 9.07 \text{ W} \cdot \text{kg}^{-1} \end{aligned}$$

Peak power was originally thought to reflect only alactic processes—alactic anaerobic capacity, in particular. However, subsequent research has shown that muscle lactate levels rise to high values as early as 10 seconds into such high-intensity work. This indicates that glycolytic processes are occurring almost immediately along with ATP-CP breakdown. Therefore, peak power cannot be interpreted as being only alactic though it is predominantly alactic (Bar-Or, 1987; Beneke et al., 2002; Bertuzzi et al., 2015).

The second variable calculated is mean power, which can also be expressed in both absolute and relative units. Mean power is the average power sustained throughout the 30-second ride. The

formula for mean power is

mean power ($\text{kg} \cdot 30 \text{ sec}^{-1}$) = total number of revolutions in 30 seconds \times distance that the flywheel travels per revolution (m) \times force setting (kg)

or

$$3.2 \quad \text{MP} = \text{rev (total) in 30 seconds} \times D \cdot \text{rev}^{-1} \times F$$

Example

Thus, for this example, the calculation is

$$\begin{aligned} \text{MP} &= 45.25 \text{ rev} \times 6 \text{ m} \cdot \text{rev}^{-1} \times 4.4 \text{ kg} \\ &= 1,194.6 \text{ kgm} \cdot 30 \text{ sec} \end{aligned}$$

Prorated to a full minute, MP is

$$\begin{aligned} \text{MP} &= 1,194.6 \text{ kgm} \times 2 \text{ (} 60 \text{ sec} \cdot \text{min}^{-1} \div 30 \text{ sec} = 2 \text{)} \\ &= 2,389.2 \text{ kgm} \cdot \text{min}^{-1} \end{aligned}$$

Related to body weight MP is

$$\begin{aligned} \text{MP} &= 2,389.2 \text{ kgm} \cdot \text{min}^{-1} \div 58.5 \text{ kg} \\ &= 40.84 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{kg}^{-1} \end{aligned}$$

When converted to watts, MP is

$$\begin{aligned} \text{MP} &= 40.84 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{kg}^{-1} \div 6.12 \text{ kgm} \cdot \text{min}^{-1} \cdot \text{W}^{-1} \\ &= 6.67 \text{ W} \cdot \text{kg}^{-1} \end{aligned}$$

Mean power is sometimes said to represent lactic anaerobic capacity. Although lactic anaerobic metabolism is not the only energy system operating during this time, it is the predominant energy system ([Bar-Or, 1987](#); [Beneke et al., 2002](#); [Bertuzzi et al., 2015](#)).

The third variable that can be computed is the fatigue index (FI) or the percentage of peak power dropoff during high-intensity, short-duration work. It is calculated using the highest 5-second power (PP) and the lowest 5-second power (LP). Little is known about the relationship of the fatigue index to anaerobic fitness (Bar-Or, 1987).

$$\text{Fatigue index (\%)} = 1 - \left[\frac{\text{lowest power kgm} \cdot 5 \text{ sec}^{-1}}{\text{peak power kgm} \cdot 5 \text{ sec}^{-1}} \right] \times 100$$

or

$$3.3 \quad \text{FI} = [1 - (\text{LP} \div \text{PP})] \times 100$$

Example

In our example, the highest 5-second power is 270.6 kgm. Using the peak power formula of Equation 3.1 but with the lowest 5-second total, we get

$$\begin{aligned} \text{LP} &= 5.50 \text{ rev} \times 6 \text{ m} \cdot \text{rev}^{-1} \times 4.4 \text{ kg} \\ &= 145.2 \text{ kg} \cdot 5 \text{ sec}^{-1} \\ \left[1 - \left(145.2 \text{ kg} \cdot 5 \text{ sec}^{-1} \div 270.6 \text{ kg} \cdot 5 \text{ sec}^{-1} \right) \right] \\ &\times 100 = 46.34\% \end{aligned}$$

Although the WAnT is not a purely anaerobic test (a meaningful aerobic component of ~19–22% has been measured), it is predominantly (~80%) anaerobic (with ~31–33% of the energy being supplied by the ATPPC system and 45–51% by the lactate system (Beneke et al., 2002, 2007). It compares well (with correlations generally above 0.75) with other tests of anaerobic power and capacity and is widely used (Bar-Or, 1987; Patton and Duggan, 1987).

THE MARGARIA-KALAMEN STAIR CLIMB To perform the Margaria-Kalamen test, an individual runs for 6 m on level ground and then climbs a staircase, taking three steps at a time. Power in kgm·sec⁻¹ is calculated from the weight of the subject,

the vertical height between the third and ninth steps, and the time between the third and ninth steps (Bouchard et al., 1982; Vandewalle et al., 1987). The use of electronic switch mats or photoelectric cells is essential for accuracy. This test is considered to be a test of alactic anaerobic power because of the short time involved—usually less than 5 seconds for the entire test and close to 1 second for the measured time between the third and ninth steps.

Field Tests

No field tests are available to estimate the ATP-PC used or the lactate produced during exercise. However, performance in high-intensity, short-duration activity can give an indication of anaerobic power and capacity. Sportspecific tests are available, but two types of activities are commonly used: vertical jump tests and sprints (sometimes done as shuttles) or middle-distance runs.

In the single vertical jump tests, several different protocols are used, including variations in the starting posture, continuation of movement, and use or nonuse of arms. There are two primary forms of the vertical jump test: the squat jump (in which individuals lower themselves into a squat position, pause ~2 seconds, and jump quickly as high as possible) and the counter-movement jump (in which there is no pause between moving from standing to squat to jumping). The criterion measurement is power (expressed in Watts) from a ground reaction force plate. In a field situation, the primary measurement is height jumped. The height of the vertical jump and peak power as determined from force plate data has been shown to be highly correlated (Payne et al., 2000). In addition, peak power values calculated from the vertical jump and both sprints and the Wingate Anaerobic Test are highly correlated in both males and females (Alemdaroglu, 2012; Almansba et al., 2019; Hoffman et al., 2000; Musa & Toriola, 2006; Nikolaidis et al., 2016, 2017; Sales et al., 2018; Vandewalle et al., 1987).

There are several equations in the literature for converting vertical jump height to peak power (PP). The ones most accepted for adults were derived by Sayers et al. (1999):

For the squat jump:

$$\text{Peak power (W)} = 60.7 \times \text{SJ height (cm)} + 45.3 \\ \times \text{body mass (kg)} - 2055$$

For the countermovement jump:

$$\text{Peak Power (W)} = 51.9 \times \text{CMJ height (cm)} + 48.9 \\ \times \text{body mass (kg)} - 2007$$

Adult equations should not be used for children and adolescents. One valid equation for this group was developed by [Gomez-Bruton et al. \(2019\)](#):

For the countermovement jump:

$$\text{Peak power (W)} = 54.2 \times \text{CMJ height (cm)} + 34.4 \\ \times \text{body mass (kg)} - 1520.4$$

Example:

13 yr-old girl; weight = 99 lbs (45 kg);

VJ height = 12.5 in (31.65 kg)

$$\text{PP (W)} = (54.2 \times 31.65) + (34.4 \times 45) - 1520 = 1743.03$$

For sprints, shuttle, or middle-distance runs, the involvement of the various energy systems is related to the time of the all-out activity, as shown in **Figure 3.3**. Therefore, runs whose distance can be covered in a distinct time range (depending somewhat on age, sex, and training status) can be used as field tests of anaerobic metabolism ([Cheetham et al., 1986](#); [Thomas et al., 2002](#)). For example, dashes of 40, 50, or 60 yd or m will take approximately 4–15 seconds and can be used as an indication of alactic anaerobic power and/or capacity. Longer runs, probably between 200 and 800 m (or 220 and 880 yd) and lasting 30–120 seconds, can be used as an indication of lactic anaerobic power and capacity. Faster speeds covering a given distance would indicate increased anaerobic power and/or capacity. The Hawaii Anaerobic Run Test of 200 m with each 25 m split being timed allows for the calculation of peak momentum, mean momentum, and fatigue index that has been shown to be reliable and valid when compared to the WAnT ([Kimura et al., 2014](#)).

The Anaerobic Exercise Response

Oxygen Deficit and Excess Postexercise Oxygen Consumption

When exercise begins, regardless of how light or heavy it is, there is an immediate need for additional energy. Thus, the most obvious exercise response is an increase in metabolism. All three energy systems are involved in this response, their relative contributions being proportional to the intensity, and duration of the activity.

Figure 3.9 shows two scenarios for going from rest to different intensities of exercise. In **Figure 3.9A**, the activity is a moderate submaximal bout. The oxygen requirement for this exercise is $1.4 \text{ L}\cdot\text{min}^{-1}$. The individual has a $\dot{V}\text{O}_{2\text{max}}$ of $2.5 \text{ L}\cdot\text{min}^{-1}$. Therefore, this individual is working at $56\% \dot{V}\text{O}_{2\text{max}}$. The area under the smoothed curve during both exercise and recovery represents oxygen used. Notice, however, that there is an initial lag during which the oxygen supplied and utilized is below the oxygen requirement for providing energy. This difference between the oxygen required during exercise and the oxygen supplied and utilized is called the **oxygen deficit**. Because of this discrepancy between supply and demand, anaerobic sources must be involved in providing energy at the onset of all activity.

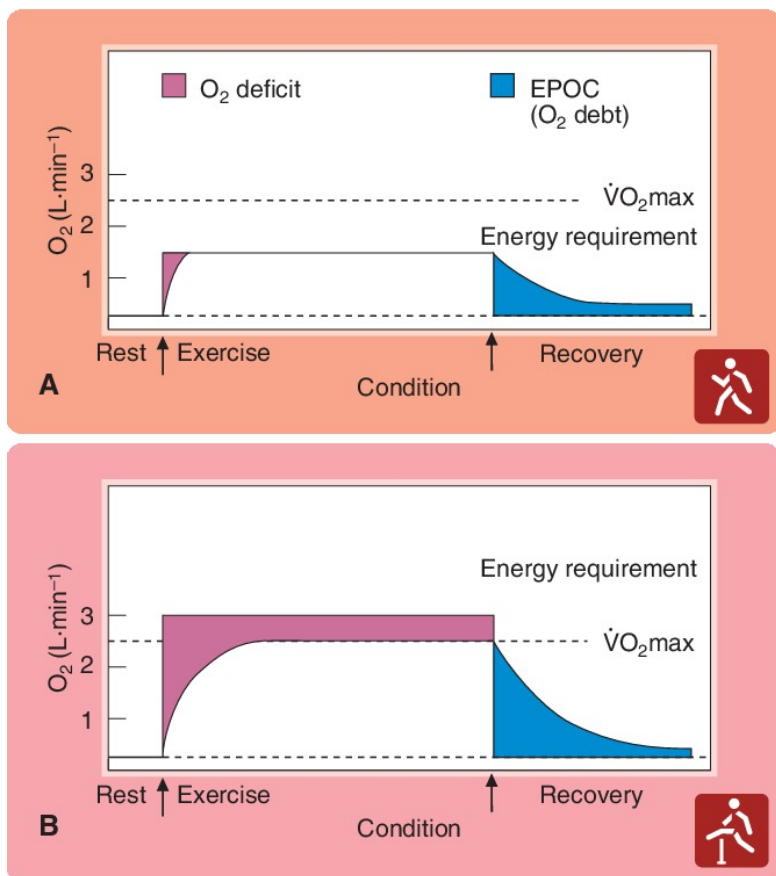


Figure 3.9 Oxygen Deficit and Excess Postexercise Oxygen Consumption (EPOC) during Submaximal Exercise and Supramaximal Exercise.

A. During light to moderate submaximal exercise, both the oxygen deficit, indicated by the convex curve at the start of exercise, and the excess postexercise oxygen consumption, indicated by the concave curve during the recovery time period, are small. **B.** During heavy or supramaximal exercise, both the O₂ deficit and the EPOC are large. Under both conditions, energy is supplied during the O₂ deficit period by using stored ATP-PC, anaerobic glycolysis, and oxygen stores in capillary blood and bound to myoglobin. The heavier the exercise, the more the reliance on anaerobic

glycolysis. The EPOC is a result of the restoration of ATP-PC, removal of lactate, restoration of O₂ stores, elevated cardiovascular and respiratory function, elevated hormonal levels, and especially, the elevated body temperature.

Oxygen Deficit The difference between the oxygen required during exercise and the oxygen supplied and utilized. Occurs at the onset of all activity.

Evidence indicates that the O₂ deficit is probably due to limited cellular utilization of O₂ as a result of metabolic adjustments in both the anaerobic and aerobic systems. The relatively slow response time of aerobic ATP production is determined by the faster speed of response of the ATP-PC system and by the content of mitochondria in the muscle. The fact that the ATP-PC system responds rapidly does not indicate any intrinsic delay or inertia of activation for either the LA or O₂ systems. All three pathways respond simultaneously in an integrated fashion. The metabolic response is regulated by a series of feedback control systems that are sensitive to the release of Ca²⁺ from muscle contraction and the breakdown of ATP (Grassi, 2005; Hughson et al., 2001; Meyer and Wiseman, 2012; Sahlin et al., 1988). The metabolic systems simply respond at different speeds. Therefore, during the transition from rest to work, energy is supplied by

1. O₂ transport and utilization
2. Utilization of O₂ stores in capillary blood and bound to myoglobin
3. The splitting of stored ATP-PC
3. Anaerobic glycolysis, with the concomitant production of lactic acid

Eventually, if the exercise intensity is low enough (as in the example in **Figure 3.9A**), the aerobic system will predominate and the oxygen supply will equal the oxygen demand. This condition is called steady-state, steady-rate, or steady-level

exercise.

Figure 3.9B shows a smoothed plot of O₂ consumption at rest and during and after an exercise bout in which the energy requirement is greater than $\dot{V}O_{2\max}$, sometimes called **supramaximal exercise**. The initial lag period between O₂ supply and demand is once again evident, and as in the first example, the added energy is provided by stored ATP-PC, anaerobic glycolysis, and stored O₂. However, in this case, when the O₂ consumption plateaus or levels off, it is at $\dot{V}O_{2\max}$, and more energy is still needed if exercise is to continue.

Supramaximal Exercise An exercise bout in which the energy requirement is greater than what can be supplied aerobically at $\dot{V}O_{2\max}$.

This plateau is not considered to be a steady state because the energy demands are not totally being met aerobically. Supplemental energy is provided by anaerobic glycolysis. The exact energy demand in this situation is difficult to determine precisely because, as stated before, lactic acid levels do not reflect production alone. However, without the anaerobic energy contribution, this activity could not continue. The maximal ability to tolerate lactic acid accumulation will determine to a large extent how long the activity can continue.

A practical application of oxygen deficit theory is a measurement called *maximal accumulated oxygen deficit (MAOD)*. MAOD is the difference between the estimated accumulated oxygen demand (based on an extrapolation from 4 to 10 bouts of at least 4 minutes each of submaximal exercise and a maximal test to determine power output-oxygen uptake relationships) and the accumulated oxygen uptake measured during exhausting supramaximal exercise of 110–125% $\dot{V}O_{2\max}$ (Bortolotti et al., 2010; Medbo et al., 1988; Noordhof et al., 2010). MAOD relates highly to the WAnT and is interpreted as a direct measure of anaerobic capacity (Bertuzzi et al., 2015; Weber and Schneider, 2001). During recovery from exercise, represented by the concave curves after exercise in **Figure 3.9A and B**, oxygen consumption

drops quickly (a fast component lasting 2–3 minutes) and then tapers off (a slow component lasting 3–60 minutes). The magnitude and duration of this elevated postexercise oxygen consumption depends on the duration, intensity, and modality of the preceding exercise. After light submaximal work (**Figure 3.9A**), recovery takes place quickly; after heavy exercise (**Figure 3.9B**), recovery takes much longer. Note that although the oxygen consumption is expressed as $\text{L}\cdot\text{min}^{-1}$ in this figure, it can also be prorated by body weight and expressed as $\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$. The term *O₂ recovery* or, more commonly, **excess postexercise oxygen consumption (EPOC)** is used to describe this phenomenon. That is, EPOC is defined as the oxygen consumption during recovery that is above normal resting values. The relationship between the size of the EPOC and the intensity of aerobic exercise is curvilinear, whereas the relationship between the size of the EPOC and the duration of aerobic exercise is more linear ([Mann et al., 2014](#)). The relationship between EPOC and resistance exercise is less well defined, but intensity seems important here as well ([Abboud et al., 2013](#); [Greer et al., 2015](#)). A comparison study between a single bout of resistance exercise, moderate-intensity steady-state exercise, and high-intensity intermittent aerobic exercise revealed that both resistance exercise and high-intensity interval exercise increased EPOC to a greater degree than did moderate-intensity steady-state exercise ([Greer et al., 2015](#)). However, the increase in EPOC following resistance exercise was not different between moderate (10,000 kg) and high (20,000 kg) load volumes, despite high load volumes leading to more muscle damage ([Abboud et al., 2013](#)).

Excess Postexercise Oxygen Consumption (EPOC) Oxygen consumption during recovery that is above normal resting values.

A critical question is what causes this elevated metabolism in recovery. Although EPOC cannot be completely explained at this time, seven factors have been suggested. Factors 1 and 2 are known as the “fast” components of EPOC, as they are achieved more quickly, and factors 3–7 are considered “slow” components

of EPOC, as they take longer to occur.

1. Restoration of ATP-PC stores: About 10% of the EPOC is utilized to rephosphorylate creatine and ADP to PC and ATP, respectively, thus restoring these substances to resting levels (Bahr, 1992; Gaesser and Brooks, 1975). Approximately 50% of the ATP-PC is restored in 30 seconds (Margarita et al., 1933). This time is called the half-life restoration of ATP-PC. Full recovery requires 2–8 minutes (Fox, 1973; Harris et al., 1976; Hultman et al., 1967).
2. Lactate removal: The lactate that has accumulated must be removed. However, its contribution as a causative factor for EPOC is minimal (Gaesser and Brooks, 1975; Stainsby and Barclay, 1970).
3. Restoration of O₂ stores: Although the amount of O₂ stored in the blood (bound to hemoglobin) and muscle (bound to myoglobin) is not large, it does need to be replenished when exercise stops. Replenishment probably occurs completely within 2–3 minutes (Bahr, 1992; Stainsby and Barclay, 1970).
4. Elevated cardiovascular-respiratory function: Both the respiratory system and the cardiovascular system remain elevated postexercise; that is, neither the breathing rate and depth nor heart rate recovers instantaneously. Although this enables the extra amounts of oxygen to be processed, the actual energy cost of these cardiovascular-respiratory processes probably accounts for only 1–2% of the excess oxygen (Bahr, 1992; Stainsby and Barclay, 1970).
5. Elevated hormonal levels: During exercise, the circulating levels of the catecholamines (epinephrine and norepinephrine), thyroxine, and cortisol are all increased (see Figure 2.17 and Table 2.3 in Chapter 2). In addition to their fuel mobilization and utilization effects, these hormones increase Na⁺–K⁺ pump activity in muscles and nerves by changing cell membrane permeability to Na⁺ and K⁺. As an active transport process, the Na⁺–K⁺ pump requires ATP. The increased need for ATP means an increased need for O₂. Until the hormones are cleared from the bloodstream, the additional O₂ and ATP use is a significant contributor to the

EPOC (Bahr, 1992; Gaesser and Brooks, 1975).

6. Elevated body temperature: When ATP is broken down to supply the energy for chemical, electrical, or mechanical work, heat is produced as a by-product. During exercise, heat production may exceed heat dissipation, causing a rise in body core temperature. For each degree Celsius that body temperature rises, the metabolic rate increases approximately 13–15% (Kapit et al., 2000). Thus, in recovery, although high levels of energy are no longer needed to support the exercise, the influence of the elevated temperature remains, because cooling takes some time to occur. This temperature effect is by far the most important reason for EPOC, accounting for as much as 60–70% of the slow component after exercise at 50–80% $\dot{V}O_2\text{max}$ (Bahr, 1992; Gaesser and Brooks, 1975).
7. Energy substrate shift: During recovery, the primary fuel utilized shifts from carbohydrate to fat. Fat requires more oxygen to process than does CHO and thus may be important in prolonging the slow component of EPOC (Børsheim and Bahr, 2003).

Check your understanding of EPOC by completing the [Check Your Comprehension—Case Study 1](#) in the box below.

CHECK YOUR COMPREHENSION—Case study 1

You are working as a fitness trainer at a wellness center. Yeen Kuen is a new client who wants to work out 3 days a week combining aerobic and resistance exercise. Yeen Kuen does not need to lose weight but wants to tone muscles and improve endurance. Using the data presented below from a study by Drummond et al. (2005), make a recommendation as to whether this client should do the resistance workout first or the aerobic workout first. Explain your reasoning.

Check your answer in [Appendix C](#). $\dot{V}O_2$

Physiological Responses to Sequential Activity

| Sequence | Activity | HR (b·min ⁻¹) during | VO ₂ (mL· kg ⁻¹ ·min ⁻¹) during | VO ₂ (mL· kg ⁻¹ ·min ⁻¹) EPOC (10 min) |
|----------|-----------------------|--|---|--|
| RE-RU | Treadmill (25 min) | 172 ± 4.0* | 40.0 ± 1.3* | 5.14 ± 0.2* |
| | 3 sets/10 reps | 140 ± 9.0 | | |
| RU-RE | Treadmill (25 min) | 161 ± 4.0 | 38.0 ± 1.3 | 5.7 ± 0.1 |
| | 3 sets/10 reps | 144 ± 5.0 | | |

RE, resistance exercise, 7 lifts at 70% 1-RM; RU, treadmill running at 70% $\dot{V}O_{2\max}$; * $P < 0.05$ compared to RU-RE.

ATP-PC Changes

Figure 3.10 indicates what happens to ATP and PC levels in muscle during constant-load, supramaximal exercise (105–110% $\dot{V}O_{2\max}$) lasting 3 minutes or less. As shown in the figure, the ATP level in the muscle decreases only slightly. In fact, the maximum ATP depletion observed in skeletal muscle after heavy exercise is only about 30–40% in both males and females. Thus, even after exhaustive work, 60–70% of the resting amount of ATP is still present (Cheetham et al., 1986; Gollnick and King, 1969).

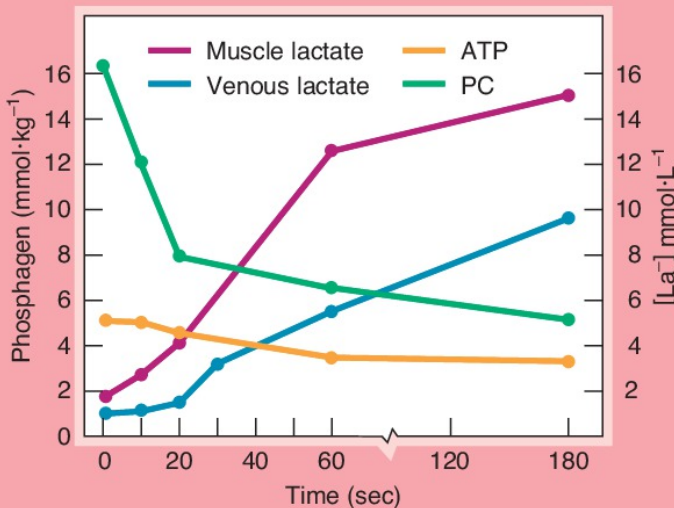




Figure 3.10 Time Course for the Utilization of PC and ATP and the Accumulation of Lactate in Muscle and Veins.

Muscle levels of ATP are maintained relatively constant during high-intensity, short-duration exercise at the expense of PC. Muscle lactate levels rise sooner and higher than do venous levels owing to the diffusion time lag and dilution.

Source: Modified with permission from Gollnick, P. D., & L. Hermansen: Biochemical adaptations to exercise: Anaerobic metabolism. In Wilmore, J. H. (ed.): *Exercise and Sport Sciences Reviews*. New York, NY: Academic Press (1973).

Conversely, the level of PC changes dramatically. The greatest depletion of PC occurs in the initial 20 seconds of exercise, with the result that ATP is maintained almost at resting levels during that time span. From 20 to 180 seconds, the decline in PC and ATP is both gradual and parallel (Gollnick and Hermansen, 1973). Obviously, the ATP level is maintained at the expense of the PC. However, some ATP is also being provided from glycolysis, as indicated by the rise in lactate in both muscle and blood, and an additional amount by oxidation.

Lactate Changes

Lactate concentrations in response to exercise depend primarily on the intensity of the exercise. Acute exercise produces an increase in both MCT1 and MCT4 with the change being greater in MCT1 than MCT4 (Thomas et al., 2012). In addition, transmembrane lactate and hydrogen ion gradients increase. Lactate transport is faster in oxidative fibers than in glycolytic ones. The fast transport of lactate by oxidative fibers may reflect lactate's role as an energy substrate, while the slower transport in glycolytic fibers may contribute to a greater retention of lactate

during recovery for reconversion into glycogen (Gladden, 2000).

Short-Term, High-Intensity Anaerobic Exercise

Figure 3.10 includes both muscle and blood lactate responses to high-intensity, short-duration (3 minutes or less), supramaximal exercise. As the figure shows, muscle lactate levels rise immediately with the onset of such hard work (105–110% $\dot{V}O_{2\max}$) and continue to rise throughout the length of the task. Blood lactate values show a similar pattern, if the lag for diffusion time is taken into account. This lactate response (a rapid and consistent accumulation) is representative of what occurs when the exercise bout is greater than 90% $\dot{V}O_{2\max}$ (Gollnick and Hermansen, 1973).

Short- and Long-Term, Light to Moderate Submaximal Aerobic Exercise

Figure 3.11 depicts what occurs in both short-term and long-term low- to moderate-intensity submaximal aerobic activity. During the first 3–5 minutes of such steadystate work, the lactate (La^-) concentration rises (**Figure 3.11A**). This increase reflects the lactate accumulated during the oxygen deficit (Gollnick and Hermansen, 1973). Between approximately 5 and 10 minutes, lactate decreases from this elevated point to a stable near resting level and remains there for 30–60 minutes (**Figure 3.11B**). The reason for this result lies in the balance between lactate production (the rate of lactate appearance) and lactate clearance (the rate of lactate disappearance).

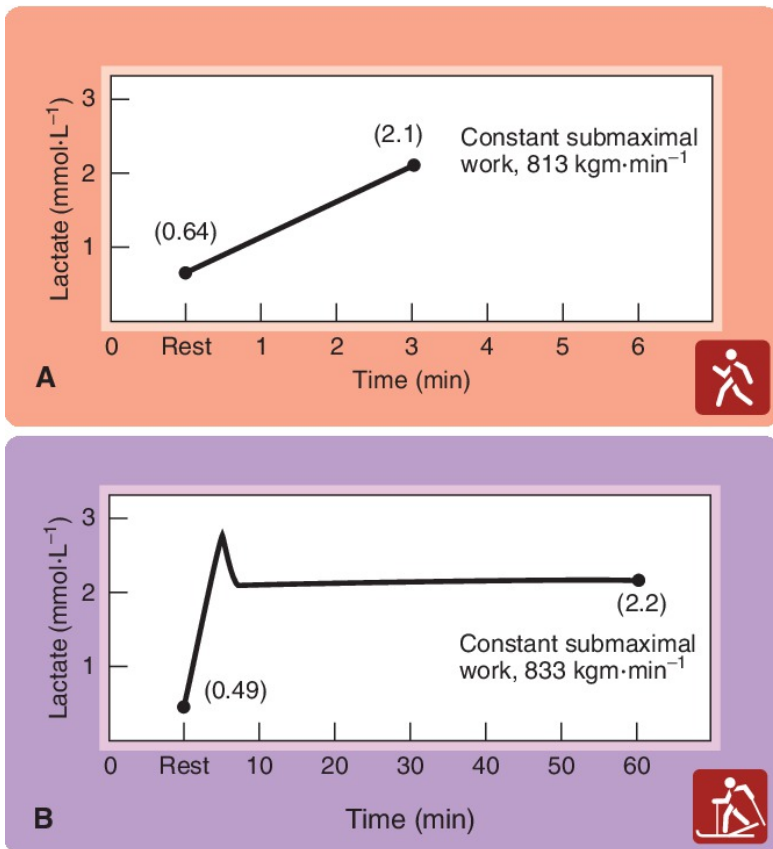


Figure 3.11 Lactate Accumulation during Short- and Long-Term Dynamic Aerobic Constant Submaximal Work.

A. During a short bout of constant submaximal work, lactate accumulation increases but is minimal. **B.** If this submaximal load continues for a long duration, the initial rise in lactate accumulation declines slightly, levels off and then remains stable.

Figure 3.12 shows the results from a study that directly measured the rate of lactate appearance (R_a) and the rate of lactate disappearance (R_d) by radioactive tracers as well as the blood concentrations of lactate ($[\text{La}^-]$) (Brooks, 1985). In the transition from rest to steady-state submaximal exercise, the rate

of both lactate appearance and lactate disappearance increased. During the next 30 minutes of exercise, the turnover rate was higher than at rest. The result was an initial increase in $[La^-]$, which declined after 5 minutes of activity to almost resting levels by 30 minutes. When the workload was increased, the rate of clearance (R_d) was no longer able to keep up with the rate of production (R_a), and the lactate concentration in the blood $[La^-]$ increased sharply.

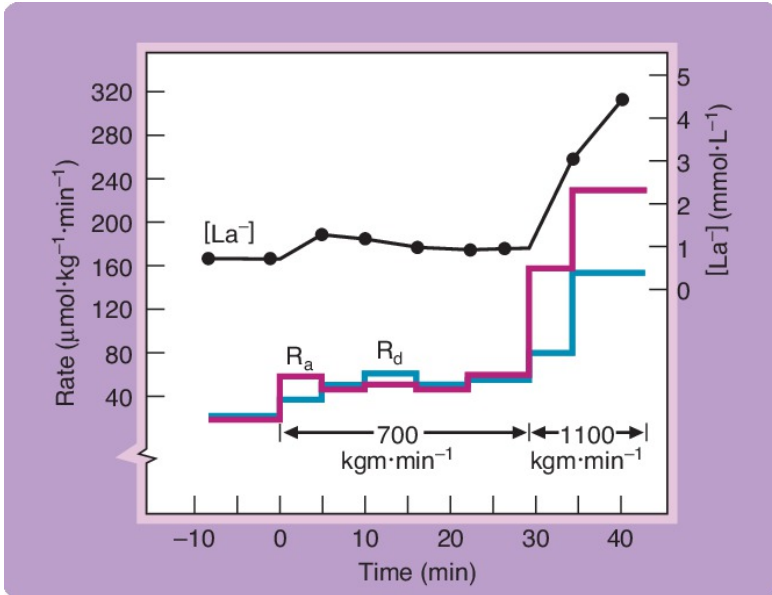


Figure 3.12 The Impact of the Rates of Lactate Appearance and Disappearance on Lactate Accumulation during Light and Heavy Submaximal Aerobic Exercise.

During light ($700 \text{ kgm} \cdot \text{min}^{-1}$) submaximal aerobic exercise, the rate of lactate disappearance (R_d) (left y-axis) lags behind the rate of lactate appearance (R_a) (left y-axis) to a

small extent, but then catches up so that the 30-minute lactate concentration $[La-]$ value (right y-axis) approximates rest. During heavy submaximal exercise (30–45 minutes), the Rd lags behind Ra and $[La-]$ increases.

Source: Reprinted with permission from Brooks, G. A.: Anaerobic threshold: Review of the concept and directions for future research. *Medicine and Science in Sports and Exercise*. 17(1):22–31 (1985). Copyright ©1985 The American College of Sports Medicine.

Figure 3.13 shows how lactate concentration varies with competitive distance (and thus duration) in highly trained male runners. At the shorter distances, the predominant energy source is anaerobic, and the anticipated high lactate values occur. As the distance increases and more energy is supplied aerobically, the intensity that can be maintained decreases. As a result, lactate also decreases, doing so in a negative exponential curvilinear pattern. By approximately 30 km (18 mi), the lactate concentrations are almost the same as at rest.

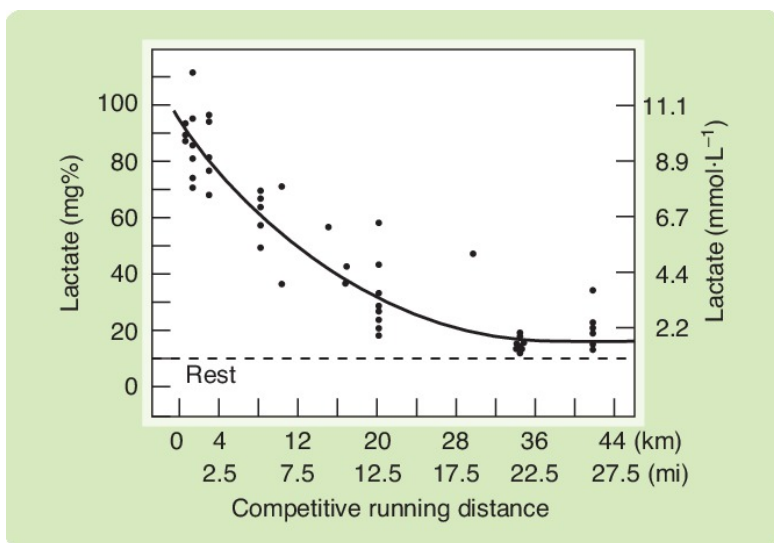


Figure 3.13 Lactate Accumulation Resulting from Increasing Distances of Competitive Running Races.

Blood lactate accumulation shows an inverse curvilinear relationship with distance in running races. **Source:** Modified with permission from Costill, D. L.: Metabolic responses during distance running. *Journal of Applied Physiology*. 28(3):251–255 (1970). Copyright © 1970 The American Physiological Society. All rights reserved.

Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

The importance of the intensity of exercise, even at the marathon distance, is illustrated in **Figure 3.14**. Two groups of runners were matched according to their $\dot{V}O_{2\max}$. One group ran a simulated marathon on the treadmill at 73.3% $\dot{V}O_{2\max}$ (in 2 hours 45 minutes or less); the other group ran the same distance but at 64.5% $\dot{V}O_{2\max}$ (in 3 hours 45 minutes or slightly less). Within the slow group, blood lactate values remained relatively stable and at a level considered to be within normal resting amounts. The blood lactate levels in the fast group were statistically significantly higher throughout the marathon than were those of the slow group. As absolutes, however, both sets of values were low, with the slow group being within a normal resting range and the fast group only slightly above normal resting levels (O'Brien et al., 1993).

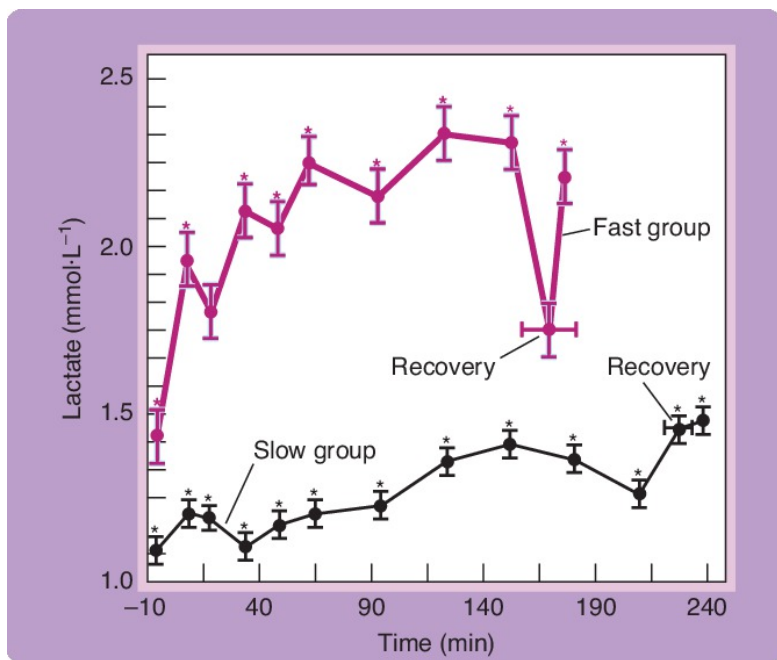


Figure 3.14 Blood Lactate Accumulation during the Marathon.

Blood lactate accumulation during a fast marathon (2 hours and 45 minutes or less) was greater than the accumulation during a slow marathon (3 hours and 45 minutes or less).

The lactate levels for the slow marathoners never exceeded normal resting levels, and the lactate levels for the fast marathoners barely exceeded normal resting levels. **Source:**

Reprinted with permission from O'Brien, M. J., C. A. Viguie, R. S. Mazzeo, & G. A. Brooks: Carbohydrate dependence during marathon running. *Medicine and Science in Sports and Exercise*. 25(9):1009–1017 (1993). Copyright ©1993 The American College of Sports.

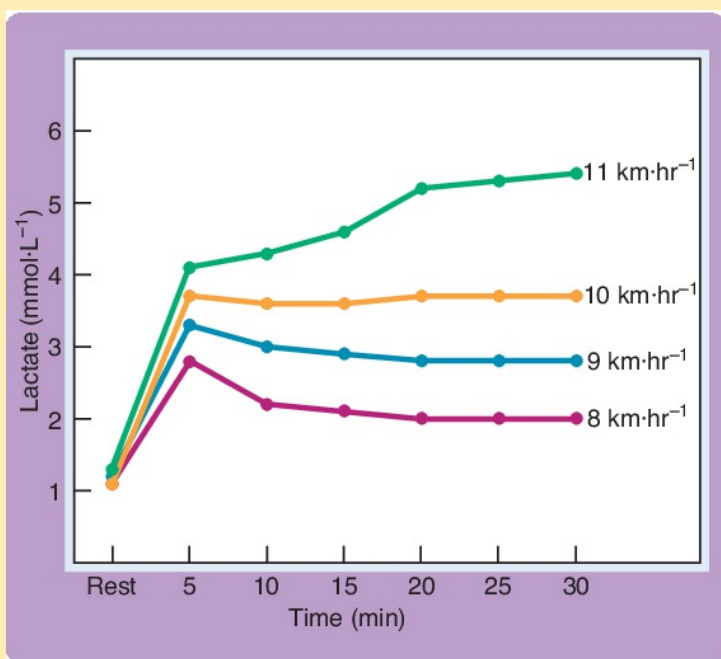
At moderate- to heavy-intensity continuous activity between 50 and 85% $\dot{V}O_{2\max}$ (depending on the individual's genetic characteristics and training status), lactate levels rise rapidly during the first 5–10 minutes of exercise. If the workload continues for more than 10 minutes, the lactate level may continue to rise, may stabilize, or may decline, depending on the individual and other conditions.

One of these “other conditions” may be the exercise intensity in relation to the individual's maximal lactate steady state. **Maximal lactate steady state (MLSS)** is the highest workload that can be maintained over time without a continual rise in blood lactate; it indicates an exercise intensity above which lactate production exceeds clearance. MLSS is assessed by a series of workloads performed on different days. Each succeeding workload gets progressively harder until the blood lactate accumulation increases more or less steadily throughout the test or increases more than 1 mmol·L⁻¹ after the initial rise and establishment of a plateau in the early minutes. Thus, in a 30-minute test, changes in the first 10 minutes are ignored and only the last 20 minutes are used to determine if the change is less than or greater than 1 mmol·L⁻¹. When the blood lactate concentration meets this criterion, the previous workload that exhibited a plateau in lactate throughout the duration (after the initial rise) is labeled as the MLSS workload. Often the MLSS workload is compared to the individual's maximal workload and expressed as a percentage known as MLSS intensity. MLSS values between 1.9 and 7.5 mmol·L⁻¹, which corresponded to exercise intensities between 54 and 83% of maximal aerobic power, have been reported. MLSS is highly related to $\dot{V}O_{2\max}$ and endurance performance (Beneke et al., 2011). Performances at the MLSS intensity result in a steady state for lactate for approximately 30–60 minutes; performances below this intensity show declining lactate values, and performances above this level exhibit progressively increasing lactate values. Extensive endurance performance cannot be completed above the MLSS, but portions of the event certainly may be (Beneke et al., 2000; Billat et al., 2003). Complete the [Check Your Comprehension 2—Case Study 2](#) to practice your understanding of MLSS. Check your answer in [Appendix C](#).

Maximal Lactate Steady State (MLSS) The highest workload that can be maintained over time without a continual rise in blood lactate; it indicates an exercise intensity above which lactate production exceeds clearance.

CHECK YOUR COMPREHENSION—CASE STUDY 2

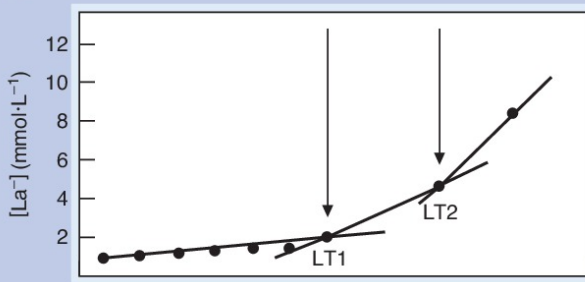
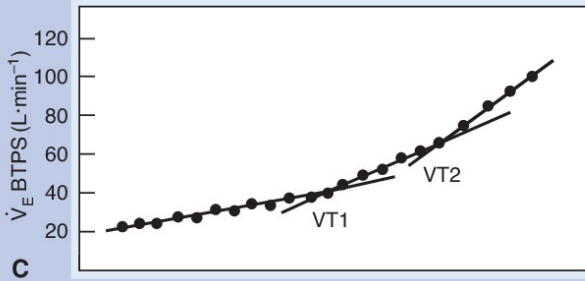
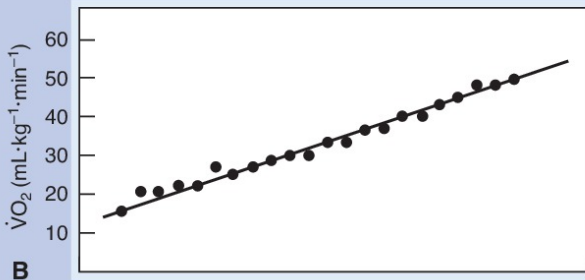
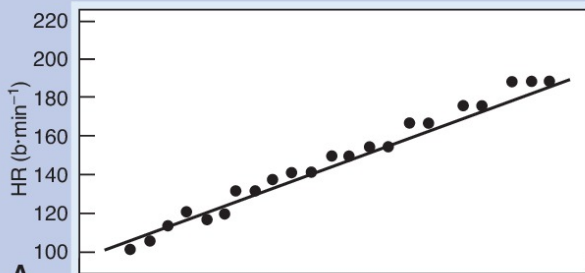
The accompanying graph shows the test results for Miriam a 23-year-old runner. Miriam has entered the Huskie 15-km run. Which speed indicates her maximal lactate steady state? Based on these results, what is a reasonable time goal for Miriam for this 15-km run? Check your answer in [Appendix C](#).



Incremental Aerobic Exercise to Maximum

Figure 3.15 depicts physiological responses during incremental exercise to maximum. Heart rate and oxygen consumption

(**Figure 3.15A and B**) increase in a rectilinear pattern to meet the increasing demands for energy, but both ventilation and blood lactate (**Figure 3.15C and D**) show very little initial change and then increase more sharply. As depicted, this pattern is often described as a rectilinear rise with two breakpoints or thresholds. Alternately, by smoothing these points, the pattern can be described as positively accelerating exponential curves.



D Incremental work expressed as workload or $\% \dot{V}\text{O}_2 \text{max}$



Figure 3.15 Ventilatory and Lactate Thresholds during Incremental Work to Maximum.

Both heart rate (A) and O₂ consumption (B) increase in direct rectilinear patterns during an incremental work task whether the work is expressed in terms of absolute workload or percentage of $\dot{V}O_{2\max}$. In contrast, both ventilation (\dot{V}_E) (C) and lactate (D) appear to exhibit two distinct breakpoints as they rise. The circumstance of VT1 occurring at the same time as LT1 and VT2 occurring at the same time as LT2 is coincidental.

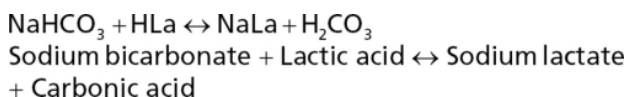
Since the early 1970s, much attention has been paid to a concept that has been labeled variously as the *anaerobic threshold(s)*, the *ventilatory threshold(s)*, or the **lactate threshold(s)**. The original concept of an anaerobic threshold is based on the lactate response to incremental exercise, as depicted in **Figure 3.15D**, and the relationship of the lactate response to minute ventilation (E), the volume of air breathed each minute, **Figure 3.15C**).

Lactate Thresholds Points on the linear-curvilinear continuum of lactate accumulation that appear to indicate sharp rises, often labeled as the first (LT1) and second (LT2) lactate threshold.

As espoused by [Wasserman et al. \(1973\)](#), the *anaerobic threshold* is defined as the exercise intensity, usually described as a percentage of $\dot{V}O_{2\max}$ or absolute workload, above which blood lactate concentrations rise and minute ventilation increases disproportionately in relation to oxygen consumption. The onset

of anaerobic metabolism (or anaerobiosis), which is assumed to lead to the lactate accumulation, is attributed to the failure of the cardiovascular system to supply the oxygen required to the muscle tissue. The disproportionate rise in ventilation is attributed to excess carbon dioxide resulting from the buffering of the lactic acid (Jones and Ehrsam, 1982; Wasserman and McIlroy, 1964).

Theoretically, these interactions can occur as follows: lactic acid is a strong acid, and as noted earlier in this chapter, it readily dissociates into hydrogen ions (H^+) and lactate. Because excess hydrogen ions would change the pH (or acid-base balance) of the muscles and blood, the body attempts to bind these hydrogen ions to a chemical buffer. For example, sodium bicarbonate (a weak base) may be used as a buffer in the reaction:



Carbonic acid is a much weaker acid than lactic acid and can be further dissociated into water and carbon dioxide:



Carbon dioxide is a potent stimulant for respiration and can easily be removed from the body through respiration, thereby assisting in the maintenance of pH (Pitts, 1974). The carbon dioxide thus formed is said to be *nonmetabolic carbon dioxide*, since it does not result from the immediate breakdown of an energy substrate (carbohydrate, fat, or protein).

Figure 3.15 clearly shows distinct breaks from linearity in respiration (**Figure 3.15C**) despite a continuous rectilinear rise in heart rate (**Figure 3.15A**) and oxygen consumption (**Figure 3.15B**) during incremental work to maximum. These breakpoints (**Figure 3.15C**) have been labeled VT1 (the first ventilatory threshold) and VT2 (the second ventilatory threshold). They were originally thought to result from corresponding lactate thresholds (labeled in **Figure 3.15D** as LT1 and LT2 for the first and second lactate threshold) as a result of the buffering of lactic acid. The

original work by Wasserman and others postulated only one anaerobic threshold (which would have been VT1 in **Figure 3.15C**), but later work identified at least two thresholds, which were given various names (Carey et al., 2010; Jacobs, 1986; Reinhard et al., 1979; Skinner and McLellan, 1980). The designations VT1 and VT2 and LT1 and LT2 are used in this text for simplicity and because no causal mechanism is implied.

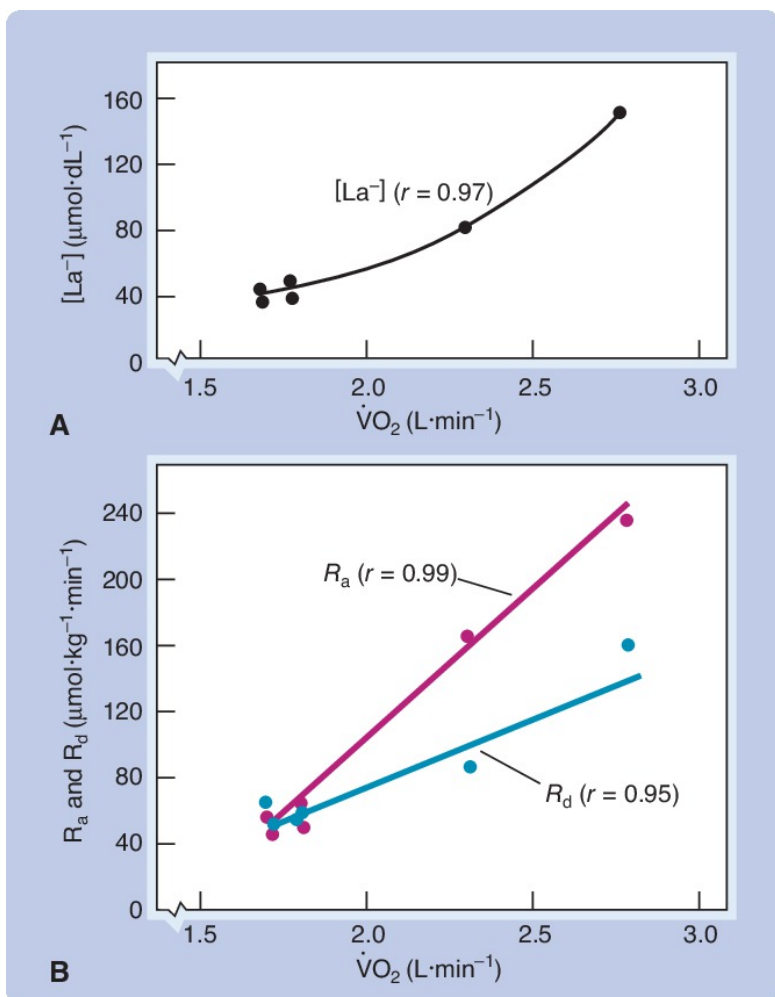




Figure 3.16 The Rate of Appearance (R_a), the Rate of Disappearance (R_d), and the Resultant Accumulation of Lactate as a Consequence of Incremental Exercise.

The measured change in lactate concentration [$La -$] (A) is a result of a growing imbalance between the rate of appearance (R_a) and the rate of disappearance (R_d) (B) as exercise intensity increases. **Source:** Reprinted with permission from Brooks, G. A.: Anaerobic threshold: Review of the concept and directions for future research. *Medicine and Science in Sports and Exercise*. 17(1):22–31 (1985). Copyright ©1985 The American College of Sports Medicine.

The idea that the point of lactate accumulation can be determined noninvasively by respiratory values typically measured during laboratory exercise testing of oxygen consumption is appealing, since few people enjoy having a catheter inserted or multiple blood samples taken. However, the terminology, determination, and mechanistic explanations are not without controversy (Walsh and Banister, 1988). Four concerns have been raised, as described below.

The primary concern is that the presence of lactate does not automatically mean that the oxygen supply is inadequate (Brooks, 2018, 2020; Hughes et al., 1982). This fact was discussed in detail earlier in this chapter. Lactate accumulation occurs not at the time of increased production but when the turnover rate (or balance between production and removal) cannot keep up and appearance exceeds clearance.

Figure 3.16A shows this concept graphically for incremental exercise. That is, as the exercise and oxygen consumption increase, the rate of lactate appearance (R_a) in the muscle does also (**Figure 3.16B**). At low-intensity exercise, the rate of lactate disappearance (R_d) does not differ much from R_a . However, as the intensity increases, the gap between R_a and R_d grows

progressively wider. The result is the blood lactate concentration $[La-]$ depicted in **Figure 3.16A**. Thus, it is incorrect to consider the appearance of elevated concentrations of lactate in the blood an anaerobic threshold. Rather it is a lactate threshold that represents the point at which the metabolic clearance of lactate becomes limited (Messonnier et al., 2013).

A second concern is exactly how to interpret the lactate response to incremental work. Look closely at the lactate pattern in **Figure 3.15** and recall that this pattern can be described as either a rectilinear rise with two breakpoints, or thresholds (Skinner and McLellan, 1980), or as an exponential curve (Hughson et al., 1987). Experimental evidence and mathematical models (Hughson et al., 1987) support the curvilinear interpretation. Nevertheless, the term *lactate threshold* continues to be used to indicate marked increases in the accumulation of lactate. The ventilatory thresholds are always considered to be true breakpoints.

A third concern involves carbon dioxide. Carbon dioxide is involved in the control of respiration (see **Chapter 9**). An excess of hydrogen ions from a source such as lactic acid can cause an increased amount of carbon dioxide through the bicarbonate buffering system described earlier. However, the presence of lactic acid is not the only mechanism that can account for the increased carbon dioxide or the concomitant increase in minute ventilation (Inbar and Bar-Or, 1986). Evidence is particularly strong in McArdle's syndrome patients. McArdle's syndrome patients are deficient in the enzyme glycogen phosphorylase, which is necessary for breaking down glycogen, which can ultimately be converted to lactic acid. Thus, no matter how hard these patients exercise, their lactic acid values remain negligible. On the other hand, their minute ventilation values have the same distinctive breakpoints shown by anyone not deficient in glycogen phosphorylase (Hagberg et al., 1982). Evidence by Péronnet and Aguilaniu (2006) has shown that the decrease in bicarbonate concentration is not the mirror image of the increase in lactate concentration. These researchers believe that the disproportionate increase in $\dot{V}O_2$ is due to hyperventilation and low plasma pH—not that the hyperventilation is due to the increased CO_2 .

A fourth concern is that the lactate thresholds and the ventilatory thresholds do not change to the same extent in the same individuals as a result of training, glycogen depletion, caffeine ingestion, and/or varying pedaling rates (Hughes et al., 1982; Poole and Gaesser, 1985). If they were causally linked, they should change together.

FOCUS ON RESEARCH

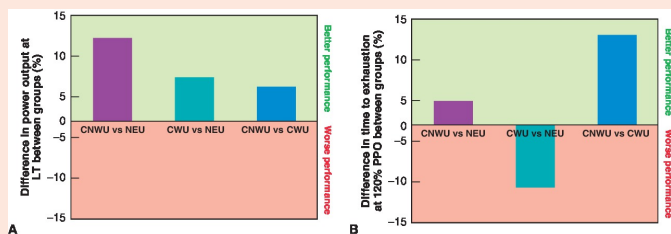
The Effect of Cold Ambient Temperature on Lactate Kinetics

Seven trained female cyclists and triathletes completed baseline, lactate threshold (LT), and time to exhaustion at 120% peak power output (PPO) tests to familiarize the participants with the protocol. The following visits consisted of participation in three LT and PPO tests in different environmental conditions: thermoneutral (20°C: NEU), cold with no warm up (0°C: CNWU), and cold with an active warm up (0°C: CWU). The active warm up consisted of 5 minutes of cycling at 1.5 watts·kg⁻¹ for 5 minutes followed by 20 minutes of cycling at 2 watts·kg⁻¹ for 20 minutes—these 20 minutes included a 10-second sprint occurring at minutes 15, 17, 19 with a resistance set to 5.0 watt·kg⁻¹. All experimental visits were completed in a randomized order and occurred during the follicular phase of the menstrual cycle.

The overall power output at LT was 10.2% greater with CNWU than NEU. Similarly, the power output at LT during the CNWU condition was 4.2% greater compared to CWU condition. This 10.2% increase in power output from NEU to CNWU indicates a small potential performance enhancement (Panel A).

There was no difference in time to exhaustion at 120% of PPO between the CNWU and NEU conditions. Time to exhaustion was, however, 11% longer in the CNWU than the CWU condition. These results suggest that exercising in the

cold without prior warm-up slightly enhance performance (Panel B).



Percent differences between CNWU, NEU, and CWU.

A. Peak power output (PPO) at lactate threshold (LT). **B.** Time to exhaustion test at 120% of PPO. Values above 0 indicate better performance and values below 0 indicate worse performance in each comparison. LT, lactate threshold; PPO, peak power output; CNWU, cold no warm-up; CWU, cold with active warm-up; NEU, thermoneutral. Data are presented as means.

Source: Morrissey, M. C., J. N. Kisiolek, T. J. Ragland, B. D. Willingham, R. L. Hunt, R. C. Hickner, & M. J. Ormsbee: The effect of cold ambient temperature and preceding active warm-up on lactate kinetics in female cyclists and triathletes. *Applied Physiology Nutrition & Metabolism*. 44(10): 1043–1051 (2019).

Current theory, therefore, concludes that although lactate increases and ventilatory breaks or thresholds often occur simultaneously, these responses are due to coincidence, not cause and effect (Brooks, 1985; Walsh and Banister, 1988).

Despite a lack of complete understanding of the lactate threshold, the amount of work an athlete can do before accumulating large amounts of lactate has a definite bearing on performance. Distance running performance, for example, depends to a large extent on some combination of $\dot{V}O_{2\max}$, the oxygen cost of running at a given submaximal speed (called economy), and the ability to run at a high percentage of

$\dot{V}O_{2\max}$ without a large accumulation of lactate (Costill et al., 1973; Farrell et al., 1979; Kinderman et al., 1979). Recently, it was reported that the 2-hour marathon attempt requires a sustained oxygen cost equivalent to 94% of O_{2peak} and a lactate threshold corresponding to 83% of O_{2peak} (Jones et al., 2021). An indication of the running speed that represents the optimal percentage of $\dot{V}O_{2\max}$ can be achieved by determining the lactate thresholds. The first lactate threshold (LT1) generally occurs between 40 and 60% of $\dot{V}O_{2\max}$; the second lactate threshold (LT2) is generally over 80% of $\dot{V}O_{2\max}$ and possibly as high as 95% of $\dot{V}O_{2\max}$. LT1 is sometimes equated with a lactate concentration of 2 mmol·L⁻¹, and LT2 with a lactate concentration of 4 mmol·L⁻¹, but these values may or may not coincide for any given individual. This 4 mmol·L⁻¹ level, also called the *onset of blood lactate accumulation (OBLA)*, is frequently used to decide both training loads and racing strategies (Hermansen et al., 1975; Jacobs, 1986).

The relationship between the various lactate thresholds determined during an incremental exercise test and the maximal lactate steady state (MLSS) described earlier is variable and largely based on testing modality, protocol, and training status of the participants. Although sometimes not statistically different, typically MLSS is slightly lower than LT2 or OBLA. The techniques should not be used interchangeably to determine training prescriptions (Beneke, 1995; de Souza et al., 2012; Klusiewicz, 2005; Svedahl and MacIntosh, 2003).

Dynamic Resistance Exercise

Lactate responses to dynamic resistance exercise vary greatly because of the many different possible combinations of exercises, repetitions, sets, and rest periods. In addition, samples are generally not taken repeatedly during the workout; instead, the most frequently reported values are only postexercise. In general, postexercise lactate values have been shown to range from approximately 4–21 mmol·L⁻¹. The higher values result from high-volume, moderate-load, short rest period sequences and circuit-type exercise bouts (Bangsbo et al., 1994; Burleson et al.,

1998; Keul et al., 1978; Reynolds et al., 1997; Tesch, 1992). Concentric contractions elicit higher lactate responses than do eccentric contractions (Durand et al., 2003). Initially, blood lactate concentrations increase rapidly with constant resistance exercise, after which (around 10–15 minutes) a steady state is reached, thus demonstrating a typical response of lactate in resistance training (Garnacho-Castaño et al., 2015). Resistance programs designed to increase strength primarily through hypertrophy (increased muscle size) produce greater lactate responses than do those designed to increase strength primarily through neural adaptation or dynamic power programs (Crewther et al., 2006).

Table 3.3 summarizes the anaerobic metabolic exercise responses discussed in this section.

| TABLE 3.3 Lactic Anaerobic Exercise Response | | | | | |
|--|--|---|--|---|--|
| Short-Term, Light to Moderate, Submaximal Aerobic Exercise | Short-Term, Moderate to Heavy, Submaximal Aerobic Exercise | Long-Term, Moderate to Heavy, Submaximal Aerobic Exercise | Short-Term, High-Intensity, Anaerobic Exercise | Incremental Exercise to Maximum | Dynamic Resistance Exercise |
| ≤2 mmol·L ⁻¹ | ~4–6 mmol·L ⁻¹ | Depends on relationship to MLSS | Large increase; interval may go to 32 mmol·L ⁻¹ | Positive exponential curve; LT1 ~2 mmol·L ⁻¹ ; LT2 ~4 mmol·L ⁻¹ ; max ≥8 mmol·L ⁻¹ | 4–21 mmol·L ⁻¹ ; greatest with high volume and circuit type |

Benefits of Lactate

While lactic acid has long been deemed a waste product and “bad guy” with multiple deleterious effects, lactate does, in fact, have quite a few qualities that make it a “good guy.” Overall, given that it is now known that lactate is produced even when oxygen is not lacking, it can be seen as a link between glycolytic and aerobic pathways. More specifically, lactate shuttling between producer and consumer cells functions a) to coordinate whole-body metabolism as an energy source; b) as the major gluconeogenic precursor; and c) as a signaling molecule. Although not an exhaustive list, examples of these functions are described below.

Lactate as an Energy Source

Challenges to the ATP supply triggers the production of lactate leading to increased ATP production and homeostasis. As described earlier in [Chapter 3](#), shifting of the hydrogen atoms from $\text{NADH} + \text{H}^+$ to pyruvate to form lactate serves to maintain the redox potential of the cell. There is a finite amount of NAD^+ available in the cytoplasm to accept hydrogen, thus when pyruvate accepts H^+ from $\text{NADH} + \text{H}^+$ to form lactate, it allows fast glycolysis to continue to provide ATP.

Glucose flux may increase 2–3 times in humans during the transition from rest to exercise; lactate flux is far more expansive rising from a rate of 50% of that of glucose at rest to a value 4 times that of glucose during exercise. As indicated previously, the vast majority of this lactate (50% at rest and 75–80% or more during exercise) is disposed of via oxidation providing ATP. Thus, in terms of fueling active tissues during exercise lactate plays a major role. In addition to muscle tissue, lactate itself, when circulated throughout the body, can be utilized as fuel in both the heart and potentially the brain. In the heart, lactate is preferred over glucose as a fuel. In resting individuals, glucose is the preferred fuel for the brain. During exercise, net lactate uptake can provide 25% of the brain's energy need. When used in the brain, lactate serves as a glucose-sparing substrate ([Brooks, 2018](#); [Ferguson et al., 2018](#)).

Lactate as the Major Gluconeogenic Precursor

Lactate is reconverted into glucose in the liver by gluconeogenesis (Cori cycle). It can also be converted to glycogen by glycogenesis in liver or resting glycolytic skeletal muscle. In so doing, it may spare the breakdown of glucose and help maintain blood glucose concentrations. ([Brooks, 2018](#); [Ferguson et al., 2018](#))

Lactate as a Signaling Molecule

Lactate is known to act as a signaling molecule with autocrine, paracrine, and endocrine-like effects and has been called a “lactormone.” The rate-limiting enzyme PFK is up-regulated by lactate in hypoxia and down-regulated in normoxia. In the short-term, lactate both inhibits lipolysis (the breakdown of fat) and

mitochondrial FFA oxidation. In the long-term, lactate up-regulates mitochondrial biogenesis (the growth of mitochondria), glucose tolerance, and lipid oxidation. Lactate release during heavy exercise has a role in fluid volume regulation. Lactate accumulation in physical exercise has the potential to affect gene regulation in diverse cells ([Brooks, 2020](#); [Ferguson et al., 2018](#)).

Why Is Lactate Accumulation a Problem?

Despite the known benefits of lactate, there are some detrimental effects of lactate. It is the hydrogen ions (H^+) that dissociate from lactic acid, rather than lactate (La^-) that present the primary (but not only) problems to the body. This distinction is important, because at normal pH levels, 99% of the lactic acid is dissociated immediately to H^+ and La^- ($C_3H_5O_3^-$) ([Brooks, 1985](#); [Gladden, 2004](#)). As long as the amount of free H^+ does not exceed the ability of the chemical and physiological mechanisms to buffer them and maintain the pH at a relatively stable level, there are few problems. Most problems arise when the amount H^+ exceeds the body's immediate buffering capacity and the pH decreases ([Gladden, 2004](#)). The blood has become more acidic. At that point, pain is perceived and performance suffers. The mechanisms of these results are as follows.

Pain

Anyone who has raced or run the 400-m distance all out understands the pain associated with H^+ production. The 400-m run takes between approximately 45 seconds and 3 minutes (depending on the ability of the runner) and relies heavily on the ATP-PC and LA systems to supply the needed energy. The resultant hydrogen ions accumulate and stimulate pain nerve endings in the muscle ([Hall and Hall, 2021](#)).

Performance Decrement

The decrement in performance associated with lactic acid results from fatigue that is metabolic and possibly muscular in origin.

METABOLIC FATIGUE Metabolic fatigue results from a reduced

production of ATP linked to enzyme changes, changes in membrane transport mechanisms, and changes in substrate availability.

Enzymes—in particular, the rate-limiting enzymes in the metabolic pathways—can be inactivated by high hydrogen ion concentrations (low pH). The hydrogen ion attaches to these enzyme molecules and in so doing changes their size and shape and thus their ability to function. Phosphofructokinase (PFK)—the rate limiting enzyme of glycolysis—is thought to be particularly sensitive, although oxidative enzymes can also be affected (Hultman and Sahlin, 1980).

At the same time, changes occur in membrane transport mechanisms (either to the carriers in the membrane or to the permeability channels). These changes affect the movement of molecules across the cell membrane and between the cytoplasm and organelles such as the mitochondria (Hultman and Sahlin, 1980).

Energy substrate availability can be inhibited by a high concentration of hydrogen ions. Glycogen breakdown is slowed by the inactivation of the enzyme glycogen phosphorylase. Fatty acid utilization is decreased because lactic acid inhibits mobilization. Thus, a doublejeopardy situation occurs. With fatty acid availability low, a greater reliance is placed on carbohydrate sources at the time when glycogen breakdown is inhibited. At the same time, phosphocreatine breakdown is accelerated, leading to a faster depletion of substrate for ATP regeneration (Åstrand et al., 2003; Davis, 1985; Hultman and Sahlin, 1980; Rooney and Trayhurn, 2011).

Thus, both the inactivation of enzymes and the decrement in substrate availability will lead to a reduction in the production of ATP and, ultimately, a decrement in performance.

MUSCULAR FATIGUE Muscular fatigue is evidenced by reduced force and velocity of muscle contraction. The contraction of skeletal muscle and the influence of lactic acid on muscular fatigue are detailed in Chapters 17 and 18, respectively. Suffice it to say here that the idea that lactic acidosis directly and only negatively affects the contractile elements of muscle and force/velocity production has been largely abandoned (Meyer and

Wiseman, 2012). Recent studies question the role of lactic acid in muscular fatigue, even to the point of considering changes brought about by lactate and acidosis during exercise to be beneficial. That is, it is unclear whether lactate is a causative or a preventive agent of muscle fatigue (Bangsbo, 2006; Gladden, 2008b; Lamb, 2006; Lindinger, 2011; Robergs et al., 2004). While the controversy remains, what is certain is that although lactate/ H^+ may contribute to fatigue, lactic acid is not the sole cause of muscular fatigue (Bangsbo, 2006; Cairns, 2006; Lamb, 2006). For example, phosphate ions (P_i) also contribute to muscular fatigue. These P_i are produced during the breakdown of ATP and CP when these two molecules break down rapidly and P_i accumulates. The P_i accumulation may directly inhibit muscular contraction (more on this in Chapter 17) (Mougios, 2006).

Time Frame for Lactate Removal Postexercise

Lactate is removed from the bloodstream relatively rapidly following exercise (Gollnick et al., 1986). However, removal does not occur at a constant rate. If it did, then higher levels of lactate would take proportionately longer to dissipate than would lower levels. By analogy, if you do one push-up in 2 seconds and you couldn't change that rate or speed, then 10 push-ups would take twice as long to do as 5 push-ups (20 vs. 10 seconds). On the other hand, if you could change the rate, you might do 10 in the same time as you did 5 (in 10 seconds). Many chemical reactions have this ability to change the rate or speed at which they occur. The rate is proportional to the amount of substrate and product present. The more substrate available and the less product, the faster the reaction proceeds, and vice versa. This characteristic is called the *mass action effect*. Lactate appears to be one of those substrates whose utilization and conversion are linked with the amount of substrate present (Bonen et al., 1979).

Thus, despite wide differences among individuals (which may be related to muscle fiber type), in a resting recovery situation, approximately half of the lactate is removed in about 15–25 minutes regardless of the starting level. This time is called the *half-life of lactate*. Near-resting levels are achieved in about 30–60

minutes, regardless of the starting level. Thus, the initial postexercise concentration of lactate is the first factor that influences the rate of removal. The higher the concentration, the faster is the rate of removal (Bonen et al., 1979; Hermansen and Stensvold, 1972; Hogan et al., 1995).

Figure 3.17 shows typical resting recovery curves from cycling and running studies. Note that in each case, the value close to 50% (shown in parentheses) of the initial postexercise lactate levels occurs between 15 and 25 minutes of recovery.

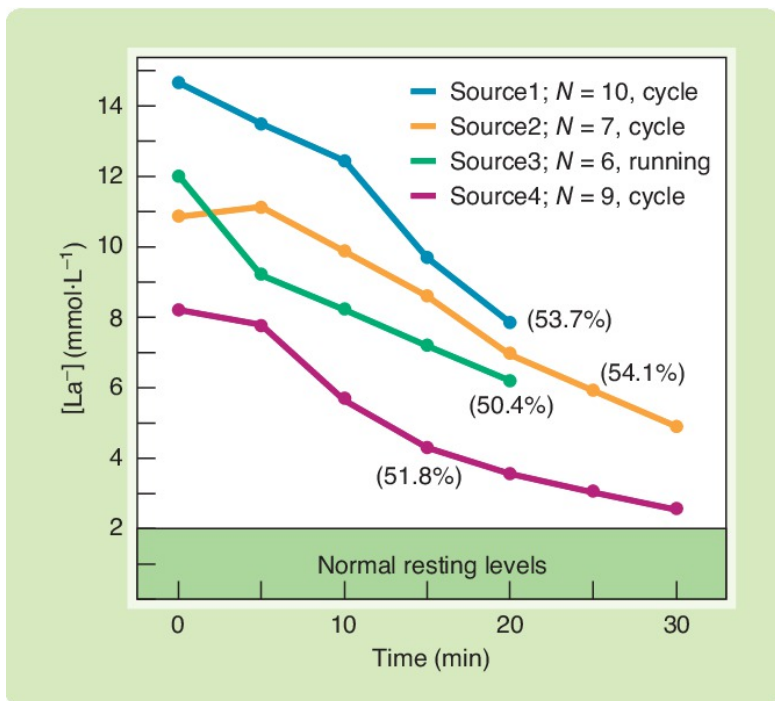


Figure 3.17 The Time Course of Lactate Removal during Resting Recovery from Exercise.

During resting recovery from exercise, lactate exhibits a half-life of 15–25 minutes. **Sources:** Based on data from Belcastro and Bonen (1975); Bonen et al. (1979); Bonen and Belcastro (1976); McGrail et al. (1978).

The second factor that determines the rate of lactate removal

is whether the individual follows a rest (passive) recovery or an exercise (active) recovery regimen. Third, with exercise recovery, the intensity of the exercise (expressed as a percentage of $\dot{V}O_{2\max}$) makes a difference. Fourth, the modality of the exercise used in the recovery phase may influence the optimal percentage of $\dot{V}O_{2\max}$ at which removal occurs. And finally, whether the recovery exercise is continuous or intermittent seems to make a difference.

Evidence suggests that lactate removal occurs more quickly when an individual exercises during recovery than when he or she sits quietly (Bangsbo et al., 1994; Nalbandian et al., 2017). **Figure 3.18** shows the results of a study conducted by Bonen and Belcastro (1976). Six trained runners completed a mile run on three different occasions. In randomized order, they then performed three different 20-minute recoveries: (1) seated rest, (2) continuous jogging at a self-selected pace, and (3) self-selected active recovery. During self-selected active recovery, the subjects did calisthenics, walked, jogged, and rested for variable portions of the total time.

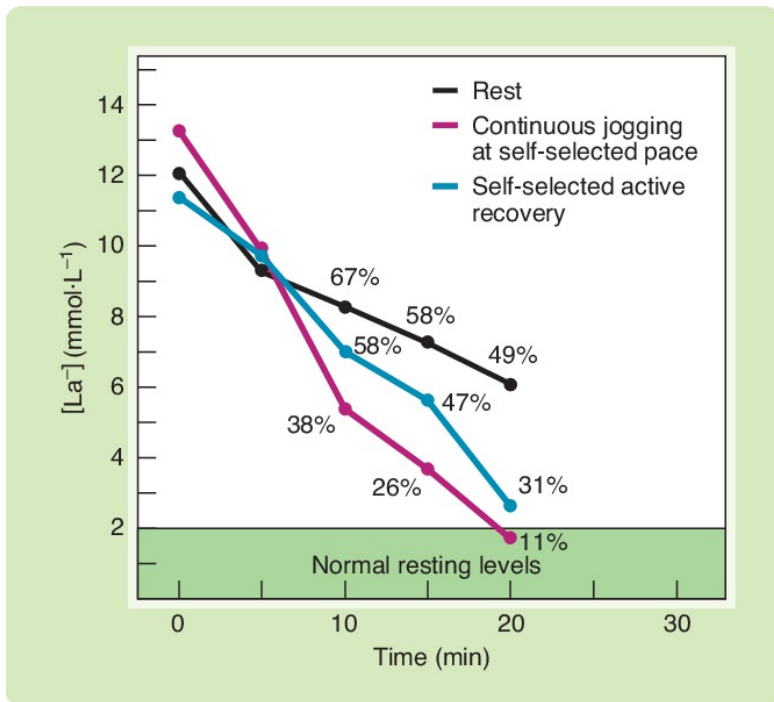


Figure 3.18 Lactate Removal in Active versus Passive Recovery.

Lactate removal is faster under active than passive conditions, although the magnitude of the benefit of active recovery depends on the type and intensity of the activity.

In this study, continuous jogging at 61.4% $\dot{V}O_2$ max was more effective than was a mixture of walking, jogging, and calisthenics. **Source:** Bonen and Belcastro (1976).

As shown in **Figure 3.18**, after 5 minutes, there is no appreciable difference in the level of lactate among the different recovery protocols. However, over the next 15 minutes, the rate of lactate removal was significantly faster for the jogging recovery than for either the self-selected active recovery or the rest recovery. The self-selected activity (what athletes typically do at a track meet), although not as good as continuous jogging (in part because it was intermittent), was still significantly better than was resting recovery.

After 20 minutes of jogging recovery, the lactate that remained was within the normal resting levels of 1–2 mmol·L⁻¹. The self-selected active recovery had removed 70% of the lactate, but the resting recovery had only removed 50% of the lactate. Thus, full recovery was, and generally is, delayed with seated rest. Because athletes who run distances from 400 to 1,500 m (or the English equivalents of 440 yd to 1 mi) often double at track meets, such a delay could impair their performances in the second event. Based on these results, athletes competing or training at distances that are likely to cause large accumulations of lactate should cool down with an active continuous recovery.

Why does activity increase the rate of lactate removal? The rate of lactate removal by the liver appears to be the same whether an individual is resting or exercising. However, during exercise, blood flow is increased, as is the oxidation of lactate by skeletal and cardiac muscles (Bangsbo et al., 1994; Belcastro and Bonen, 1975; Gollnick and Hermansen, 1973; Hogan et al., 1995; McGrail et al., 1978). These changes appear to be primarily responsible for the beneficial effects of an active recovery.

At what intensity should an active recovery be performed? Studies have found an inverted U-shaped response (Belcastro and Bonen, 1975; Dotan et al., 2000; Hogan et al., 1995). That is, up to a point, a higher intensity of exercise (as measured by percentage of $\dot{V}O_{2\max}$) during recovery is better. But after that point, as the intensity continues to rise, the removal rate decreases. The optimal rate for recovery from cycle ergometry for both children and adults is between 29 and 45% of $\dot{V}O_{2\max}$. Data from track and treadmill exercises show the same type of inverted U-shaped curve response, but in the 55–70% $\dot{V}O_{2\max}$ range (Belcastro and Bonen, 1975; Hogan et al., 1995). This difference in optimal intensity may be a function of the modality (there is a higher static component for the cycle ergometer than for running) or of the training status of the subjects tested.

The actual value of these optimal percentages should not be surprising. The LT1 has been shown to occur between 40 and 60% of $\dot{V}O_{2\max}$ and may be higher in trained individuals. Thus, it appears that the optimal intensity for recovery would be just below an individual's first lactate threshold, where lactate production is minimal but clearance is maximized. Direct

measurement of lactate clearance when recovery intensity was set according to % LT1 has confirmed this ([Devlin et al., 2014](#); [Menzies et al., 2010](#)). The rate of blood lactate clearance exhibits a dose-response relationship during active recovery. Active recovery intensities at 80–100% of LT were more effective than exercise intensities at 60, 40, or 0% LT. In the Menzies 2010 study, although there was a wide variation, in general, study participants self-selected a recovery intensity very close to the 80% LT value, so self-regulation may be possible for many individuals.

Conversely both deep and superficial massage and passive static stretching have been found to be ineffective for accelerating the rate of blood lactate clearance after exercise and may, indeed, impede it. The cause is likely related to the mechanical pressure exerted during the massage and intramuscular pressure during stretching as well as an elongation of the blood vessels running parallel to the muscle fibers—all of which result in a reduction of blood flow ([Cè et al., 2013](#); [Wiltshire et al., 2010](#)).

One word of caution: Although active recovery is best for lactate removal, it can delay glycogen resynthesis by further depleting glycogen stores ([Choi et al., 1994](#)). This has more relevance for an individual attempting to recover from a hard interval training session than for someone just completing a middle distance race and preparing for another. Glycogen depletion is likely to be more severe in the former case, and removing lactate quickly is a more immediate concern in the latter case. For athletes recovering from an interval training session or competing in heats on successive days, the best procedure may be to utilize an initial dynamic active recovery (just to the point of regaining a near-resting heart rate) and then engage in a passive recovery during which carbohydrates are consumed.

Male versus Female Anaerobic Exercise Characteristics

The anaerobic characteristics of females are generally lower than are those of males in the young and middle-aged adult years.

Much of the difference is undoubtedly related to the smaller overall muscle mass of the average female compared with that of the average male (Wells, 1991).

The Availability and Utilization of ATP-PC

Neither the local resting stores of ATP per kilogram of muscle nor the utilization of ATP-PC during exercise varies between the sexes (Brooks, 1986; Gollnick et al., 1986; Kappenstein et al., 2013; Willcocks et al., 2014). However, in terms of total energy available from these phosphagen sources, males will have more than females because of muscle mass differences.

The Accumulation of Lactate

Resting levels of lactate are the same for males and females.

Lactate thresholds, when expressed as a percentage of $\dot{V}O_{2\max}$, are also the same for both sexes, although the absolute workload at which the lactate thresholds occur is higher for males than females. Thus, at any given absolute workload that is still submaximal but above LT1 or LT2, females have a higher lactate value than do males. Consequently, the workload is more stressful for females and requires a greater anaerobic contribution.

However, at a given relative workload or percentage of $\dot{V}O_{2\max}$ above the lactate thresholds, lactate concentrations are equal for both sexes (Wells, 1991). The [La-] at maximal steady state (MLSS) is not influenced by sex (Smekal et al., 2012).

Lactate values at maximal exercise from ages 16 through 50 years are higher by approximately 0.5–2.0 mmol·L⁻¹ for males than for females (see **Figure 3.19**). Once again, females are generally doing less in terms of an absolute workload than were males at maximum.

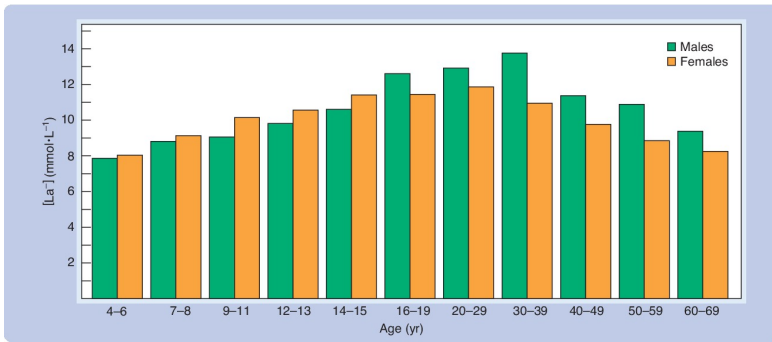


Figure 3.19 Lactate Values after Maximal Exercise as a Function of Age and Sex.

Sources: Compiled from computed mean value data for 4- to 20-year-old subjects: Åstrand et al. (1963); Cumming et al. (1985); Eriksson (1972); and Saris et al. (1985).

Computed mean value data for 20- to 60-year-old subjects: Åstrand (1952); Åstrand (1960); Bouhuys et al. (1966); Sidney and Shephard (1977); and Robinson (1938).

FOCUS ON APPLICATION

Exercise, Lactic Acid, and Lactation

The impact of the accumulation of lactate [La] following exercise on infants' acceptance of postexercise breast milk is an area of special interest. Some of the lactate present in the blood diffuses into breast milk.

Wallace and Rabin (1991) reported that the concentration of lactic acid in breast milk following maximal treadmill exercise increased from 0.79 mmol-L⁻¹ preexercise to 1.62

mmol·L⁻¹ postexercise and remained elevated for at least 30 minutes. This milk [HLA] was approximately 25% of the postexercise blood concentration. Infants' acceptance of the postexercise milk was not tested in this study, but the investigators speculated that because these levels were high enough to change the taste of the breast milk, this could be a problem. Infants have fully developed taste buds at birth and can detect sour tastes such as that produced by lactic acid.

A follow-up study (Wallace et al., 1992) again revealed elevated postmaximal exercise milk [HLA] (2.84 mmol·L⁻¹ at 10 minutes and 2.97 mmol·L⁻¹ at 30 minutes). Infants were fed 2 mL of the pre- and two postexercise milk samples in a randomized fashion through a medicine dropper. Mothers' ratings of their infants' acceptance of the milk were significantly lower for both postexercise samples than the preexercise sample.

Reasoning that nursing mothers typically would not be doing maximal exercise, Quinn and Carey (1999) compared three exercise intensities on three separate days (maximal, at LT1, and 20% below LT1 abbreviated as LT1-LT20) to a resting control trial. Mothers were divided into two groups based on controlled carbohydrate intakes of 63 or 52% of total caloric intake. Breast milk [HLA] was significantly increased immediately after maximal exercise (1.27 and 1.52 mmol·L⁻¹) and LT1 exercise (0.19 and 0.25 mmol·L⁻¹), but not after the LT1-LT20 exercise (0.11 and 0.12 mmol·L⁻¹). Only the level following maximal exercise remained elevated 30 minutes after exercise.

More recently, Wright et al. (2002) tested the impact of maximal- and moderate-intensity (LT1-LT20) intensity exercise compared to a resting control session on acceptance of postexercise breast milk by infants in a well-designed, controlled experiment. As in the prior research, breast milk [HLA] was significantly elevated over the control condition after maximal exercise (0.21 vs. 0.09 mmol·L⁻¹) but not after the moderate-intensity exercise. Infants were offered the posttesting milk in a bottle. The mother and three lactation consultants (via videotape) rated the infants' acceptance of the milk. Both the mothers and the lactation consultants

judged no differences in the infants' milk acceptance. Infants consumed an average of 104 mL of milk at the feedings.

These results seem to indicate that moderate exercise and lactation are compatible and that nursing mothers can enjoy the benefits of both for themselves and for their infants. Indeed, a 1994 study (Dewey et al., 1994) demonstrated fitness benefits for lactating women participating in a moderate submaximal aerobic exercise program (60–70% HRR progressing from 20 to 45 min·d⁻¹, 5 d·wk⁻¹ for 12 weeks). Although not a primary interest in this study, these researchers reported that none of these mothers mentioned any difficulties with nursing after exercise.

Sources: Dewey et al. (1994); Quinn and Carey (1999); Wallace and Rabin (1991); Wright et al. (2002).

Mechanical Power and Capacity

As previously mentioned, on average males produce higher absolute work output than do females. Data available from the WAnT show that values for peak power for women are approximately 65% of values for men if expressed in watts, improve to 83% if expressed in watts per kilogram of body weight, and come close to being equal at 94% when expressed in watts per kilogram of lean body mass. The corresponding comparisons for mean power are 68, 87, and 98%, respectively. The peak power of women (in watts per kilogram of body weight) is very similar to the mean power of men. The fatigue index does not show a significant sex difference, indicating that both sexes tire at the same rate (Kappenstein et al., 2013; Makrides et al., 1985). It has been shown that females provide a relatively higher portion of the energy for the WAnT aerobically than do males. This may mean that the total power output during a WAnT actually underestimates the sex difference in anaerobic capacity between males and females (Hill and Smith, 1993).

The maximal accumulated oxygen deficit (MAOD) is significantly higher in males than females (Hill, and Vingren, 2014; Weber and Schneider, 2000).

Anaerobic Exercise Characteristics of Children

The anaerobic characteristics of children are not as well developed as those of adults. Furthermore, children tend not to exhibit metabolic specialization as adults do. For example, one would not expect Usain Bolt (the 2020 world record holder in the 100-m dash, with a time of 9.58) to do well at the marathon distance, nor Brigid Kosgei (who set the women's marathon world best at 2:14:04 in 2019) to be successful at the sprint distances. Yet watch children at play (**Figure 3.20**) and, more often than not, the fastest at short distances or strength-type events also do well at long distances or in aerobic-type games such as soccer. At the same time, children naturally seem to preferentially participate in intermittent, high-intensity, short-duration activities. Although more research is needed to explain the anaerobic differences between children and adults, some patterns are apparent ([Bar-Or, 1983](#); [Rowland, 2005](#)).



Figure 3.20 Children Sprinting, an Anaerobic Activity.

The Availability and Utilization of ATP-PC

Local resting stores of ATP per kilogram of wet muscle weight appear to be the same for children and adults (Boisseau and Delamarche, 2000; Rowland, 2005). Levels of resting PC per kilogram of wet muscle weight have been reported as slightly lower in children (Bar-Or, 1983; Shephard, 1982) or no different from adult levels (Eriksson, 1972; Shephard, 1982). At the onset of high-intensity intermittent exercise, a higher portion of the ATP demand is provided by oxidative ATP formation by children compared with adults, giving children a clear advantage in the transition from rest to exercise. This means that the decrease in PC is lower in children and adolescents than adults during fatiguing high-intensity exercise. Because children initially use less PC, they can start the next repetition with a higher PC level than can adults (Kappenstein et al., 2013; Willcocks et al., 2014). Finally, children have been shown to have faster or similar intramuscular PC restoration and recovery half-times after exercise than do adults (Falk and Dotan, 2006; Kappenstein et al., 2013; Willcocks et al., 2014).

The Accumulation of Lactate

On average, blood lactate values obtained during submaximal exercise and after maximal exercise are lower in children than in adults (Boisseau and Delamarche, 2000; Rowland, 1990, 2005; Shephard, 1982; Van Praagh, 2007). Furthermore, peak lactate values after maximal exercise exhibit a relatively positive rectilinear increase with age until adulthood. This increase can be as much as 50% for boys between the ages of 6 and 14 years and slightly less for girls (Rowland, 2005). **Figure 3.19** includes children's values. It also shows that there is no meaningful sex difference in children in the accumulation of lactate, although the girls' values are slightly higher than the boys' values throughout the growth period. Anatomical differences between children and adults allow for a faster transition time for metabolites (La^- , H^+) from the muscle to blood; thus, $[\text{La}^-]$ peaks sooner after exercise in children than in adults. However, the rate of elimination (the time for half-life disappearance of La^-) is the same in children, adolescents, and adults. The lower lactate concentrations in children and adolescents are also reflected in higher pH levels during intense exercise although there is no difference in resting

muscle pH between children and adults. Children appear to recover from physical exertion more quickly than do adults, in large part because their lower maximal power output means they have less to recover from. However, the early peak of lactate is also a major advantage over adults in recovery. As a consequence, children and adolescents may require shorter rest periods between high-intensity bouts of activity (Boisseau and Delamarche, 2000; Falk and Dotan, 2006; Kappenstein et al., 2013), similar to adult endurance athletes (Birat et al., 2018).

The Lactate Threshold(s)

The phenomenon of ventilatory breakpoints and lactate thresholds is seen in children as well as in adults, but they typically occur at a higher % $\dot{V}O_{2\max}$ than for adults (Gaisl and Buchberger, 1979; Klentrou et al., 2006; Rowland, 2005). Because children do not utilize anaerobic metabolism as early in work as adults do, the work level of children at the fixed lactate levels of 4 mmol·L⁻¹ is relatively higher than that of adults (Kanaley and Boileau, 1988; Reybrouck, 1989). Williams et al. (1990) report a value of almost 92% of $\dot{V}O_{2\max}$ for 4 mmol·L⁻¹ of lactate in 11- to 13-year-old boys and girls. Values for adults tend to be about 15% below this value. Thus, children are working relatively harder (at a higher % $\dot{V}O_{2\max}$) than adults at the same lactate level. Consequently, the assessment of exercise capacity and the monitoring of training by a 4 mmol·L⁻¹ value is inappropriate in prepubertal children. MLSS (mmol·L⁻¹) is independent of age and sex between children and adults despite the fact that the MLSS workload is significantly higher in adults than in children (Beneke et al., 2009).

Mechanical Power and Capacity

Absolute peak power and relative peak power (power per kilogram of body weight) have consistently been shown to be lower in prepubertal boys and girls than in adolescents or young adults when evaluated by the Margaria-Kalamen Stair Climb test. The results are similar to those for the Wingate Anaerobic Test, although information is lacking about females for the Wingate

Anaerobic Test except for isolated ages (10–13 years) ([Armstrong et al., 1997, 2000](#); [Beneke et al., 2007](#); [Chia et al., 1999](#)) (**Figure 3.21**). In boys, peak power and mean power increase consistently from age 10 year to young adulthood. This is true both in absolute terms (watts) and when corrected for body weight (watts per kilogram). The absolute differences between children and adults, however, are much greater (children can achieve only about 30% of adult values) than the relative differences (children can achieve about 60–85% of the adult values). Peak values seem to occur in the late thirties for the legs and the late twenties for the arms ([Bar-Or, 1988](#); [Hughson et al., 1987](#)).

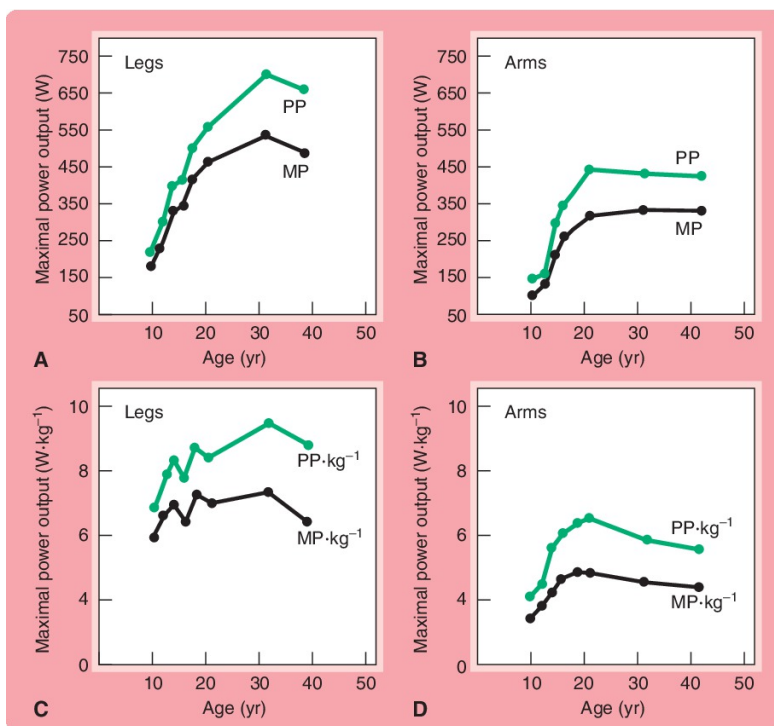


Figure 3.21 The Effect of Age on Anaerobic

Performance.

Cross-sectional data on 306 males who performed both an arm and a leg WAnT. The pattern of increase in values for both peak power (PP) and mean power (MP) from childhood to young adulthood is similar for leg (A, C) and arm (B, D) cycling whether the unit of measurement is absolute (A, B) or relative (C, D). **Source:** Reprinted with permission from Inbar, O., & O. Bar-Or: Anaerobic characteristics in male children and adolescents. *Medicine and Science in Sports and Exercise*. 18(3):264–269 (1986). Copyright ©1986 The American College of Sports Medicine.

Data are available to compare boys and girls on peak power from a variation of an all-out cycle ergometer test called the force-velocity test (Martin et al., 2004; Santos et al., 2002, 2003). The results of the most comprehensive study in terms of age range are shown in **Figure 3.22**. These results indicate that from ages 7 to 16, girls increased peak power by 273% and then plateaued between ages 16 and 17. Boys' peak power increased by 375% between ages 7 and 17. Sex differences were not apparent in peak power until age 14 (Martin et al., 2004).

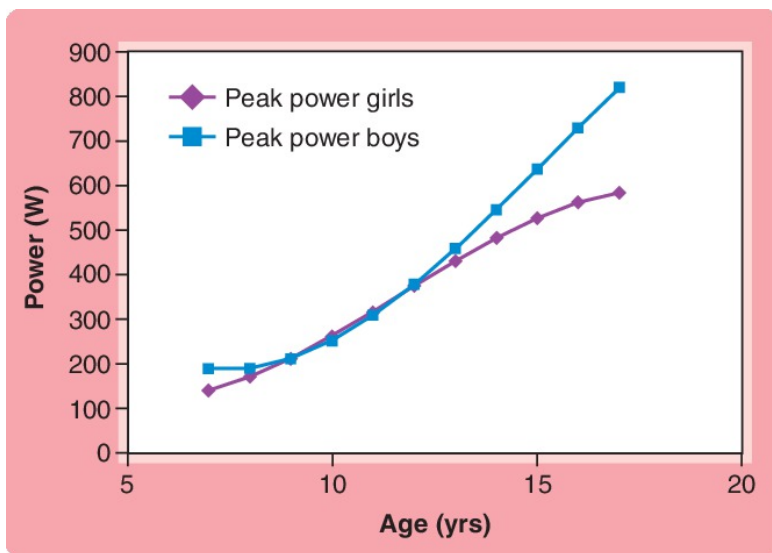




Figure 3.22 Peak Power by Age in Boys and Girls.

Force-velocity test results indicate that both boys and girls increase the production of PP from 7 to approximately 17 years of age. Sex differences are not apparent until age 14, when boys' PP production increased more than did girls'.

Source: Based on data from [Martin et al. \(2004\)](#).

Fatigue resistance to high-intensity intermittent exercise (maximal knee flexion and extension) shows a gradual decline from childhood through adolescence to young adulthood in males. In females, fatigue resistance declines from childhood to adolescence, but plateaus from there to young adulthood ([Dipla et al., 2009](#)).

Mechanisms

Several theories, described in the following sections, have been postulated to explain children's inability to function at a high level anaerobically in activities lasting 15 seconds to 2 minutes and/or requiring large power outputs. These theories are based on two assumptions: that the children tested did indeed put forth a maximal effort and that there is a physiological cause for the observed differences.

The Muscle Enzyme Theory

[Eriksson \(1972\)](#) and others have shown that phosphofructokinase (PFK) activity in 11- to 13-year-old boys is 2.5–3 times lower than in trained or sedentary adult men. Since PFK is a rate-limiting enzyme of glycolysis, this lowered activity may indicate a decreased ability to produce lactic acid. In addition, the availability and the utilization of glycogen as a substrate, as well as the activity of lactic dehydrogenase (LDH), are both lower in

children than in adults. Studies have shown that the glycogen content of children's muscles is approximately 50–60% that of adults (Boisseau and Delamarche, 2000). Anaerobic enzymes evolve with pubertal maturation, however, and by 12–14 years of age, LDH activity has reached adult levels (Berg et al., 1986). Therefore, it appears that prepubertal youngsters do have lower glycolytic enzyme activity than do fully mature individuals (Boisseau and Delamarche, 2000; Rowland, 2005).

In general, the oxidative enzymes show higher activity in young children than in older individuals (Berg et al., 1986; Williams et al., 1990). This result, along with higher mitochondrial density and intracellular lipids, means that lipid utilization is greater in children. Thus, it may be that children have a finer balance between aerobic and anaerobic metabolism than do adults. Additional evidence for this observation comes from the fact that children show a lower oxygen deficit and reach a steady state faster than do adults (Åstrand, 1952; Boisseau and Delamarche, 2000; Reybrouck, 1989; Rowland, 2005; Shephard, 1982).

Muscle Characteristics Theory

Several muscle characteristics have been suggested as explanations for the lower glycolytic anaerobic function in children and adolescents than adults. These include muscle fiber differentiation, muscle fiber recruitment, contractile properties, muscle fiber size, muscle size, and muscle mass (Boisseau and Delamarche, 2000; Falk and Dotan, 2006; Van Praagh, 2000, 2007).

The development of muscle fiber-type distribution continues from birth to approximately 6 years of age, when the individual achieves his or her profile of slow-twitch and fast-twitch fibers. However, the relative proportion of fast-twitch oxidative glycolytic (FOG) and fast-twitch glycolytic (FG) fibers continues to change through late adolescence. Typically, FOG fibers predominate more than do FG fibers during childhood and adolescence. Boys develop greater FG cross-sectional area than do girls as they mature. The contractile properties of FG fibers favor force and power expression. Children may be limited in their ability to recruit FG fibers.

Muscle fiber size, measured as diameter, and muscle size, measured as cross-sectional area, both increase rectilinearly with age from birth to young adulthood. Muscle force changes closely parallel changes in cross-sectional area, and these power changes during growth favor boys over girls after puberty. However, when normalized for muscle cross-sectional area, age and sex differences in muscle force disappear.

Finally, total muscle mass is directly related to the ability to generate force/power. As the child grows, obviously so does his or her total muscle mass. However, even normalized for muscle mass, force production is lower in children and adolescents than adults ([Van Praagh, 2000](#)). Body composition changes (more total muscle mass, less fat for boys) favor males in force production ([Armstrong et al., 1997](#)).

Sexual Maturation Theory

Limited evidence suggests that the increase in glycolytic capacity (and thus the production of lactate) in children is related to the hormonal changes that bring about sexual maturation. In particular, testosterone is thought to have a role, but research evidence has not been definitive ([Boisseau and Delamarche, 2000](#); [Rowland, 2005](#); [Williams et al., 1990](#)).

Neurohormonal Regulation Theory

It has been shown that sympathoadrenal system activity is significantly lower in children than in adults at maximal exercise ([Boisseau and Delamarche, 2000](#)). Epinephrine is a potent stimulator of muscle glycolysis. Another result of sympathetic stimulation during exercise is hepatic vasoconstriction. The liver plays a major role in the clearance of lactate. If blood flow to the liver is reduced, less lactate is cleared. Thus, because the child maintains a higher liver blood flow, more lactate can be cleared. This implies that children are not deficient in the production of lactate but are, instead, better able to remove or reconvert it than adults ([Berg and Keul, 1988](#); [Mácek and Vávra, 1985](#); [Rowland, 1990, 2005](#)).

Which of these theories—muscle enzyme, muscle characteristics, sexual maturation, neurohormonal regulation—or

which combination of them is correct remains to be shown.

Anaerobic Exercise Characteristics of Older Adults

Detailed evidence of the anaerobic characteristics of older adults is scarce, undoubtedly in part because of caution by researchers and in part because of the uncertain motivation of subjects facing high-intensity exercise. The available data indicate that anaerobic variables show a common aging pattern; that is, there is a peak in the second or third decade and then a gradual decline into the sixth decade. One must always remember when interpreting aging results, however, that no one knows how much of this reduction results directly from aging, how much results from detraining accompanying the reduced activity of the elderly, and how much results from disease. Despite this, older adults can still participate successfully in basically anaerobic activities (**Figure 3.23**).



Figure 3.23 Older Adult Engaged in Anaerobic Activity.

Anaerobic metabolic processes function less effectively in older individuals than in younger adults, but participation in anaerobic activity is still possible.

The Availability and Utilization of ATP-PC

Local resting stores of ATP-PC are reduced and levels of creatine and ADP are elevated in muscles of older adults ([Kanaley and Boileau, 1988](#)). Results from the Margaria-Kalamen Stair Climb test have shown a reduction in ATP-PC power of as much as 45% and a reduction in ATP-PC capacity of 32% from youth to old age ([Shephard, 1982](#)). This means that ATP-PC stores are both reduced and unable to be used as quickly. The result is a decrease in alactic anaerobic power.

The Accumulation of Lactate

On average, resting concentrations of blood lactate are

remarkably consistent across the entire age span, varying only from 1 to 2 mmol·L⁻¹.

Lactate values during the same absolute submaximal work tend to be higher in individuals over the age of 50 (Åstrand, 1952, 1956, 1960; Robinson, 1938). However, this generalization is confounded by the fact that at any given absolute load of work, the older individual is working at a higher percentage of his or her maximal aerobic power, which would be expected to involve more anaerobic metabolism (Sidney and Shephard, 1977). When younger and older individuals work at the same relative workload (% $\dot{V}O_{2\max}$), lactate concentrations are lower in older people than in the young, probably because the elderly are using less muscle mass to do less work (Kohrt et al., 1993).

Maximal lactate concentrations as a result of incremental dynamic exercise to maximum reach a peak between 16 and 39 years of age and then show a gradual decline (see **Figure 3.19**). Both males and females exhibit the same pattern, although the absolute values of females are considerably lower than are those of males.

Direct measurement of ATP produced from anaerobic glycolysis indicates that older males have an overall lower production than do younger males (Lanza et al., 2005) during 60 seconds of maximal static contraction. Because this reflects a lower utilization of the glycolytic pathway, exercise lactate values should be lower.

Physiological factors appear to contribute to the decline in anaerobic ATP production and maximal lactate values with age (Shephard, 1982; Smith and Serfass, 1981). First, activity of the enzyme lactate dehydrogenase (which catalyzes the conversion of pyruvic acid to lactic acid) decreases in all muscle fiber types. This decrease effectively slows glycolysis. The reduction in glycolysis may also be related to lower amounts of glycogen stored in the skeletal muscles of the elderly.

Second, older individuals possess a smaller ratio of muscle mass to blood volume, a smaller ratio of capillary to muscle fiber, and a concomitant slower diffusion of lactic acid out of active muscle fibers and into the bloodstream (Shephard, 1982). Thus, it may not be that the elderly have a large deficiency in anaerobic capacity at the cellular level; rather, it may simply be that

measurements from blood samples are underestimations. Of course, a combination of the first and second factors may also be operating.

Lactate Threshold(s)

Although individuals continue to exhibit at least one lactate threshold as they age, the point at which this is evident, expressed as % $\dot{V}O_{2\max}$, appears to shift to a higher value in both sexes. In a study of 111 male and 57 female runners from 40 to 70 years of age, the lactate threshold increased from approximately 65 to 75% across the decades. However, not all agree. Data on older Master's endurance athletes have shown a reduction in LT as age increases that contributes to reduced endurance performance but that this is secondary to a reduction in $\dot{V}O_{2\max}$. However, as a percentage of $\dot{V}O_{2\max}$, there appears to be no change in these older athletes (Tanaka and Seals, 2008). In addition, there is some controversy about the ability of the LT to predict endurance performance in this age group (Wiswell et al., 2000).

Mechanical Power and Capacity

The average peak power value obtained on the Margaria-Kalamen Stair Climb test (Figure 3.24A) declines precipitously from 20 to 70 years of age (Bouchard et al., 1982). There are no published results for the Wingate Anaerobic Test for individuals over the age of 40 and for females except in the 18- to 28-year range (Maud and Shultz, 1989). However, Makrides et al. (1985) presented data on 50 male and 50 female subjects from 15 to 70 on a test similar to the WAnT (Figure 3.24B). In this case, peak power represented an instantaneous value rather than a 5-second value. Mean power was still the average of 30 seconds of pedaling, but at a controlled rate of 60 rev·min⁻¹. The results in this study showed a decline of approximately 6% for each decade of age for sedentary individuals of both sexes. However, the absolute values for the females were consistently lower than those for the males. Indeed, in the Makrides study, the peak power of the females coincided with the mean power of the

males. For both sexes, lean thigh volume was closely related to peak power and mean power, but this did not account for all the variation in the values or in the decline with age. A more recent study (Kostka et al., 2009) has shown a decline in maximal anaerobic power per kg of body weight of approximately 10.3% per decade from 20 to 88 years in males.

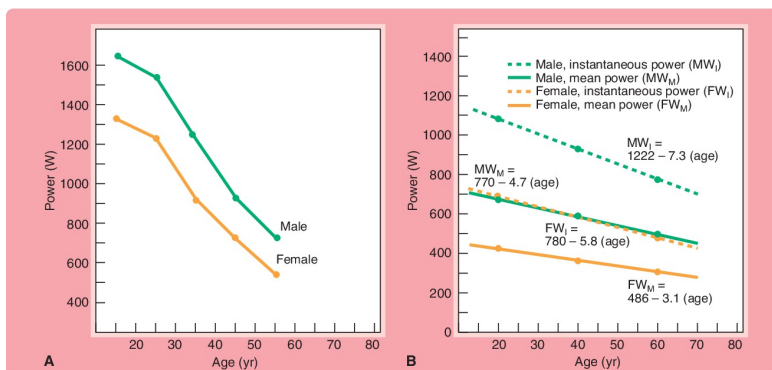


Figure 3.24 Mechanical Power Changes with Age in Males and Females.

A. Average peak power ratings determined from the Margaria-Kalamen Stair Climb are higher for males than females across the age span from adolescence to middle adulthood. Males and females show steady and parallel declines in peak power during the adult years. **B.**

Instantaneous peak power and mean power measured during a 30-second cycle ergometer test are higher for males (MW_I and MW_M, respectively) than for females (FW_I and FW_M, respectively) across the age span from late adolescence to older adulthood. Female instantaneous peak power (FW_I) is equal to male mean power (MW_M).

Instantaneous peak power and mean power show rectilinear and parallel declines with age in males and females. Each

line was calculated from the experimentally determined equation associated with that line on the graph. **Sources:** (A) Data from [Bouchard et al. \(1982\)](#). (B) Data from [Makrides et al. \(1985\)](#).

An analysis of weight lifting (e.g., snatch, clean, and jerk) and power lifting (e.g., dead lift, squat, and bench press) records for males and females from 40 to 70 years showed declines in anaerobic muscle performance. The decline was greatest (and curvilinear) in the weight-lifting tasks that required explosive coordinated movements and high-balance skills. The decline in weight lifting was higher in females (approximately -70%) than males (approximately -50%). The overall decline in the power lifts was rectilinear and similar in males (approximately -40%) and females (approximately -50%). Upper and lower body muscular power demonstrated similar rates of decline with aging ([Anton et al., 2004](#)).

Undoubtedly, muscle mass is lost with age. Between the ages of 30 and 70, almost 25% of muscle mass is lost in both males and females ([Rogers and Evans, 1993](#)). Obviously, the loss of muscle mass leads to a concomitant loss in force and power production. In addition, as individuals age, the percentage of fast-twitch muscle fibers, particularly fast-twitch glycolytic fibers, decreases. With this shift, there is a decline in potential glycolytic function and force production. Finally, neural activation and integration decline with age. Thus, moves requiring high levels of coordination may present a greater risk ([Anton et al., 2004](#); [Larsson et al., 2001](#)).

Summary

1. Anaerobic metabolism does not require oxygen to produce adenosine triphosphate (ATP), but aerobic metabolism does.
2. Anaerobic and aerobic metabolism work together to provide ATP and thus energy for exercise. Depending on the duration and intensity of the activity, one or the other predominates.
3. The adenosine triphosphate-phosphocreatine (ATP-PC)

system predominates in high-intensity exercise that lasts 30 seconds or less. The LA system predominates in high-intensity exercise lasting 30–75 seconds. The aerobic system predominates in exercise lasting from approximately 2 minutes to hours. This sequence is called the time-energy system continuum.

4. There is no generally accepted way to directly measure the anaerobic energy contribution to exercise. One indirect approach is to describe the changes in ATP, PC, and lactate levels. Another is to quantify the amount of work performed or power generated during short-duration, high-intensity activity.
5. Lactate production depends on the use of glycogen as fuel, the formation of pyruvate, and the necessity of preserving the redox potential of the cell. It results from:
 - a. Muscle contraction that in turn depends on calcium release and results in glycogenolysis
 - b. The high activity of the enzyme lactate dehydrogenase compared with other glycolytic and oxidative enzymes
 - c. Recruitment of fast-twitch glycolytic (FG, type IIA and FOG, type IIX) muscle fibers
 - d. Activation of the sympathetic nervous system, which ultimately results in glycogenolysis
 - e. Insufficient oxygen, which results in anaerobiosis or the onset of anaerobic metabolism
6. Lactate clearance utilizes both an intracellular and extracellular shuttle system. MCT1 and MCT4 transporters move lactate by facilitated exchange down concentration and pH gradients.
7. Lactate accumulation results when production (appearance) exceeds clearance (disappearance).
8. The onset of all exercise is characterized by a discrepancy between oxygen demand and oxygen utilization known as the oxygen deficit. This deficit is due to the varying speeds of response of the two anaerobic and aerobic systems to the contraction of muscle and the breakdown of ATP.
9. During the transition from rest to exercise—the O₂ deficit

period—energy is supplied by:

- a. Oxygen transport and utilization
 - b. The use of oxygen stores in venous blood and on myoglobin
 - c. The splitting of stored ATP-PC
 - d. Anaerobic glycolysis with the concomitant production of lactic acid
10. During recovery from exercise, oxygen consumption remains elevated, a phenomenon called excess postexercise oxygen consumption (EPOC). The following factors appear to be responsible for EPOC.
- a. Restoration of ATP-PC stores
 - b. Lactate removal
 - c. Restoration of oxygen stores
 - d. Elevated cardiorespiratory function
 - e. Elevated hormonal levels
 - f. Elevated body temperature
 - g. Energy substrate shift from CHO to fat
11. During exercise, the balance between aerobic and anaerobic metabolism depends on the intensity of the activity in relation to the individual's maximal ability to produce energy using oxygen ($\dot{V}O_{2\max}$).
12. During high-intensity, short-duration (3 minutes or less) anaerobic exercise, ATP levels decrease 30–40%, PC levels decrease 60–70%, and lactate accumulation can increase well over 1,000%.
13. The lactate response (increase, decrease, no change after an initial rise) to long-term moderate to heavy submaximal exercise depends to a large extent on the intensity of the exercise in relation to the maximal lactate steady-state (MLSS) intensity.
14. During incremental work to maximum, lactate accumulates slowly until approximately 40–60% $\dot{V}O_{2\max}$ when the continual accumulation is exponentially curvilinear. The term lactate threshold (LT) is commonly used to describe the

point where large increases in lactate accumulation occur.

15. LT1 is typically found between 40 and 60% $\dot{V}O_{2\max}$, and LT2 is found between 80 and 95% $\dot{V}O_{2\max}$. An absolute value of 4 mmol·L⁻¹ is termed the onset of blood lactate accumulation (OBLA) and is often used as a training and racing guideline for adults.
16. The relationship between the lactate thresholds and ventilatory thresholds appears to be primarily coincidental.
17. The term anaerobic threshold is a misnomer and should not be used because the presence of lactate does not automatically mean that the oxygen supply is inadequate.
18. Lactate has benefits that include serving as:
 - a. An energy source
 - b. A gluconeogenic precursor
 - c. A signaling molecule
19. Performance decrements are related to high concentrations of H⁺ because of:
 - a. Pain
 - b. Reduced production of ATP through inactivation of enzymes or changes in membrane transport
 - c. Inhibition of energy substrate availability because glycogen breakdown is slowed or because fatty acid mobilization is slowed
 - d. Possible reduced force and velocity of muscle contraction
20. Lactate removal from the bloodstream after exercise follows the law of mass action: the more lactate present, the faster the rate of removal. The half-life of lactate is approximately 15–25 minutes, with full removal achieved between 30 and 60 minutes.
21. The time required for lactate removal can be decreased by doing an active recovery at an intensity of approximately 30–45% $\dot{V}O_{2\max}$ for cycling and 55–70% $\dot{V}O_{2\max}$ for running—at or slightly below the first lactate threshold.
22. During the adult years, males accumulate higher levels of blood lactate as a result of maximal work than do females,

- and males exhibit higher mechanical power and capacity even when they are adjusted for body weight.
23. Peak lactate values after incremental exercise to maximum decline after approximately 40 years of age. Females exhibit considerably lower peak values than do males.
 24. The anaerobic capacities of children are not well-developed compared to adults. The lower amount of ATP-PC available is related to children's small body size.
 25. Peak lactate values after incremental exercise to maximum are lower in children than adults and vary directly with age throughout the growth years. There is no meaningful male-female difference in the ability to accumulate lactate during childhood.
 26. Mechanical power and capacity are lower for children and adolescents than for adults, whether expressed in absolute terms or corrected for body weight.
 27. Anaerobic metabolic processes decline from young and middle-aged adults to older adults. The decline includes lower resting levels of ATP-PC.
 28. The mechanical power and capacity of older adults decline steadily. The decline is greater in females than in males.

Review Questions

1. Describe the energy continuum. For each of the following sports or events, determine the percentage contribution from the anaerobic and aerobic systems.
 - a. 100-m dash
 - b. 800-m run
 - c. Soccer (not goalie)
 - d. Triathlon
 - e. Volleyball spike
 - f. 100-m swim
 - g. Mile run
 - h. Stealing a base

i. Wrestling period

2. List the major variables typically measured to describe the anaerobic response to exercise. Where possible, provide an example of an exercise test that can measure the variable.
3. Explain the five physiological reasons for the production of lactate. What determines whether or not lactate accumulates in the blood? How is lactate cleared?
4. Rank the ATP-PC, LA, and O₂ systems from highest to lowest in terms of (a) power and (b) capacity. What is the difference between power and capacity?
5. Diagram the oxygen deficit and excess postexercise oxygen consumption for an activity that requires 110% $\dot{V}O_{2\max}$ in an individual whose $\dot{V}O_{2\max}$ equals 4 L·min⁻¹ and whose resting oxygen is 0.25 L·min⁻¹. Explain how energy is provided during the oxygen deficit time period and why oxygen remains elevated during recovery.
6. Diagram and explain the changes that take place in ATP, PC, and [La⁻] during constant-load, heavy exercise lasting 3 minutes or less.
7. Explain the concept of maximal lactate steady state and why MLSS is important in endurance performance.
8. Diagram the lactate response to incremental work to maximum. The ventilatory and lactate thresholds often occur at approximately the same time. Debate whether this is a result of cause and effect or coincidence. Can either the lactate thresholds or the ventilatory thresholds be accurately described as anaerobic thresholds? Why or why not?
9. What are the physiological effects of lactate production and lactate accumulation on metabolic energy production?
10. What is the best way to clear lactate quickly during recovery?
11. What are the effects of sex and age on anaerobic metabolism during exercise?
12. Compare the positive and negative effects of lactate and lactate accumulation.

Literature Search

1. We discussed lactate threshold in this chapter. Lactate threshold is important to understand to develop exercise training intensities and training plans for some athletes. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.
 - a. Search lactate threshold, this will yield a huge selection of articles.
 - b. Refine your search using key terms that may reflect your interest in this area. For example:
 - i. Lactate threshold and altitude
 - ii. Lactate threshold and training
 - iii. Lactate threshold and sex
 - iv. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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4 Aerobic Metabolism during Exercise



CHAPTER OUTLINE

Introduction

Laboratory Measurement of Aerobic Metabolism

- Calorimetry

- Spirometry

Aerobic Exercise Responses

- Oxygen Consumption and Carbon Dioxide Production

- The Oxygen Cost of Breathing

- Respiratory Quotient/Respiratory Exchange Ratio

- Estimation of Caloric Expenditure

- The Metabolic Equivalent

Field Estimates of Energy Expenditure during Exercise

Metabolic Calculations Based on Mechanical Work or
Standard Energy Use

Motion Sensors and Accelerometers

Activity Recalls and Questionnaires

Efficiency and Economy

Efficiency

Economy of Walking and Running

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- List and explain the major variables used to describe the aerobic metabolic response to exercise.
- Explain the laboratory and field assessment techniques used to obtain information on aerobic metabolism during exercise.
- Compare and contrast oxygen consumption during aerobic (1) short-term, light- to moderate-intensity exercise; (2) long-term, moderate to heavy submaximal exercise; (3) incremental aerobic exercise to maximum; (4) static and dynamic resistance exercise; and (5) very-short-term, high-intensity anaerobic exercise.
- Describe how the oxygen cost of breathing changes during exercise.
- Calculate the respiratory exchange ratio and interpret what it means in terms of energy substrate utilization.
- Calculate the metabolic cost of activity in both kilocalories and metabolic equivalents, and explain how each can be applied.
- Differentiate among gross efficiency, net efficiency, and delta efficiency; differentiate between the efficiency and the economy of movement.
- List the ways in which an exercising individual can optimize

his/her efficiency in walking/running or cycling events.

- Compare the walking and running economy of children and older adults with that of young or middle-aged adults, and discuss possible reasons for the differences.
- Explain why efficiency and economy are important for exercise performance.

Introduction

Chapter 3 concentrates on anaerobic exercise responses: situations in which energy is provided predominantly by stored ATP-PC or by the production of ATP through anaerobic glycolysis. **Chapter 3** also describes anaerobic participation in exercises of lower intensity and longer duration and incremental exercise to maximum. This chapter focuses on the aerobic responses to the different intensities, durations, and types of exercise. Keep in mind throughout this chapter that aerobic metabolism predominates in activity lasting approximately 2 minutes or longer but that the onset of all activity involves some amount of anaerobic metabolism during the oxygen deficit period. Excess postexercise oxygen consumption (EPOC) occurs following submaximal as well as maximal exercise and is evident following aerobic as well as dynamic resistance exercise.

Laboratory Measurement of Aerobic Metabolism

The primary goal of measuring aerobic metabolism is to quantify how much energy is needed to complete a given activity. This goal can be approached in two ways. To understand these approaches, consider two known aspects of aerobic metabolism. First, aerobic metabolism requires oxygen; and second, it produces heat as a byproduct. Therefore, aerobic metabolism can be assessed by measuring oxygen consumption or heat production. Oxygen consumption is measured by indirect open-circuit spirometry, and heat production is measured by calorimetry, as summarized in **Figure 4.1**.

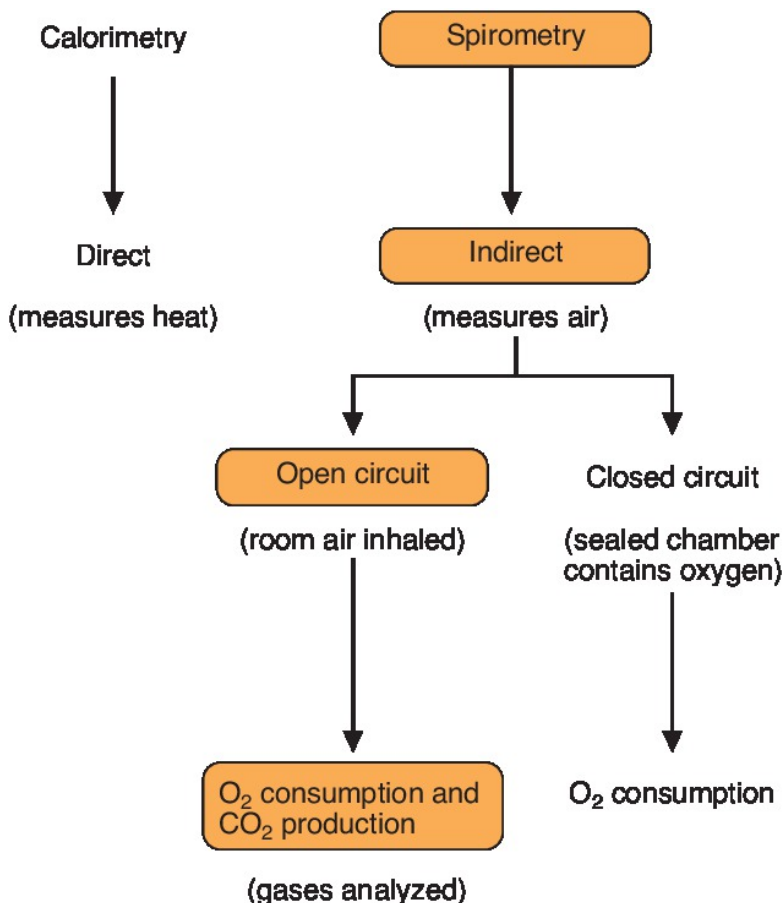


Figure 4.1 Measurement of Aerobic Metabolism.

Aerobic metabolism can be measured by direct calorimetry or indirect spirometry. In exercise physiology, open-circuit indirect spirometry/open-circuit indirect calorimetry, in which expired gases are analyzed for O₂ consumed and CO₂ produced, is typically used. **Note:** Colored boxes indicate processes typically used in exercise physiology.

Calorimetry

The term *calorimetry* is derived from the word *calorie*, the basic unit of heat energy. **Calorimetry** is the measurement of heat

energy liberated or absorbed in metabolic processes. *Direct calorimetry* actually measures heat production. This measurement requires the use of specially constructed chambers in which the heat produced by a subject increases the temperature of the air or water surrounding the walls and is thereby measured.

Calorimetry The direct measurement of heat energy liberated or absorbed in metabolic processes.

Accurate exercise data are difficult to obtain because exercise equipment, even if it fits into the usually small space required, can also emit heat. In addition, the body may store heat (as evidenced by a rise in body temperature) and/or sweat (which must be accounted for). Despite these drawbacks, the direct measurement of heat is the most precise use of the term *calorimetry*.

Spirometry

Spirometry is an indirect calorimetry method for estimating heat production in which expired air is measured and analyzed for the amount of oxygen consumed and carbon dioxide produced. It is also the direct measurement of air breathed. This method is based on the fact that oxygen consumption at rest or during submaximal exercise is directly proportional to the aerobic production of ATP and, when expressed as calories, is equal to the heat produced by the body as measured by direct calorimetry. However, since heat is not measured directly, spirometry is an *indirect* measure.

Spirometry An indirect calorimetry method for estimating heat production in which expired air is analyzed for the amount of oxygen consumed and carbon dioxide produced.

In a *closed system*, the subject breathes from a sealed container filled with gas of a designated composition (often 99.9% O₂).

Expired CO₂ is usually absorbed by a chemical such as soda lime. The rate of utilization of the available O₂ is then determined. This system has a large error and is rarely used. It is mentioned here so that the terminology of an open circuit can be understood.

In *open-circuit spirometry*, the subject inhales room or outdoor air from his or her surroundings and exhales into the same surroundings. The oxygen content of the inhaled air is normally 20.93%; the carbon dioxide does not need to be absorbed but is simply exhaled into the surrounding atmosphere. A sample of the expired air is analyzed for oxygen and carbon dioxide content.

Putting these factors together results in the descriptor *open-circuit indirect spirometry*. The term *open-circuit indirect calorimetry* should technically be reserved for use when calories are calculated from oxygen consumption, but, in fact, that term is often used interchangeably with open-circuit indirect spirometry, and we will do so in this text.

Measuring oxygen consumption by open-circuit indirect spirometry is a valid way to assess aerobic metabolism during resting and steady-state submaximal exercise conditions when the relationship between oxygen consumption and ATP production remains linear. However, in situations also involving anaerobic energy production, the actual energy cost of the exercise will be underestimated, because the linear relationship no longer exists and there is no way to account for the anaerobic portion.

Open-circuit indirect spirometry can be used to measure oxygen consumption during any physical activity. However, the size, sensitivity, and lack of portability of the equipment have, until recently, limited its use to modalities that can be performed in a laboratory or a special swimming pool setup. By far, the most popular exercisetesting modalities in the laboratory are the motor-driven treadmill and the cycle ergometer. Measurements can be performed with the subject at rest, during submaximal exercise, or at maximal levels of exertion. The following sections describe in detail how these measurements are done and the aerobic responses to varying patterns of exercise.

Aerobic Exercise Responses

Figure 4.2 shows an individual attached to an analysis system for open-circuit indirect spirometry in a laboratorybased setup. The individual uses a breathing valve, which permits air to flow in only one direction at a time—in from room air and out toward the sampling chamber. The nose clip ensures that the individual breathes only through the mouth.

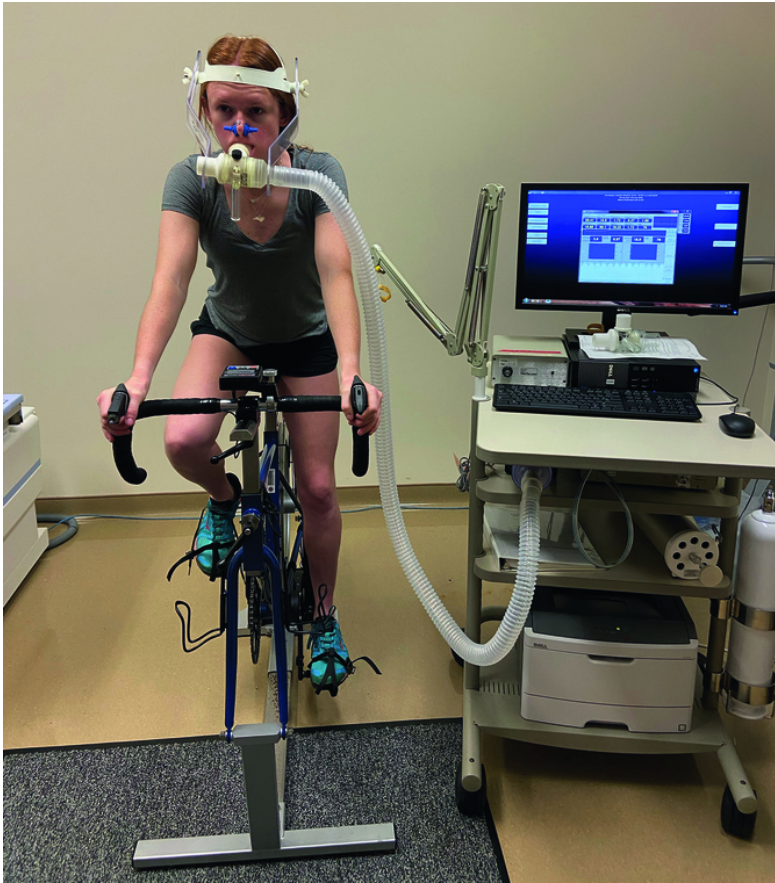


Figure 4.2 Subject Undergoing Assessment of Aerobic Metabolism during Exercise.

See animation, Oxygen Consumption, on Lippincott Connect.

Oxygen Consumption and Carbon Dioxide Production

Figure 4.3 shows schematic configurations of an open-circuit system in which the volume of either inspired air (**Figure 4.3A**) or expired air (**Figure 4.3B**) is measured and the expired air is analyzed for the percentage of oxygen and carbon dioxide. Although oxygen consumption is the variable of primary interest, given its direct relationship with ATP, determining the amount of carbon dioxide produced is also important because that measure enables a determination about fuel utilization and caloric expenditure.

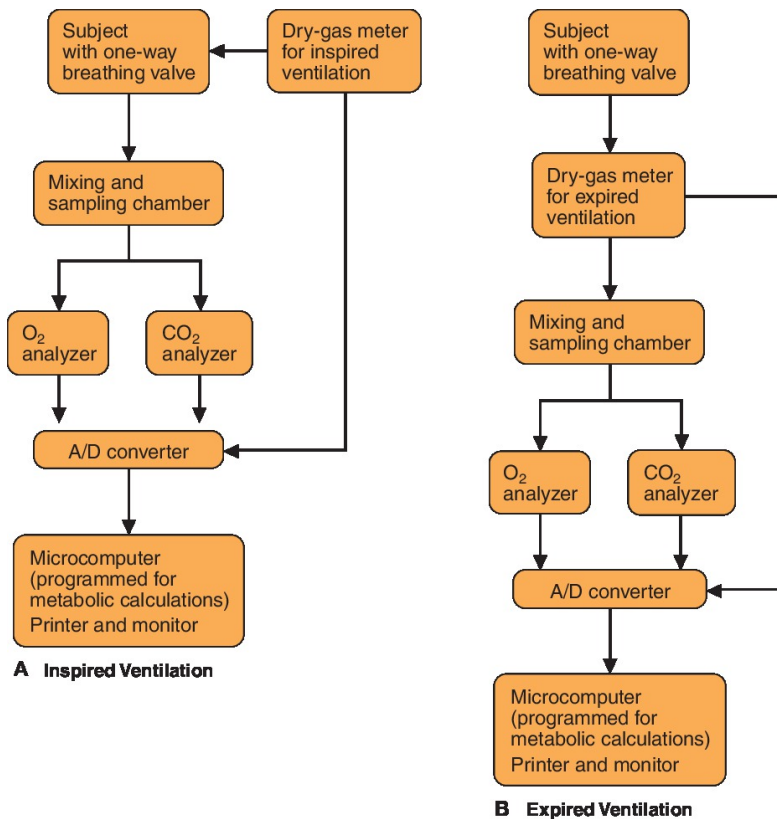


Figure 4.3 Open-Circuit Indirect Spirometry with Online Computer Analysis.

A. Flow chart for measurement of oxygen consumption when inspired ventilation is measured. **B.** Flow chart for measurement of oxygen consumption when expired ventilation is measured.

Oxygen consumption ($\dot{V}O_2$) is technically the *amount* of oxygen taken up, transported, and used at the cellular level. It equals the amount of oxygen inspired minus the amount of oxygen expired. However, as the symbol $\dot{V}O_2$ indicates, it is commonly labeled as the *volume* of oxygen consumed. Similarly, **carbon dioxide produced** ($\dot{V}CO_2$) is technically the amount of carbon dioxide generated during metabolism, primarily from

aerobic cellular respiration. It equals the amount of carbon dioxide expired minus the amount of carbon dioxide inspired. As with $\dot{V}O_2$, $\dot{V}CO_2$ is commonly described as the volume of carbon dioxide produced.

Oxygen Consumption ($\dot{V}O_2$) The amount or volume of oxygen taken up, transported, and used at the cellular level.

Carbon Dioxide Produced ($\dot{V}CO_2$) The amount or volume of carbon dioxide generated during metabolism.

The *amount* of a gas equals the volume of air (either inhaled or exhaled) times the percentage of the gas. Therefore, to determine these amounts, the volume of air either inhaled or exhaled is measured, as are the percentages of oxygen and carbon dioxide in the exhaled air. The percentages of oxygen and carbon dioxide in inhaled air are known to be 20.93% and 0.03%, respectively. These mathematical relationships can be formulated as follows:

oxygen consumption ($L \cdot min^{-1}$) = (volume of air inspired [$L \cdot min^{-1}$] \times percentage of oxygen in inspired air) – (volume of air expired [$L \cdot min^{-1}$] \times percentage of oxygen in expired air)

or

$$4.1 \quad \dot{V}O_{2\text{cons}} = (\dot{V}_I \times \%O_2 \text{ insp}) - (\dot{V}_E \times \%O_2 \text{ expir})$$

carbon dioxide produced ($L \cdot min^{-1}$) = (volume of air expired [$L \cdot min^{-1}$] \times percentage of carbon dioxide in expired air) – (volume of air inspired [$L \cdot min^{-1}$] \times percentage of carbon dioxide in inspired air)

or

$$4.2 \quad \dot{V}CO_{2\text{prod}} = (\dot{V}_E \times \%CO_2 \text{ expir}) - (\dot{V}_I \times \%CO_2 \text{ insp})$$

The volume of air inhaled is usually not exactly equal to the volume of air exhaled. However, knowing either the value of air inhaled or exhaled and the gas percentages allows us to calculate the other air volume. These calculations are fully described in [Appendix B](#).

Most laboratories use computer software to solve [Equations 4.1](#) and [4.2](#). **Table 4.1** gives the results of such a computer program for a metabolic test. The first 3 minutes represent resting values. The values for the next 8 minutes were obtained during treadmill walking at $3.5 \text{ mi}\cdot\text{hr}^{-1}$ ($94 \text{ m}\cdot\text{min}^{-1}$ or $5.6 \text{ km}\cdot\text{hr}^{-1}$) at 7% grade. Ignore for the time being minutes 33 through 37 as well as the missing minutes. Locate in [Figure 4.3](#) the pieces equipment referred to in the following discussion. The dot above the volume symbol (\dot{V}) indicates per unit of time, which is usually 1 minute.

TABLE 4.1 Aerobic Metabolic Responses at Rest and during Submaximal Exercise

| Sex = Female | | Ambient Temperature = 18°C | | | | | | |
|--|---|--------------------------------|-------------------|-------------------------------------|--------------------------------------|--|------|---------------------------|
| Age = 22 y | | Barometric Pressure = 752 mmHg | | | | | | |
| Weight = 53.4 kg | | Relative Humidity = 5% | | | | | | |
| Time (min) | \dot{V}_E STPD (L·min ⁻¹) | O ₂ % | CO ₂ % | $\dot{V}O_2$ (L·min ⁻¹) | $\dot{V}CO_2$ (L·min ⁻¹) | $\dot{V}O_2$ (mL·kg ⁻¹ ·min ⁻¹) | RER | HR (b·min ⁻¹) |
| Rest (Standing) | | | | | | | | |
| 1 | 7.57 | 17.11 | 3.26 | 0.30 | 0.23 | 5.61 | 0.81 | 75 |
| 2 | 7.58 | 17.21 | 3.20 | 0.29 | 0.23 | 5.43 | 0.82 | 75 |
| 3 | 8.64 | 16.95 | 3.37 | 0.34 | 0.28 | 6.55 | 0.80 | 75 |
| \bar{x} | 7.93 | 17.09 | 3.28 | 0.31 | 0.25 | 5.86 | 0.81 | 75 |
| 3.5 mi·hr ⁻¹ Walking (Steady State); 7% Grade | | | | | | | | |
| 1 | 21.96 | 15.85 | 4.34 | 1.14 | 0.93 | 21.53 | 0.81 | 120 |
| 2 | 24.20 | 15.85 | 4.60 | 1.25 | 1.10 | 23.59 | 0.87 | 136 |
| 3 | 23.46 | 15.73 | 4.76 | 1.25 | 1.10 | 23.40 | 0.88 | 136 |
| 4 | 26.07 | 16.02 | 4.66 | 1.29 | 1.20 | 24.15 | 0.93 | 136 |
| 5 | 25.71 | 15.95 | 4.73 | 1.29 | 1.20 | 24.34 | 0.92 | 136 |
| 6 | 27.53 | 16.03 | 4.68 | 1.35 | 1.27 | 25.46 | 0.93 | 136 |
| 7 | 25.71 | 15.91 | 4.79 | 1.29 | 1.22 | 24.34 | 0.93 | 136 |
| 8 | 28.64 | 16.06 | 4.68 | 1.39 | 1.33 | 26.21 | 0.94 | 136 |
| \bar{x} | 25.41 | 15.93 | 4.66 | 1.28 | 1.17 | 24.13 | 0.90 | 136 |
| 3.5 mi·hr ⁻¹ Walking (Oxygen Drift); 7% Grade | | | | | | | | |
| 33 | 28.64 | 16.26 | 4.56 | 1.33 | 1.29 | 25.09 | 0.96 | 140 |
| 34 | 31.21 | 16.33 | 4.55 | 1.43 | 1.41 | 26.96 | 0.98 | 144 |
| 35 | 31.21 | 16.32 | 4.54 | 1.43 | 1.39 | 26.96 | 0.97 | 144 |
| 36 | 30.84 | 16.16 | 4.67 | 1.47 | 1.43 | 27.71 | 0.96 | 146 |
| 37 | 32.31 | 16.19 | 4.68 | 1.52 | 1.50 | 28.65 | 0.97 | 150 |

This individual had a $\dot{V}O_2 \text{ max}$ of $47.64 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, or $2.54 \text{ L}\cdot\text{min}^{-1}$.

The volume of air inspired (\dot{V}_I) or expired (\dot{V}_E) is measured by a pneumoscan or flowmeter (labeled as the dry gas meter in **Figure 4.3**). Typically, all ventilation values are reported as expired values that have been adjusted to standard temperature (0°C), standard pressure (760 mmHg), and dry (without water vapor) conditions, known as STPD. The process and rationale for standardizing ventilatory volumes are discussed fully in **Chapter 9**. Standardization permits comparisons between data collected under different conditions. Two things should be noted about the \dot{V}_E STPD values. First, the values for exercise (Ex) are higher than those at rest (R) ($\bar{X}_R = 7.93 \text{ L}\cdot\text{min}^{-1}$, $\bar{X}_{Ex} = 25.41 \text{ L}\cdot\text{min}^{-1}$). Second, within each condition, the values are very stable.

The air the subject exhales is sampled from a mixing chamber and analyzed by electronic gas analyzers that have been previously calibrated by gases of known composition. Data about the percentage of expired oxygen (O2% in **Table 4.1**) and carbon dioxide (CO2% in **Table 4.1**) are relayed from the gas analyzers along with ventilation values through an analog-to-digital (A/D) converter to the computer.

Remember that room air is composed of 20.93% O2, 0.03% CO2, 79.04% N2, and other trace elements such as argon and krypton. N2 is considered to be inert in human metabolism. However, as we have seen in the metabolic pathways, O2 is consumed and CO2 is produced when ATP is generated aerobically. Therefore, in general, the exhaled O2% will be lower than the value in ambient air, ranging around 15–16% during moderate exercise. Exhaled CO2 values will be greater than that in room air, increasing to somewhere around 4–6% during moderate exercise. Note these values in **Table 4.1**. During rest, not much oxygen is used, so the percentage exhaled is relatively high ($\bar{X}_R = 17.09\%$). The accompanying percentage of CO2 is, as would be expected, relatively low ($\bar{X}_R = 3.28\%$). During exercise, more O2 is used to produce energy, so there is a lower percentage of O2 exhaled ($\bar{X}_{Ex} = 15.93\%$). The use of more O2 to produce more energy also results in the production of more CO2 ($\bar{X}_{Ex} = 4.66\%$).

The computer software uses metabolic formulas to correct

ventilation for temperature (room temperature if \dot{V}_I is measured), expired air temperature (if \dot{V}_E is measured), relative humidity (if \dot{V}_I is measured), and barometric pressure. Then using the expired percentage values of O₂ and CO₂, it calculates values for the volume of O₂ consumed ($\dot{V}O_2$ in L·min⁻¹ and $\dot{V}O_2$ in mL·kg⁻¹·min⁻¹), CO₂ produced ($\dot{V}CO_2$ in L·min⁻¹), and the ratio of the volume of CO₂ produced to the volume of O₂ consumed, known as the respiratory exchange ratio (RER).

Sometimes this relationship is designated simply as R, but this text will consistently use RER. The $\dot{V}O_2$ and $\dot{V}CO_2$ values are actual volumes of the gases that are used and produced by the body, respectively. The L·min⁻¹ unit represents the absolute amount of gas on a per-minute basis. The mL·kg⁻¹·min⁻¹ unit takes into account body size (body weight [BW] is given at the top of the table) and is therefore considered a relative value; it describes how many milliliters of gas are consumed (or produced) for each kilogram of BW each minute.

The absolute unit (L·min⁻¹) is highly influenced by body size, with large individuals showing the highest values. Therefore, it is most useful when comparing an individual to himself or herself under different conditions, such as before and after a training program, to determine whether a change in fitness has occurred. Use of the absolute unit is particularly important if the individual has lost weight, because the mL·kg⁻¹·min⁻¹ relative value goes up as weight goes down regardless of whether or not actual fitness has improved. The mL·kg⁻¹·min⁻¹ value thus, to an extent, equates individuals by factoring out the influence of body size. It is therefore typically used for comparisons between individuals.

Another unit used to express O₂ consumed and CO₂ produced on a relative basis is mL·kg FFB⁻¹·min⁻¹ or mL·kg LBM⁻¹·min⁻¹, where FFB stands for fat-free body and LBM stands for lean body mass. To calculate these units, the individual's percentage of body fat must be known and used to determine what portion of the total BW is fat free or lean. This unit is often used in comparisons between the sexes. However, it is not a very

practical measure because no one has yet figured how to avoid carrying one's fat during exercise.

At rest, the subject in the example in **Table 4.1** is using about $0.31 \text{ L}\cdot\text{min}^{-1}$, or $310 \text{ mL}\cdot\text{min}^{-1}$, of oxygen and producing about $0.25 \text{ L}\cdot\text{min}^{-1}$ of carbon dioxide. During exercise, the subject is using $1.28 \text{ L}\cdot\text{min}^{-1}$ of oxygen and producing $1.17 \text{ L}\cdot\text{min}^{-1}$ of carbon dioxide.

Short-term, Light- to Moderate-Intensity Submaximal Exercise

Look at minutes 1–8 in **Table 4.1**. During this light exercise, as anticipated, there is very little minute-to-minute variation in any of the variables after the first minute of adjustment (Robergs, 2014). A comparison of the individual's mean oxygen cost of these 8 minutes ($24.13 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) with her maximal oxygen consumption ($\dot{V}\text{O}_2 \text{ max}$) ($47.64 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) shows that she is working at approximately $50\% \dot{V}\text{O}_2 \text{ max}$.

When the exercise performed is at less than $70\% \dot{V}\text{O}_2 \text{ max}$ and the duration is 5–10 minutes, the oxygen consumption should level off and remain relatively constant for the duration of the work after the initial rise (**Figure 4.4A**). This condition is known as *steady-state* or *steady-rate* exercise. The time to achieve steady state varies from 1 to 3 minutes in youths and young adults at low and moderate levels of intensity, but it increases with higher-intensity exercise (Morgan et al., 1989b). The time to achieve steady state is longer in older untrained adults.

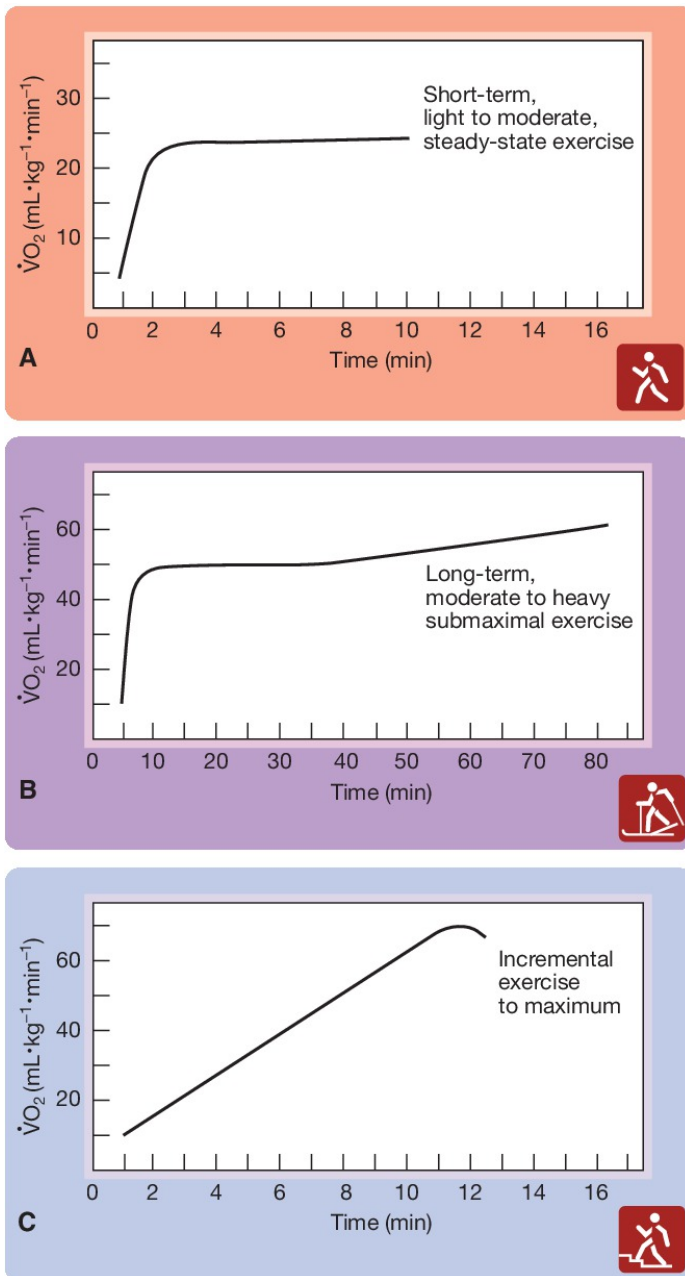


Figure 4.4 Oxygen Consumption Responses to Various Exercises.

A. Short-term, light to moderate, submaximal aerobic

exercise. B. Long-term, moderate to heavy, submaximal dynamic aerobic exercise. C. Incremental aerobic exercise to maximum.

Long-Term, Moderate to Heavy Submaximal Exercise

Now look carefully at minutes 33 through 37 in **Table 4.1**. Pay particular attention to the $\dot{V}O_2$ L·min⁻¹ and mL·kg⁻¹·min⁻¹ values. Despite the fact that the workload has not changed, a gradual increase occurs in these variables. When exercise is performed at a level greater than 70% $\dot{V}O_{2\text{ max}}$; or when exercise is performed at a lower percentage of $\dot{V}O_{2\text{ max}}$, as in this case, but for a long duration; or if the conditions are hot and humid, a phenomenon known as **oxygen drift** occurs. In oxygen drift, the oxygen consumption increases despite the fact that the oxygen requirement of the activity has not changed. A schematic representation of oxygen drift is presented in **Figure 4.4B**.

Oxygen Drift A situation that occurs in submaximal activity of long duration, or above 70% $\dot{V}O_{2\text{ max}}$, or in hot and humid conditions where the oxygen consumption increases, despite the fact that the oxygen requirement of the activity has not changed.

The oxygen consumption increases (drifts upward) because of rising blood levels of catecholamine hormones (epinephrine and norepinephrine), lactate accumulation (if the percentage of $\dot{V}O_2$ is high enough), shifting substrate utilization (to greater carbohydrate), increased cost of ventilation, and increased body temperature. Thus, although any given level of exercise typically requires a specific amount of oxygen, this amount has some individual and circumstantial variation (**Daniels, 1985**).

Incremental Aerobic Exercise to Maximum

Table 4.2 provides the results of a computer program for a constant work-rate transition incremental treadmill test to maximum. In this case, the subject was a very fit male senior exercise science major who regularly and successfully competed in long-distance running, cycling, and duathlon events. In an incremental exercise test such as this, the participant is asked to continue to the point of *volitional fatigue*, that is, until he is too tired to go on any longer. Throughout the test, the speed and/or grade is systematically raised so that the exercise becomes progressively harder. The protocol used in this particular test is called the modified Balke. For this test, the speed was kept constant at 3.5 mi·hr⁻¹ (94 m·min⁻¹). The grade was 0% for the first minute and 2% for the second minute, increasing 1% per minute thereafter. At the treadmill limit of 25% grade, speed was then increased 13.4 m·min⁻¹ each minute. Because the increments are small, the individual should be able to adjust to the load change in just 1 minute for most of the submaximal portion. Typically, the goal is for a maximal test to last 8–12 minutes. However, in apparently healthy individuals, the duration may be between 7 and 26 minutes for the cycle ergometer and between 5 and 26 minutes for the treadmill depending on stage length and treadmill grade with shorter tests requiring a sufficient warm-up (Midgley et al., 2008). Because of his very high fitness and ability to handle the steep slope, this individual was able to continue for 28 minutes.

TABLE 4.2 Aerobic Metabolic Responses during an Incremental Treadmill Test (Modified Balke Protocol)

| Energy System | Power | | Time | Capacity | |
|---|------------------------|----------------------|----------|-----------|-------------|
| | kcal·min ⁻¹ | kJ·min ⁻¹ | | kcal | kJ |
| ATP-PC (phosphagen) | 72 | 300 | :09–:10 | 11 | 45 |
| LA (anaerobic glycolysis) | 36 | 150 | 1:20 | 48 | 200 |
| O ₂ (aerobic glycolysis + Krebs cycle +ETS/OP; fuel = CHO) | 7.2–19.1 | 30–80 | 2:21:00* | 359–1,268 | 1,500–5,300 |

Because the work is different, the ventilation, and $\dot{V}\text{CO}_2$ responses are also different from those of the submaximal steady-state exercise in **Table 4.1**. In the incremental task, ventilation (\dot{V}_E), and $\dot{V}\text{CO}_2$ values all increase as a result of the increasing demands for and production of energy. For this particular

individual, the volume of expired air (\dot{V}_E STPD) rose from 25.21 L·min⁻¹ at minute 2 to 139.04 L·min⁻¹ at minute 28. This demonstrates the reserve capacity in the ventilatory system. Oxygen consumption increased from slightly over 1 L·min⁻¹ (or almost 17 mL·kg⁻¹·min⁻¹) to just over 5 L·min⁻¹ at maximum. At this point, the individual was producing as much ATP as he could aerobically.

The highest amount of oxygen an individual can take in, transport, and utilize to produce ATP aerobically while breathing air during heavy exercise is called **maximal oxygen consumption** ($\dot{V}O_{2\text{ max}}$). The exercise tester has to decide whether any given test truly is a maximal effort before labeling the highest $\dot{V}O_2$ value as maximal. Several physiological criteria have traditionally been used to determine whether the test is a maximal test (ACSM, 2022):

Maximal Oxygen Consumption ($\dot{V}O_{2\text{ max}}$) The highest amount of oxygen an individual can take in, transport and utilize to produce ATP aerobically while breathing air during heavy exercise.

- (a) an RER (which is described fully later in this chapter) of ≥ 1.1
- (b) a heart rate ± 10 b·min⁻¹ of predicted maximal heart rate (220 minus age)
- (c) a plateau in oxygen consumption (< 2 mL increase in oxygen consumption with an increase in workload)
- (d) a rating of perceived exertion (RPE) of ≥ 18 on the Borg Scale

The plateau in oxygen consumption is considered to be the primary criterion for obtaining a true maximal test. The classic definition of a plateau is a rise of 2.1 mL·kg⁻¹·min⁻¹ or less, or a rise of 0.15 L·min⁻¹ or less, in oxygen consumption ($\dot{V}O_2$) with an increase in workload that represents a change in grade of

2.5% while running at $7 \text{ mi}\cdot\text{hr}^{-1}$ ($11.2 \text{ km}\cdot\text{hr}^{-1}$) with 3-minute stages (Taylor et al., 1955). Use of this criterion for all protocols, however, has been questioned (Howley et al., 1995). One alternative is to define a plateau as an increase of less than half the expected theoretical rise based on the change in speed, grade, or speed and grade (Plowman and Liu, 1999) or Watts for cycle ergometry (Wagner et al., 2020). The expected increase can be calculated using the American College of Sports Medicine (ACSM) (2022) equations provided in Appendix B. In the treadmill example above, the expected difference in oxygen consumption between the last 2 minutes (27 and 28) is $5.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

The secondary criteria have come under a great deal of scrutiny recently based in part on large variability in HRmax, RER and blood lactate responses, the failure of these criteria to be applicable to children and the elderly, and the fact that these

criteria do not differentiate individuals who demonstrate a $\dot{\text{V}}\text{O}_2$ plateau from those who do not. This has led to recommendation

that a verification phase be included in $\dot{\text{V}}\text{O}_2 \text{ max}$ testing. A verification phase consists of some variation of a short constant speed run to exhaustion performed after the incremental phase of the max test and a short recovery period. It is highly dependent on the participants' motivation. A peak oxygen uptake in the

verification phase that is similar (within $\sim 2\%$ or the $\dot{\text{V}}\text{O}_2 \text{ max}$ value attained in the incremental phase) would indicate that a

true $\dot{\text{V}}\text{O}_2 \text{ max}$ had been elicited. A heart rate max within 4 bpm between the two tests would indicate a consistent maximal effort. Research continues to determine the best protocol for verification tests and criterion for the primary and secondary variables that indicate a true max test as well as whether a plateau is a prerequisite for being able to measure a true

$\dot{\text{V}}\text{O}_2 \text{ max}$ (Howley, 2007; Mann et al., 2013; Midgley et al., 2007, 2009; Mier et al., 2012; Nolan et al., 2014; Snell et al., 2007; Wood et al., 2010). Complete the Check Your Comprehension 1 to apply these criteria.

CHECK YOUR COMPREHENSION 1—CASE STUDY 1

Did the individual in **Table 4.2** meet the HR and criteria for a true maximal test? Include the following results from his verification run in your discussion:

$$\dot{V}O_2 \text{ max} = 75.97 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{Smin}^{-1}; \quad \text{RER} = 1.20;$$
$$\text{HRmax} = 197 \text{ b} \cdot \text{min}^{-1}$$

Check your answer in [Appendix C](#).

The rectilinear increase in $\dot{V}O_2$ obtained during an incremental test can be generalized to all healthy individuals, both male and female, young and old, and those who are high and low in fitness. This pattern is schematically represented in **Figure 4.4C**. When a ramp protocol is used, that is, the work rate is increased in a linear and continuous fashion rather than in discrete incremental timed steps, minor positive or negative deviations from strict linearity may or may not occur above VT2 ([Boone and Bourgois, 2012](#)). However, a plateau is not always evident, especially in children and the elderly. When no plateau is achieved, the term *peak oxygen consumption* ($\dot{V}O_{2\text{peak}}$) to describe the highest value attained is more accurate than the term maximal oxygen consumption ($\dot{V}O_{2\text{max}}$).

As a result of the increasing ATP production, the amount of CO₂ produced also increases. Note in **Table 4.2** that the amount of CO₂ produced ($\dot{V}CO_2$) is generally not equal to the amount of O₂ consumed ($\dot{V}O_2$). Note also that despite the incremental nature of this test, the O₂% and CO₂% do not vary much. There is a slight decline in O₂% and a parallel rise in CO₂% after the first 10 minutes and then relatively steady values until the last 2 minutes, when the O₂% goes back up and the CO₂% goes back down just a little. Frequently, as a subject nears maximal exertion, the O₂% may rise to 17% and the CO₂% may drop to 3%. These small percentage changes, especially when they occur in the last couple of minutes, are compensated for by the increasingly larger volumes of air being ventilated.

Static and Dynamic Resistance Exercise

Physiological responses to static exercise are generally described in relation to the percentage of maximal voluntary contraction (MVC) at which they take place. Depending on the muscle group used, static contractions below 15–25% MVC do not fully occlude blood flow, such that oxygen can still be delivered to working muscles. At such loads, however, little extra energy above the resting level is required, and oxygen consumption increases minimally, perhaps as little as 50 mL·min⁻¹, but for as long as half an hour (Asmussen, 1981; Shepard et al., 1981).

The higher the percentage of MVC of the static contraction, the greater the intramuscular pressure is, the more likely it is that the blood flow will be completely arrested or occluded, and the shorter the time that the contraction can be maintained. If occlusion of blood flow is not complete, oxygen consumption increases. This increased oxygen consumption is higher than the amount at lower percentages of MVC but still far below the rise in values that occurs during the aerobic endurance exercises previously described.

In addition to a lower oxygen consumption, there is one other major difference between static and dynamic-endurance exercise. At the cessation of static exercise, when blood flow is fully restored, a sudden increase in oxygen consumption occurs (**Figure 4.5**) before the slow, gradual decline of the typical EPOC curve begins (Asmussen, 1981; Shepard et al., 1981).

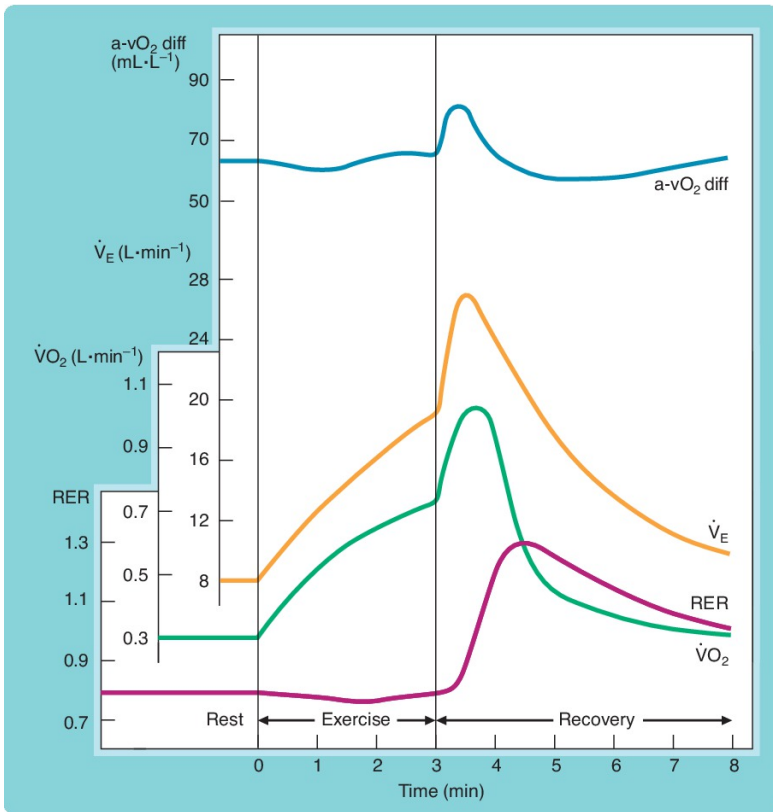


Figure 4.5 Respiratory and Metabolic Responses to Heavy Static Exercise.

Heavy static exercise causes small respiratory (a-vO₂diff and) and metabolic ($\dot{V}O_2$ and RER) responses during the actual contraction. However, each of these variables shows an increased rebound effect immediately upon cessation of the exercise before slowly returning to preexercise values.

Source: Reprinted with permission from Inbar, O., & O. Bar-Or: Anaerobic characteristics in male children and adolescents. *Medicine and Science in Sports and Exercise*.

The primary source of energy for dynamic resistance activity such as weight lifting or wrestling is anaerobic (Fleck and Kraemer, 1987; Fox and Mathews, 1974). In part, anaerobic energy is used because dynamic resistance exercise has a static component. In part, anaerobic energy is used due to the high intensity and short duration of the exercise. Despite the predominance of anaerobic energy sources, dynamic resistance activity has an aerobic component as well. The more the repetitions and the longer the duration of the sets in a weightlifting workout, the greater is the aerobic contribution.

$\dot{V}O_2$ has been shown to exhibit a linear relationship to mean concentric power as well as total concentric work in the chest press (Buitrago et al., 2014). Actual values are not available for different routines because such activities are intended for anaerobic, not aerobic, benefits. However, an example of the oxygen contribution to five sets of 6–12 repetitions per set of supine leg press exercise can be seen in **Figure 4.6** (Tesch et al., 1990). In this group of untrained males, a gradual rise in oxygen consumption occurs over the first three sets, at which point the oxygen consumption basically stabilizes for the remaining two sets. The oxygen cost represents approximately 33–47% of the average $\dot{V}O_{2\text{ max}}$ for these subjects. Thus, the aerobic contribution during weightlifting exercise can be expected to be somewhat less than the oxygen costs of most aerobic endurance activities but higher than the cost of purely static exercise (Tesch et al., 1990).

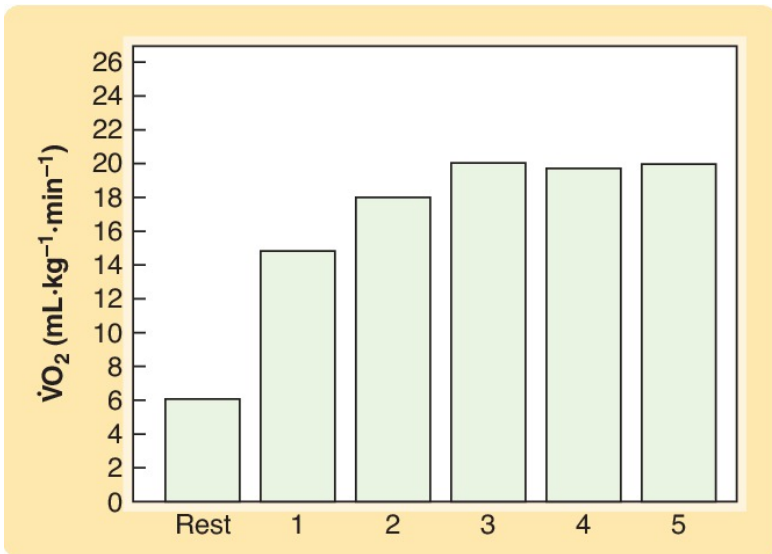


Figure 4.6 Aerobic Contribution to Dynamic Resistance Exercise.

Oxygen consumption was measured before and during five sets of 6–12 repetitions of supine leg presses. The values represent the combined results of two groups: one performed only the concentric (lifting) phase and the other performed both the concentric and eccentric (lowering) phases. The addition of the eccentric phase represented such a low additional energy cost just above the concentric energy expenditure that it was not separated out. **Source:** Based on [Tesch et al. \(1990\)](#).

Very-Short-Term, High-Intensity Anaerobic Exercise

As stated in [Chapter 3](#), historically the contribution of aerobic metabolism to short, intense exercise has probably been

underestimated because of the difficulty of obtaining accurate measurements. However, even the most widely used anaerobic test, the 30-second Wingate Anaerobic Test, has an aerobic energy contribution of at least 20%.

The Oxygen Cost of Breathing

Part of the oxygen used both at rest and during exercise goes to support the respiratory muscles. This value does not remain constant but varies with the intensity of activity. During rest, the respiratory system uses about 1–2% of the total body oxygen consumption, or $2.5 \text{ mL} \cdot \text{min}^{-1}$ of oxygen. The oxygen cost of ventilation is higher in children than in middle-aged or older adults (Bar-Or, 1983; Pardy et al., 1984).

During light to moderate submaximal dynamic aerobic exercise, where \dot{V}_E is less than $60 \text{ L} \cdot \text{min}^{-1}$, the respiratory oxygen cost changes to about $25\text{--}100 \text{ mL} \cdot \text{min}^{-1}$, $1.8 \text{ mL } \dot{V}\text{O}_{2L} \cdot \text{L}^{-1} \dot{V}_E \cdot \text{min}^{-1}$, or 3–5% of total body $\dot{V}\text{O}_2$. At heavy submaximal exercise, when \dot{V}_E is between 60 and $120 \text{ L} \cdot \text{min}^{-1}$, respiratory oxygen use may rise to $50\text{--}400 \text{ mL} \cdot \text{min}^{-1}$. During incremental exercise to maximum, the initial \dot{V}_E during the lower exercise stages shows a very gradual curvilinear rise, reflecting the submaximal changes described previously. At workloads above those requiring a \dot{V}_E greater than $120 \text{ L} \cdot \text{min}^{-1}$, a dramatic exponential curve occurs. In this curve, by the time a \dot{V}_E of $180 \text{ L} \cdot \text{min}^{-1}$ is achieved in a very fit individual, $1,000\text{--}1,300 \text{ mL} \cdot \text{min}^{-1}$, approximately $2.9 \text{ mL } \dot{V}\text{O}_{2L} \cdot \text{L}^{-1} \dot{V}_E \cdot \text{min}^{-1}$ or 8–10% (untrained) to 13–16% (trained) of total body oxygen is used simply to support respiration (Aaron et al., 1992a, 1992b; Pardy et al., 1984). The oxygen cost of breathing is higher in both younger and older females than in males (Dominelli et al., 2015; Topin et al., 2003).

Theoretically, there may be a maximal level of ventilation above which any further increase in oxygen consumption would

be used entirely by the ventilatory musculature, thus limiting maximal exercise (Otis, 1954; Pardy et al., 1984; Vella et al., 2006). At what precise point this critical level of ventilation occurs is unknown. However, even if a critical ventilation level does not exist, respiration does utilize a significant portion of the $\dot{V}O_2$ during heavy exercise (Shephard, 1966; Vella et al., 2006). In old age, the higher oxygen cost of breathing may be a significant factor in limiting exercise performance (Shepard et al., 1981).

Smoking increases the oxygen cost of respiration during exercise. However, an abstinence of even 1 day can substantially reduce this effect of cigarette smoking (Rode and Shephard, 1971; Shepard et al., 1981).

Respiratory Quotient/Respiratory Exchange Ratio

When the $\dot{V}O_2$ consumed and $\dot{V}CO_2$ produced during exercise are known, much useful information can be derived. One such derived variable—**RER** or **respiratory exchange ratio**—is given in both [Tables 4.1](#) and [4.2](#).

Respiratory Exchange Ratio (RER) Ratio of the volume of CO_2 produced divided by the volume of O_2 consumed in the body as a whole.

The RER reflects on a total body level what is happening at the cellular level. The ratio of the amount of CO_2 produced to the amount of O_2 consumed at the cellular level is termed the **respiratory quotient (RQ)**. The formula is

Respiratory Quotient (RQ) Ratio of the amount of carbon dioxide produced divided by the amount of oxygen consumed at cellular level.

respiratory quotient = carbon dioxide produced
(molecules) ÷ oxygen consumed (molecules)

or

$$4.3 \quad RQ = \frac{CO_2}{O_2}$$

This formula may also be computed using L·g⁻¹ in place of molecules.

The values of carbon dioxide produced and oxygen consumed are known for the oxidation of carbohydrate, fat, and protein, both on a cellular level and in absolute amounts (L·g⁻¹). The latter values are presented in **Table 4.3**. The amount of oxygen consumed and the amount of carbon dioxide produced vary among the major fuel sources because of the differences in their chemical composition. The following examples compute the cellular level RQ for each major fuel source both per mole of a specific substrate (the carbohydrate, glucose, C₆H₁₂O₆; the fat, palmitic acid, C₁₆H₃₂O₂; and the protein, albumin, C₇₂H₁₁₂N₂O₂₂S) and per gram of carbohydrate (glucose), fat (fatty acid), total protein, and branched chain amino acids. In each case, the oxygen used (left side of the equation) and carbon dioxide produced (right side of the equation) are inserted into Equation 4.3 to determine the RQ. The values of 1.0 for carbohydrate, 0.7 for fat, and 0.81 for protein calculated in the example are the classic accepted values for RQ.

TABLE 4.3 Energy Production from Carbohydrate, Fat, and Protein

| | Carbohydrate | | Protein | | Fat |
|---|---|---|---|---|---|
| Primary utilization in exercise | High-intensity, short-duration exercise | | Ultradistance exercise and glucose precursor | | Low-intensity, long-duration exercise and glucose precursor |
| Form in which utilized by muscles | Glucose | Glycogen | * | Branched chain amino acids | Fatty acids |
| Oxygen needed to utilize per gram (L·g ⁻¹) | 0.75 L·g ⁻¹ | 0.83 L·g ⁻¹ | 0.965 L·g ⁻¹ | 1.24 L·g ⁻¹ | 2.02 L·g ⁻¹ |
| Energy produced per gram (kcal·g ⁻¹) | 3.75 kcal·g ⁻¹ 15.68 kJ·g ⁻¹ | 4.17 kcal·g ⁻¹ 17.43 kJ·g ⁻¹ | 4.3 kcal·g ⁻¹ 17.97 kJ·g ⁻¹ | 3.76 kcal·g ⁻¹ 18.09 kJ·g ⁻¹ | 9.3 kcal·g ⁻¹ 38.87 kJ·g ⁻¹ |
| Energy produced per liter of oxygen [†] (kcal·L ⁻¹ O ₂) | 5.03 kcal·L ⁻¹ O ₂ 21.03 kJ·L ⁻¹ O ₂ | 5.03 kcal·L ⁻¹ O ₂ 21.03 kJ·L ⁻¹ O ₂ | 4.46 kcal·L ⁻¹ O ₂ 18.64 kJ·L ⁻¹ O ₂ | 3.03 kcal·L ⁻¹ O ₂ 12.67 kJ·L ⁻¹ O ₂ | 4.61 kcal·L ⁻¹ O ₂ 19.27 kJ·L ⁻¹ O ₂ |
| Carbon dioxide produced (L·g ⁻¹) | 0.75 L·g ⁻¹ | 0.83 L·g ⁻¹ | 0.781 L·g ⁻¹ | 0.92 L·g ⁻¹ | 1.43 L·g ⁻¹ |

*Values for protein are given. However, the BCAA values are

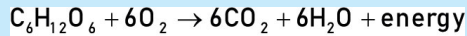
more realistic for muscle activity per 1 g PRO = 1.17 g AA (Morgan et al., 1989b; Péronnet et al., 1987).

†Energy produced per liter of oxygen equals the caloric equivalent.

Example

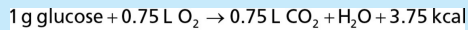
For carbohydrates:

Glucose (per mole)



$$\text{RQ} = \frac{6\text{CO}_2}{6\text{O}_2} = 1.0$$

Glucose (per gram, from **Table 4.3**)



$$\text{RQ} = \frac{0.75 \text{ L CO}_2}{0.75 \text{ L O}_2} = 1.0$$

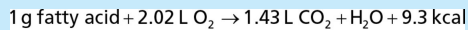
For fat:

Palmitic acid (per mole)



$$\text{RQ} = \frac{16\text{CO}_2}{23\text{O}_2} = 0.7$$

Fatty acids (per gram, from **Table 4.3**)



$$\text{RQ} = \frac{1.43 \text{ L CO}_2}{2.02 \text{ L O}_2} = 0.71$$

For protein:

Albumin (per mole)

$$\text{C}_7\text{H}_{112}\text{N}_2\text{O}_{22}\text{S} + 77\text{O}_2 \rightarrow 63\text{CO}_2 + 38\text{H}_2\text{O} + \text{SO}_3 + 9\text{CO}(\text{NH}_2)(\text{urea})$$

$$\text{RQ} = \frac{63\text{CO}_2}{77\text{O}_2} = 0.82$$

Protein (per gram, from **Table 4.3**)

$$1 \text{ g protein} + 0.965 \text{ L O}_2 \rightarrow 0.781 \text{ L CO}_2 + \text{H}_2\text{O} + 4.3 \text{ kcal}$$

$$\text{RQ} = \frac{0.781 \text{ L CO}_2}{0.965 \text{ L O}_2} = 0.81$$

Note that if the branched chain amino acids are used, the RQ would be lower:

$$1 \text{ g BCAA} + 1.24 \text{ L O}_2 \rightarrow 0.92 \text{ L CO}_2 + \text{H}_2\text{O} + 3.76 \text{ kcal}$$

$$\text{RQ} = \frac{0.92 \text{ L CO}_2}{1.24 \text{ L O}_2} = 0.74$$

Because these values for RQ have been derived from the cellular oxidation of specific foodstuffs, knowing the RQ allows one to estimate the fuels utilized in different activities. However, because individuals seldom use only one fuel, these “classic values” are rarely seen. Nonetheless, in a resting individual, an RQ of 0.93 indicates a high reliance on carbohydrate, and an RQ of 0.75 indicates a high reliance on fat. An RQ of 0.82 indicates either a fasting individual burning protein (usually from muscle mass, as in a starvation situation) or, more likely, an individual using a normal mixed diet of all three fuels. Remember that protein is not normally used as a major fuel source, especially at rest.

The ability to transition from relying primarily on fat as a fuel for ATP production to carbohydrates use for ATP production as exercise intensity increases is referred to as **metabolic flexibility**. Some individuals, such as people with metabolic syndrome, are considered metabolically inflexible; that is, they have an impaired ability to use fat for energy. Thus, even at low exercise intensities, they have an increased reliance on carbohydrates for energy. Alternatively, as one becomes more aerobically trained, metabolic flexibility improves, and the individuals have a greater ability to use fat for energy at higher

intensities leading to a decreased reliance on carbohydrates for energy production (Kim et al., 2018; San-Millán and Brooks, 2018).

Metabolic Flexibility The ability to transition from relying primarily on fat use for ATP production to carbohydrates use for ATP production as exercise intensity increases.

Although the example interpretations are acceptable for resting individuals, there are several difficulties interpreting the RQ during exercise. In the first place, the O₂ and CO₂ values measured in open-circuit indirect spirometry are ventilatory measures that indicate total body gas exchange and not just working muscle. Second, hyperventilation from any cause will result in an excess of CO₂ being exhaled, thus falsely elevating the ratio. This often occurs in stress situations such as in anticipation of an exercise test or in early recovery from maximal work. Third, if exercise is of a high enough intensity to involve anaerobic metabolism, causing an increase in acidity (a decrease in pH) and a concomitant rise in nonmetabolic CO₂ release, RQ no longer represents just fuel utilization. Values during exercise, especially as an individual approaches maximal effort, usually exceed 1.0. In this case, it is assumed that the fuel source is carbohydrate and the excess CO₂ is a result of anaerobic metabolism. Conversely, after an initial increase during recovery, CO₂ is retained, causing low values (Newsholme and Leech, 1983). For these reasons, the term RER is more accurate than RQ

to describe the ratio of $\dot{V}\text{CO}_2$ produced to $\dot{V}\text{O}_2$ consumed when determined by open-circuit spirometry.

The involvement of anaerobic metabolism that results in RER values greater than 1.0 allows for use of RER as a criterion to determine whether an exercise test is truly maximal. As indicated earlier, the criterion for a true maximal test is an RER greater than 1.1 or at least 1.0, with the lower value predominating for children/adolescents and older adults (Holly, 1988; MacDougall et al., 1982).

Although RER is a more accurate description than RQ, RER

values still do not distinguish between different forms of a fuel, such as glucose or glycogen, fatty acids, or ketone bodies. Also, when only ventilatory CO₂ and O₂ are measured, there is no indication of protein utilization. To measure protein utilization, the amount of nitrogen excreted (in urine and sweat) must be measured. This task is at best cumbersome and at worst almost impossible to perform in exercise situations (Bursztein et al., 1989; Consolazio et al., 1963). Thus, the RER that is measured by

the $\frac{\dot{V}\text{CO}_2 \text{ L}\cdot\text{min}^{-1}}{\dot{V}\text{O}_2 \text{ L}\cdot\text{min}^{-1}}$ during exercise is a *nonprotein RER*. Again, because protein is not thought to be utilized as fuel until long-duration activity is in progress, this simplification is not deemed to materially affect the relative percentage of carbohydrate and fat utilization in most situations.

Table 4.4 presents the relative percentages of calories used from carbohydrate and fat for all RER values between 0.7 and 1.0 (Carpenter, 1921). Referring to **Table 4.1**, we see that, during rest, the individual had an RER of 0.81. From **Table 4.4**, this value indicates that 35.4% of her fuel was carbohydrate and 64.6% fat. During her 8 minutes of steady-state work, the RER averaged 0.90, indicating a major shift in fuel supply, with 66% of the fuel being carbohydrate and 34% fat.







TABLE 4.4 Percentage of Calories from Carbohydrate (CHO) and Fat and the Caloric Equivalents for Nonprotein RER Values for Each Liter of Oxygen Used

| RER | CHO% | Fat% | Caloric Equivalent (kcal·L ⁻¹ O ₂) | RER | CHO% | Fat% | Caloric Equivalent (kcal·L ⁻¹ O ₂) |
|------|------|-------|--|------|-------|------|--|
| 0.70 | 0.0 | 100.0 | 4.686 | 0.86 | 52.4 | 47.6 | 4.875 |
| 0.71 | 1.4 | 98.6 | 4.690 | 0.87 | 55.8 | 44.2 | 4.887 |
| 0.72 | 4.8 | 95.2 | 4.702 | 0.88 | 59.2 | 40.8 | 4.899 |
| 0.73 | 8.2 | 91.8 | 4.714 | 0.89 | 62.6 | 37.4 | 4.911 |
| 0.74 | 11.6 | 88.4 | 4.727 | 0.90 | 66.0 | 34.0 | 4.924 |
| 0.75 | 15.0 | 85.0 | 4.739 | 0.91 | 69.4 | 30.6 | 4.936 |
| 0.76 | 18.4 | 81.6 | 4.751 | 0.92 | 72.8 | 27.2 | 4.949 |
| 0.77 | 21.8 | 78.2 | 4.764 | 0.93 | 76.2 | 23.8 | 4.961 |
| 0.78 | 25.2 | 74.8 | 4.776 | 0.94 | 79.6 | 20.4 | 4.973 |
| 0.79 | 28.6 | 71.4 | 4.788 | 0.95 | 83.0 | 17.0 | 4.985 |
| 0.80 | 32.0 | 68.0 | 4.801 | 0.96 | 86.4 | 13.6 | 4.998 |
| 0.81 | 35.4 | 64.6 | 4.813 | 0.97 | 89.8 | 10.2 | 5.010 |
| 0.82 | 38.8 | 61.2 | 4.825 | 0.98 | 93.2 | 6.8 | 5.022 |
| 0.83 | 42.2 | 57.8 | 4.838 | 0.99 | 96.6 | 3.4 | 5.035 |
| 0.84 | 45.6 | 54.4 | 4.850 | 1.00 | 100.0 | 0.0 | 5.047 |
| 0.85 | 49.0 | 51.0 | 4.862 | | | | |

Source: Modified from [Carpenter \(1921\)](#).

During the last 5-minute interval (minutes 33–37 in [Table 4.1](#)), an even greater reliance on carbohydrate occurs with approximately 90% of the energy used being supplied by carbohydrate sources. If submaximal exercise were to continue for another 2 hours or more, the RER values would drop back down, indicating a depletion of available carbohydrate fuel stores. How low the RER values would go depends on the amount of carbohydrate originally stored as well as the intensity and duration of the activity. For this reason, athletes often try to carbohydrate load before endurance events so that carbohydrate stores are initially high and will last longer. Carbohydrate loading is fully discussed in [Chapter 6](#). [Table 4.5](#) summarizes the oxygen consumption and RER/energy substrate responses to various categories of exercise. Check your knowledge by completing the [Check Your Comprehension 2](#).

TABLE 4.5 Aerobic Exercise Response

| | | O ₂ Consumption | RER/Energy Substrate |
|---|---|--|---|
| Short-term, light to moderate submaximal exercise |  | Initial rise; plateau at appropriate steady state | 0.85 to 0.90/mixed fat and CHO to predominantly CHO |
| Short-term, moderate to heavy submaximal exercise | | Initial rise; plateau at appropriate steady state | 0.85 to 0.90 to 1.0+/mixed fat and CHO to CHO |
| Long-term, moderate to heavy submaximal exercise |  | Initial rise; plateau at steady state; positive drift | 0.85 to 0.90 to 1.0+ to 0.90 to 0.85/mixed fat and CHO to CHO; if duration is long enough, RER will decrease as CHO supplies are depleted |
| Very-short-term, high-intensity, anaerobic exercise |  | Small increase; provides ~30% or less of energy cost | 0.90 to 1.0+/predominantly CHO to all CHO |
| Incremental exercise to maximum |  | Rectilinear rise; plateau at maximum | 0.85 to ≥1.0/mixed fat and CHO to glycogen |
| Static exercise |  | Small gradual rise during exercise; rebound rise in recovery | 0.80 to 1.1+/mixed fat and CHO during exercise with rebound rise in recovery/ glycogen |
| Dynamic resistance exercise |  | Small gradual rise during exercise; the lighter the load and the higher the reps, the greater the contribution | 0.90 to 1.0+/glycogen |

CHECK YOUR COMPREHENSION 2

Using [Table 4.2](#), determine the approximate percentages of carbohydrate and fat used at minutes 2, 14, and 28. Check your answer in [Appendix C](#) to see whether you are correct. The early minute RER values are higher than expected for the workload, probably from hyperventilation in anticipation of the maximal

effort to come in the exercise test. The last three RER values listed in **Table 4.2** are greater than 1.1 and indicate the involvement of anaerobic metabolism.

Estimation of Caloric Expenditure

Table 4.3 not only shows the oxygen consumed and carbon dioxide produced when each of the energy substrates is utilized but also indicates the potential energy in terms of kilocalories per gram ($\text{kcal}\cdot\text{g}^{-1}$) or kilocalories per liter of oxygen ($\text{kcal}\cdot\text{L}^{-1}\text{O}_2$) for each substrate. The $\text{kcal}\cdot\text{L}^{-1}\text{O}_2$ figures show that carbohydrates are most efficient in the use of oxygen to provide energy, followed by fat, and finally by protein—although the substrates do not really vary a great deal.

The potential energy for carbohydrate and for protein depends on whether the form is glucose ($3.75\text{ kcal}\cdot\text{g}^{-1}$) or glycogen ($4.17\text{ kcal}\cdot\text{g}^{-1}$) or all amino acids ($4.3\text{ kcal}\cdot\text{g}^{-1}$) or just the branched chain amino acids ($3.76\text{ kcal}\cdot\text{g}^{-1}$). When food is ingested, these distinctions cannot be made; the net average energy values are rounded to the whole numbers of $4\text{ kcal}\cdot\text{g}^{-1}$ for carbohydrate and protein and $9\text{ kcal}\cdot\text{g}^{-1}$ for fat. These values are called *Atwater factors* and are used to represent the energy potential of

food. From the known values of $\dot{V}\text{O}_2$ $\text{L}\cdot\text{min}^{-1}$ and RER, it is possible to compute the kilocalorie (or kilojoule) energy expenditure.

Table 4.4 includes the **caloric equivalent**—the number of kilocalories produced per liter of oxygen consumed—for all values of RER between 0.7 and 1.0. The caloric equivalent varies from $4.686\text{ kcal}\cdot\text{L}^{-1}\text{O}_2$ at an RER of 0.7 to $5.047\text{ kcal}\cdot\text{L}^{-1}\text{O}_2$ at an RER of 1.0. If the amount of oxygen consumed and the caloric equivalent are known, the caloric cost of an activity can be computed.

Caloric Equivalent The number of kilocalories produced per liter of oxygen consumed.

To compute the $\text{kcal}\cdot\text{min}^{-1}$ cost, first find the caloric equivalent for the RER in **Table 4.4**. The rest of the computation is simply a matter of multiplying the oxygen cost by the caloric equivalent. The formula is

$$\text{caloric cost of an activity } (\text{kcal}\cdot\text{min}^{-1}) = \text{oxygen consumed } (\text{L}\cdot\text{min}^{-1}) \times \text{caloric equivalent } (\text{kcal}\cdot\text{L}^{-1}\text{O}_2)$$

Example

Assume that an individual had an RER of 0.91 during exercise that used $2.15 \text{ L O}_2\cdot\text{min}^{-1}$. The caloric equivalent for an RER of 0.91 is $4.936 \text{ kcal}\cdot\text{L}^{-1} \text{ O}_2$, so the calculation becomes

$$2.15 \text{ L O}_2\cdot\text{min}^{-1} \times 4.936 \text{ kcal}\cdot\text{L}^{-1} \text{ O}_2 = 10.61 \text{ kcal}\cdot\text{min}^{-1}$$

This **caloric cost** is the energy expenditure of the activity. It may be expressed in calories or joules per minute ($\text{kcal}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{min}^{-1}$), relative to BW ($\text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), or as total calories if the calories per minute is multiplied by the total number of minutes of participation.

Caloric Cost Energy expenditure of an activity performed for a specified period of time. It may be expressed as total calories (kcal), calories or kilojoules per minute ($\text{kcal}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{min}^{-1}$), or relative to body weight ($\text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$).

If you know the oxygen consumed during any activity but do not know the RER, you can estimate the caloric value of that activity by multiplying by $5.0 \text{ kcal}\cdot\text{L}^{-1} \text{ O}_2$. The $5.0 \text{ kcal}\cdot\text{L}^{-1} \text{ O}_2$ value is very close to the average caloric equivalent and is easy to

remember. Since $1 \text{ kcal} = 4.18 \text{ kJ}$, to convert $\text{kcal}\cdot\text{min}^{-1}$ to $\text{kJ}\cdot\text{min}^{-1}$, multiply by 4.18. For the same example as above, this is

$$10.61 \text{ kcal}\cdot\text{min}^{-1} \times 4.18 \text{ kJ}\cdot\text{kcal}^{-1} = 44.36 \text{ kJ}\cdot\text{min}^{-1}$$

Remember that this caloric cost is an estimate of the aerobic portion only. If we attempt to calculate the caloric cost of all 28 minutes of the incremental task, the result will be an underestimation because the anaerobic energy expenditure cannot be calculated. Furthermore, these values include what an individual would expend if he or she were resting quietly. *Gross energy expenditure* is the term used when resting energy expenditure is included. If resting energy expenditure is subtracted from gross energy expenditure, the *net energy expenditure*—the energy expended to do the exercise itself—is the result. Knowing the caloric costs of activities is helpful when prescribing exercise.

Complete the [Check Your Comprehensions 3](#) and [4](#) (case study 2) and check your answers in [Appendix C](#).

CHECK YOUR COMPREHENSION 3

Determine the caloric cost (in kilocalories and kilojoules) for minute 14 of [Table 4.2](#). Check your answer in [Appendix C](#).

CHECK YOUR COMPREHENSION 4—CASE STUDY 2

A reasonable and beneficial level of exercise for fat loss is 300 kcal per session ([Åstrand, 1952](#)). The oxygen cost of riding a bicycle ergometer at 2 kp ($600 \text{ kg}\cdot\text{min}^{-1}$, or 100 W) at $60 \text{ rev}\cdot\text{min}^{-1}$ is $1,200 \text{ mL O}_2\cdot\text{min}^{-1}$ above resting metabolism. At this rate, how long must an individual ride to burn an excess of 300 kcal? Ignore the cost involved in a warm-up or cooldown. Check your answer in [Appendix C](#).

The Metabolic Equivalent

Although most people think of energy cost in terms of kilocalories (kcal), exercise physiologists and physicians often use metabolic equivalent (MET) values. MET is an acronym derived from the term *Metabolic Equivalent*. One MET represents the average, seated, resting energy cost of an adult and is set at $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of oxygen, or $1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$.

In reality, resting metabolic rates (RMR) vary among individuals. A 2014 examination of 197 studies reporting resting metabolic rate found that in the six earliest studies, the RMR was indeed $1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$. However, the overall values were closer to $0.89 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ or $3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for men and $0.84 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ or $2.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for women. This is an overestimation of 10–15%, respectively, and can be higher depending on age, sex, body composition, and training status (McMurray et al., 2014). If an individual's resting, seated energy expenditure is known, that value can, and should, be used instead of the $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ average. Otherwise, in practice, the only current option is to use the traditional $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of oxygen, or $1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$, with the expectation that should further research confirm the findings of this meta-analysis, changes will be made in the calculated MET levels of activity. For now, it is probably best to assume that, if anything, the published MET levels will be higher than the actual MET levels for all but young (20–29 years), normal weight males and adjust individual exercise prescriptions or physical activity recommendations accordingly.

Multiples of the 1-MET resting baseline represent the MET level or multiples of the resting rate of oxygen consumption of any given activity. Thus, an activity performed at the level of 5 METs would require five times as much energy as is expended at rest ($5 \times 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} = 17.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$).

MET A unit that represents the MET in multiples of the resting rate of oxygen consumption of any given activity.

To calculate MET levels from measures of oxygen

consumption, divide the amount of oxygen utilized (in $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) by 3.5. For example, if an individual expends $29 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of O_2 on a task, the MET level is $29 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \div 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} = 8.3 \text{ METs}$.

To convert from MET to $\text{kcal}\cdot\text{min}^{-1}$, it is necessary to know the individual's BW and use the relationship $1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} = 1 \text{ MET}$. For example, if the 8.3-MET activity is done by a female of average weight (68 kg), the calculation is

$$8.3 \text{ METs} = \frac{(8.3 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}) \times 68 \text{ kg}}{60 \text{ min}\cdot\text{hr}^{-1}} = 9.4 \text{ kcal}\cdot\text{min}^{-1}$$

Alternately, the formula

$$\text{kcal}\cdot\text{min}^{-1} = \left[\frac{\text{METs} \times 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \times \text{bodyweight in kg}}{1,000 \text{ mL}\cdot\text{L}^{-1}} \right] \times 5 \text{ kcal}\cdot\text{L}^{-1}$$

simplified as $(\text{METs} \times 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \times \text{body weight in kg}) \div 200 \text{ kcal}\cdot\text{mL}^{-1}$; where $200 \text{ kcal}\cdot\text{mL}^{-1}$ is $1,000 \text{ mL}\cdot\text{L}^{-1} \div 5 \text{ kcal}\cdot\text{L}^{-1}$ (Swain, 2010), may be used.

The example then becomes

$$\left[\frac{8.3 \text{ METs} \times 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \times 68 \text{ kg}}{1,000 \text{ mL}\cdot\text{L}^{-1}} \right] \times 5 \text{ kcal}\cdot\text{L}^{-1} = 9.88 \text{ kcal}\cdot\text{min}^{-1}$$

FOCUS ON APPLICATION

Caloric Cost and Exercise Machines

After studying exercise physiology for hours, to take a break, you go to the campus recreation center to exercise. Your goal is to burn 300 kcal, so you hop on your favorite exercise equipment, punch in your BW, select manual protocol, and begin. When the console reads 300 kcal, you stop—proud of

having attained your goal. But did you really?

The answer to that question depends on a number of factors. The console number for kcal is derived mathematically from a prediction equation that typically takes into account BW and one or more measures of workload, such as stride rate, stride length, belt speed, elevation, and resistance or power output, depending on whether the equipment is a stair-stepping machine, treadmill, elliptical strider, rowing machine, or cycle ergometer. Research studies have generally shown that under identical conditions, calorie cost estimations are very consistent (reliable). However, the same cannot be said for the accuracy (validity) of the caloric cost values. For example, [Swain et al. \(1999\)](#) found that an elliptical motion machine significantly overestimated caloric cost (from 39% to 79%), with the larger overestimations occurring at the higher exercise intensities. In another study on an elliptical striding machine, [Heseltan et al. \(2000\)](#) found that although the mean caloric cost values at two of three workloads were not significantly different, the estimated values were systematically overestimated at levels above 300 kcal for a 30-minute workout, and the percentage of individuals whose measured caloric cost fell within 30 kcal of their estimated caloric cost was only 60%, averaged over two trials. A third study using an elliptical strider by [Mier and Feito \(2006\)](#) measured overestimations averaging 20–30% in caloric expenditure regardless of whether the participants used their legs only or arms and legs combined. Recently, it was suggested that elliptical trainers tend to overestimate caloric cost by at least 100 calories for every 30 minutes of exercise ([Glave et al., 2018](#)). Similar overestimations have been found for stairstepping machines ([Riddle and Orringer, 1990](#); [Ryan et al., 1998](#)), and several studies have reported that holding on to the point where part of the BW is supported results in a significant overestimation of caloric expenditure for both stair-steppers and the treadmill ([Åstrand, 1984](#); [Butts et al., 1993](#); [Howley et al., 1992](#)). The manufacturers of these machines try to provide accurate information to the exerciser, but errors are part of predictions, and the mathematical program cannot adjust if you “cheat” by holding on.

Therefore, the answer to the question of whether you actually burned 300 kcal is “probably not.” Console values for caloric expenditure should be interpreted as approximations rather than absolutes, and that approximation is probably high.



Sources: Åstrand (1984); Butts et al. (1993); Glave et al., 2018; Heselton et al. (2000); Howley et al. (1992); Mier and Feito (2006); Riddle and Orringer (1990); Ryan et al. (1998); Swain et al. (1999).

Table 4.6 presents a classification of absolute intensity ranges for physical activity in METs across fitness levels, where fitness level is defined as $\dot{V}O_2 \text{ max}$ and is reported in METs.

(Remember $\dot{V}O_2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \div 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} = \text{maximal METs}$.) For example, an individual with a fitness level of 8 METs should select activities between 3.8 and 5.1 METs to exercise at a moderate level. **Table 4.7** classifies work intensities as a rough guide for determining how long work can be sustained at each intensity. Individuals often work at different intensities in a laboratory setting than in the natural setting, so some variation in actual MET values is to be expected (Withers et al., 2006). Combining the information from this table and **Table 4.6**, for example, shows that most indoor household chores would be

considered very light to light work intensity and, theoretically, could be sustained indefinitely. Conversely, working as a Navy Seal or frogman is maximal work even for individuals with a maximal MET fitness level of 12 and can be expected to be sustained for only 1–2 hours occasionally. (Note: 12 METs = 42 mL·kg⁻¹·min⁻¹ $\dot{V}O_{2,max}$) Generically, sedentary behaviors are considered to be MET values less than 1.5, light-intensity activities 1.6–2.9, moderate-intensity 3–6, and vigorous 6 or more (Ainsworth et al., 2011). Happily we can see that sitting in class, taking notes, participating in discussions, and studying are relatively light metabolic work that can be sustained indefinitely.

TABLE 4.6 Classification of Physical Activity Intensity: Absolute Intensity Ranges Based on METs by Fitness Level

| | | Fitness Level | | | |
|-----------|-----------------|---------------------------|---------------------------|--------------------------|--------------------------|
| | | 12 MET $\dot{V}O_{2,max}$ | 10 MET $\dot{V}O_{2,max}$ | 8 MET $\dot{V}O_{2,max}$ | 6 MET $\dot{V}O_{2,max}$ |
| Intensity | Very light | <3.2 | <2.8 | <2.4 | <2.0 |
| | Light | 3.2–5.3 | 2.8–4.5 | 2.4–3.7 | 2.0–3.0 |
| | Moderate | 5.4–7.5 | 4.6–6.3 | 3.8–5.1 | 3.1–4.0 |
| | Hard (vigorous) | 7.6–10.2 | 6.4–8.6 | 5.2–6.9 | 4.1–5.2 |
| | Very hard | ≥10.3 | ≥8.7 | ≥7.0 | ≥5.3 |
| | Maximal | 12 | 10 | 8 | 6 |

Source: Reprinted with permission from American College of Sports Medicine: *ACSM’s Guidelines for Exercise Testing and Prescription* (9th ed.). Baltimore, MD: Wolters Kluwer Health/Lippincott Williams & Wilkins (2014).

TABLE 4.7 Work Classifications (A) and MET Levels for Selected Occupational and Home Activities (B)

| A. Classification | Time Work Can Be Sustained |
|--------------------------------|---|
| Very light to light | Indefinitely |
| Moderate | 8 hr daily |
| Hard | 8 hr daily for a few weeks only |
| Very hard | 4 hr 2–3 times per week for a few weeks consecutively |
| Maximal | 1–2 hr occasionally |
| Exhausting | Few minutes, rarely |
| B. Activities | MET Level |
| Carrying small children | 3.0 |
| Carpentry | 4.3 |
| Cleaning house | 3.8 |
| Construction | 4.0 |
| Cooking | 2.0 |
| Driving tractor | 2.8 |
| Food shopping | 2.3 |
| Electrical work/plumbing | 3.3 |
| Ironing | 1.8 |
| Fighting fires | 9.0 |
| Making bed-changing linens | 3.3 |
| Massaging | 4.0 |
| Masonry | 4.3 |
| Mowing lawn | 5.0 |
| Walk | 2.5 |
| Ride | 7.5 |
| Moving boxes | 12.0 |
| Navy seal/frogman | 1.8 |
| Playing trumpet | 4.5 |
| Painting | 3.8 |
| Playing drums | 3.8 |
| Raking leaves | 2.5 |
| Police directing traffic | 5.3 |
| Shoveling snow | 1.8 |
| Sitting in class | 2.5 |
| Snow blowing (walk) | 1.3 |
| Sitting studying | 2.5 |
| Washing dishes | 1.5 |
| Sitting talking (on/off phone) | |

Source: Modified from Ainsworth et al. (2011); <http://sites.google.com/site/compendiumofphysicalactivities>; Wells et

al. (1957).

Table 4.8 presents a classification of metabolic intensities in MET values for selected physical activities. This allows an individual to find activities for any of the intensity levels for exercise found in **Table 4.6**. For example, on average, water skiing (6 METs) would be appropriate as a moderate activity for

an individual whose $\dot{V}O_2\text{max}$ is 10 or 12 METs but not for those with lower MET max levels. Walking at $3.0 \text{ mi}\cdot\text{hr}^{-1}$ (3.5 METs) would be an appropriate activity for an individual whose

$\dot{V}O_2\text{max}$ is 6 or 8 METs. Active video games are a new addition to the compendium selected for **Table 4.8** because of their increasing popularity for individuals of all ages and in some physical education classes. Wii Fit values have been broadly categorized together into light effort (balance and yoga) 2.3 METs and moderate effort (aerobic and resistance) 3.8 METs. A more specific breakdown the MET values of 68 Wii activities has been determined by Miyachi et al. (2010). Forty-six activities were classified as light ($< 3 \text{ MET}$) and 22 as moderate (3–6 MET); no Wii activities were classified as vigorous ($> 6 \text{ MET}$).

TABLE 4.8 MET Values for Selected Physical Activities and Sports: Adults

| Activity | MET Level | Activity | MET Level | Activity | MET Level |
|-----------------------------|-----------|---------------------------------|-----------|--------------------------------|-----------|
| Archery | 4.3 | Frisbee | | Soccer | |
| Badminton | 5.5 | General | 3.0 | General | 7.0 |
| Social | 7.0 | Ultimate | 8.0 | Competitive | 10.0 |
| Competitive | 5.0 | Golf | 3.5 | Snow shoeing | 5.3 |
| Baseball/softball | 6.0 | Power cart | 4.3 | Squash | 12.0 |
| Basketball | 8.0 | Walking, carrying clubs | 3.8 | Swimming | 5.8 |
| General | 7.0 | Gymnastics | 12.0 | Freestyle (slow) | 9.8 |
| Competitive | 4.5 | Handball | 7.8 | Freestyle (fast) | 9.5 |
| Officiating | 7.8 | Hockey | 8.0 | Backstroke | 9.5 |
| Shooting | 4.0 | Field | 3.8 | Breaststroke | 13.8 |
| Wheelchair | 6.5 | Ice | 5.8 | Butterfly | 3.5 |
| Bicycling | 8.0 | Horseback riding | 7.3 | Treading water | 6.0 |
| <10 mi·hr ⁻¹ | 10.0 | Walking | 7.0 | Tennis | 8.0 |
| 10–11.9 mi·hr ⁻¹ | 12.0 | Trotting | 8.0 | Doubles | 4.0 |
| 12–13.9 mi·hr ⁻¹ | 8.5 | Canter/gallop | 11.3 | Singles | 6.0 |
| 14–15.9 mi·hr ⁻¹ | 3.5 | Kickball | 3.5 | Track and field | 10.0 |
| 16–19 mi·hr ⁻¹ | 6.8 | Lacrosse | 7.0 | Shot, discus | 2.3 |
| BMX/mountain | 8.8 | Rope jumping | 8.5 | Jumps | 3.8 |
| Stationary | 11.0 | 100–120 skips·min ⁻¹ | 5.8 | Hurdles | 7.2 |
| 50 W | 3.8 | Rowing and paddling | 5.0 | Video games | 3.0 |
| 100 W | 3.5 | Ergometer | 8.3 | Wii Fit balance and yoga | 6.0 |
| 150 W | 4.3 | 50 W | 9.8 | Wii Fit aerobic and resistance | 8.0 |
| 200 W | 2.3 | 100 W | 10.5 | | 2.8 |
| Bowling | 7.5 | 150 W | 11.5 | Exergames; Dance, Dance | 3.0 |
| Calisthenics | 9.5 | Canoe | 12.3 | Revolution, vigorous | 3.5 |
| Light, moderate | 3.0 | Moderate | 14.5 | Volleyball | 4.3 |
| Circuit | 5.5 | Kayak | 5.0 | General | 5.0 |
| Stretching | 6.0 | Running | 7.0 | Competitive | 5.0 |
| Dance | 8.0 | 12 min·mi ⁻¹ | 7.5 | Beach | 4.0 |
| Aerobic | 8.0 | 10 min·mi ⁻¹ | 6.8 | Walking (level) | 5.3 |
| 6- to 8-in. step | | 9 min·mi ⁻¹ | 9.0 | 2.0 mi·hr ⁻¹ | 3.5 |
| 10- to 12-in. step | | 8 min·mi ⁻¹ | 12.5 | 2.5 mi·hr ⁻¹ | 6.0 |
| Ballroom | | 7 min·mi ⁻¹ | 5.3 | 3.0 mi·hr ⁻¹ | 6.0 |
| Slow | | 6 min·mi ⁻¹ | 6.0 | 3.5 mi·hr ⁻¹ | 2.5 |
| Fast | | Skateboarding | | 4.0 mi·hr ⁻¹ | |
| Fencing | | Skating | | Using crutches | |
| Football | | Ice | | Pushing child | |
| Touch | | Rollerblading | | Water aerobics | |
| Competitive | | 9 mi·hr ⁻¹ | | Weight lifting | |
| | | Skiing | | Light, moderate | |
| | | Cross-country | | Heavy | |
| | | 2.5 mi·hr ⁻¹ | | Wrestling | |
| | | 4.0–4.9 mi·hr ⁻¹ | | Yoga (Hatha) | |
| | | 5.0–7.9 mi·hr ⁻¹ | | | |
| | | Downhill | | | |
| | | Moderate | | | |
| | | Water | | | |

Note: 1 MET = 1 kcal·kg⁻¹·hr⁻¹; 1 kcal·kg⁻¹·hr⁻¹/60 min·hr⁻¹ = kcal·kg⁻¹·min⁻¹.

Source: Modified from Ainsworth et al. (2011); <http://sites.google.com/site/compendiumofphysicalactivities>

Note that the compendium of MET values is intended for use with adults and probably are best applied only to young and middle-aged adults (<65 years). The absolute energy cost of walking and daily activities is higher in older adults and their resting metabolic rate is lower. Because of these factors, the use of the traditional definition of an MET to estimate the metabolic cost of activities in older adults is of questionable accuracy (Hall et al., 2014). There is a need for more information specific to this

group. Similarly, adult MET values are not necessarily applicable to children. Children have a higher basal metabolic rate per unit body mass than adults and due to various other developmental changes, the use of adult standard MET equivalency would underestimate basal metabolic rate in youth ([Butte et al., 2018](#)). [Butte and colleagues \(2018\)](#) present MET values for 196 activities for youngsters aged 6 to 18 years.

Field Estimates of Energy Expenditure during Exercise

Metabolic Calculations Based on Mechanical Work or Standard Energy Use

In situations where an accurate assessment of mechanical work is possible, energy expenditure, expressed as oxygen consumption ($\text{mL}\cdot\text{min}^{-1}$, $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, or METs), can be estimated through a series of calculations. The equations used are based on known oxygen costs for steady-state horizontal walking ($0.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for each $\text{m}\cdot\text{min}^{-1}$), horizontal running ($0.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for each $\text{m}\cdot\text{min}^{-1}$), vertical rise ($1.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for each $\text{m}\cdot\text{min}^{-1}$ of walking or $0.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for each $\text{m}\cdot\text{min}^{-1}$ of running), leg ergometer work against resistance ($1.8 \text{ mL}\cdot\text{kg}^{-1}$), and arm ergometer work against resistance ($3 \text{ mL}\cdot\text{kg}^{-1}$) for adults ([ACSM, 2022](#)). These values do not include the resting metabolic rate of 1 MET or $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of oxygen. See [Appendix B](#) for complete calculation instructions.

Example

For an individual walking on a track at a $20 \text{ min}\cdot\text{mi}^{-1}$ pace ($3 \text{ mi}\cdot\text{hr}^{-1}$), which is a velocity of $80.4 \text{ m}\cdot\text{min}^{-1}$ ($3 \text{ mi}\cdot\text{hr}^{-1} \times 26.8 \text{ m}\cdot\text{min}^{-1}\cdot\text{mi}\cdot\text{hr}^{-1}$), the calculation is as follows:

$$\begin{aligned}
 \text{Walking oxygen consumption} &= 80.4 \text{ mL} \cdot \text{min}^{-1} \times \left(\frac{0.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right) \\
 &\quad + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\
 &= 11.54 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}
 \end{aligned}$$

The resultant oxygen consumption value can easily be converted to METs as previously described by dividing by $3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. At the $20 \text{ min} \cdot \text{mi}^{-1}$ pace, this is 3.3 METs. **Table 4.8** shows that this is close to the actual measured value of 3.5 MET value for walking at $3 \text{ mi} \cdot \text{hr}^{-1}$.

These energy cost equations are useful for exercise prescription purposes in a college, health club, or clinical setting where it is not possible to directly measure energy expenditure. Physicians often prescribe exercise by MET level, assuming that the exercise leader will be able to determine the proper pace for walking or running, resistance on a cycle ergometer, or height and rate for step aerobics. The equations enable the exercise leader to do just that. Remember that the resultant values are just estimates for any given individual in any specific setting, however. Therefore, workloads should be finetuned using heart rate or RPE responses as described in [Chapter 13](#).

CHECK YOUR COMPREHENSION 5

How many METs is the subject in **Table 4.2** exercising at in minute 14?

Check your answer in [Appendix C](#).

Motion Sensors and Accelerometers

Attempts have been made to determine the energy cost in the field by measuring movement, assuming that more movement means greater calories expended. A familiar type of motion sensor is the pedometer (**Figure 4.7A**). More recent technology, such as wrist-worn WHOOP device, incorporates a pedometer but has more advanced technologies to also measure important physiological variables such as heart rate, heart rate variability,

and respiratory rate (**Figure 4.7B**). Basic pedometers are set to the individual's stride length and record the distance traveled by foot. [Crouter et al. \(2003\)](#) studied the accuracy of eight pedometers that also displayed kilocalories. It was unclear whether the pedometers were displaying net or gross kilocalories. If net kcal, the general trend was an overestimation of energy expended at every treadmill speed from 54 to 107 m·min⁻¹ (2–4 mi·hr⁻¹). If gross kcal, the accuracy of seven of the eight pedometers was within $\pm 30\%$ at all speeds. The authors concluded that pedometers are most accurate for measuring steps, less accurate for measuring distance, and even less accurate for assessing energy expenditure. These results were primarily supported in a 2011 study ([Nielson et al., 2011](#)) when step counts were found to be accurate at 100, 110, and 120 steps·min⁻¹ but not 80 or 90 steps·min⁻¹, and energy expenditure was significantly underestimated at 80 steps·min⁻¹ but overestimated at all other stride frequencies.

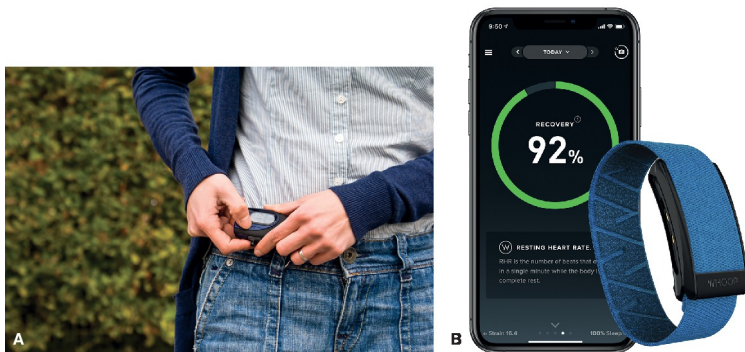


Figure 4.7 Motion Sensors and Activity Trackers.

A. A Pedometer. This woman is wearing a pedometer that measures step number and estimates distance and energy expenditure. **B.** Wrist-worn WHOOP® device incorporates a pedometer but has more advanced technologies to also measure important physiological variables such as heart rate, heart rate variability, and respiratory rate. The WHOOP® is linked to a smartphone application that also reports a daily recovery score.

Accelerometers are portable devices worn on the body that measure movement in terms of acceleration. Speed is a change in position relative to time. *Acceleration* is the change in speed relative to time. The unit of measurement is typically gravitational acceleration units (g ; $1\ g = 9.8\ \text{m}\cdot\text{sec}^{-2}$). Depending on the level of electronic sophistication, accelerometers detect acceleration(s) in one to three planes or axes (anteroposterior, mediolateral, and/or vertical). In some instances, this accelerometer information is supplemented by data from Global Positioning Systems (GPS). The raw outputs of accelerometers are known as counts. These counts are processed by linear or nonlinear regression equation software to determine the estimated energy expenditure. Some newer technology, such as wearable activity trackers or smart watches, use multisensors applied to several body segments (e.g., arm, waist, and ankle) and/or combines accelerometry with physiological sensors (e.g., for heart rate, skin temperature, or body temperature) in a single device (Chen and Bassett, 2005; Corder et al., 2007). As with pedometers, accelerometers and GPS units often are not accurate indicators of energy expenditure/metabolic equivalents (Hongu et al., 2013; Park et al., 2011); however, advancements in wrist-worn GPS activity monitors are now able to estimate maximal

oxygen uptake ($\dot{V}O_2$). $\dot{V}O_2$ numbers and the resultant energy expenditure measures are highly dependent on the mode of exercise (Passler et al., 2019) but can be useful for athletes and fitness participants who wish to base their training on these metrics. In a study comparing the accuracy of activity tracking in smartphone applications versus wearable devices, it was found that most popular smart phone applications and wearable devices were accurate in tracking step counts compared with pedometers (Case et al., 2015). Technology in pedometers and accelerometers continues to evolve in an attempt to get the best estimation of energy expenditure (Shephard and Aoyagi, 2012). Many smartphones now contain GPS units, accelerometers, gyroscope sensors, and/or databases that attempt to estimate activity energy expenditure (Jung et al., 2012; Shih et al., 2012). Information on the consistency (reliability) and accuracy (validity) of energy expenditure in smartphones and smart watches is still needed.

Activity Recalls and Questionnaires

In terms of technology, the least complex system for estimating energy expenditure is a self- or observer activity report in which either all activities or just exercise sessions are recorded (Laporte et al., 1985). The caloric cost of a particular activity or exercise session depends on the activity performed, its intensity and duration, and the individual's BW. The caloric cost can be calculated from a chart such as the ones presented in **Tables 4.7 and 4.8** by converting METs to $\text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. $1 \text{ MET} = 1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$; $1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} \div 60 \text{ min}\cdot\text{hr}^{-1} = \text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. The following formula is used:

$$\begin{aligned} &\text{total caloric cost of the activity (kcal)} = \text{caloric} \\ &\text{cost per kilogram of body weight per minute} \\ &(\text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}) \times \text{body weight (kg)} \times \text{exercise} \\ &4.5 \quad \text{time (min)} \end{aligned}$$

Example

If a 65-year-old, 84-kg female walks 3 mi in 1:15, how many kilocalories does she expend?

To use **Table 4.8**, first convert 3 mi in 1 hour and 15 minutes to mi per hour.

Table 4.8 shows that $2.5 \text{ mi}\cdot\text{hr}^{-1}$ at 3.0 METs is the closest approximation. Thus, an individual walking at $2.5 \text{ mi}\cdot\text{hr}^{-1}$ expends $0.05 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($3.0 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} \div 60 \text{ min}\cdot\text{hr}^{-1} = 0.05 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Substituting into this formula:

$$0.05 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \times 84 \text{ kg} \times 75 \text{ min} = 315 \text{ kcal}$$

Therefore, this individual expends 315 kcal in her walk, which is a good fitness workout.

For an assessment of total daily energy expenditure or even

weekly average expenditure, the process becomes more tedious and, as a result, is probably less exact. Even the most willing individuals have only so much time to write down everything they do; if activity is recorded after the fact, some activities will be forgotten. It is also possible that when asked to recall their activity and provide a record of that activity, some people may overestimate their energy expenditure. Typically, although estimated energy expenditure and activity patterns show large discrepancies from criterion values, questionnaires can make a broad distinction between active and sedentary individuals (Shephard and Aoyagi, 2012).

Efficiency and Economy

Efficiency

Walk into an appliance store to buy a furnace, hot water heater, washer, dryer, air conditioner, or refrigerator and each of the choices will have a label proclaiming its efficiency rating. Brand X uses only so much electricity (for just pennies a day!) to heat 40 gallons of water, wash a load of clothes, and so on.

Down the street, the car dealer is shouting the praises of the latest midsize economy car or hybrid; it has plenty of leg room, holds five adults comfortably, and gets 35–50 mi/gallon – 1 of gas. In each case, the concept is the same. We want to get the most output (in heating, cooling, or miles) for the least input and energy expense (electrical power, gas, or money). The same holds true for physical labor or exercise output: We want to get the most output (work) for the least input (ATP, kilocalories, or fuel used). One exception to this is probably the individual determined to lose weight.

The human body follows the *First Law of Thermodynamics*, also called the *Law of Conservation of Energy*. Simply put, this law states that energy can neither be created nor destroyed but can only be changed in form. When an individual exercises or performs other external work, the actual work achieved represents only a portion of the total energy utilized. The rest of the energy appears as heat, which must be dissipated, or body temperature will rise. The percentage of energy input that results

in useful external work is called the **mechanical efficiency**, or simply the efficiency of that task.

Mechanical Efficiency The percentage of energy input that appears as useful external work.

Efficiency can be calculated in at least three ways. The simplest calculation of efficiency is as *gross efficiency*.

$$4.6 \quad \text{gross efficiency} = \frac{\text{work output}}{\text{energy expended}} \times 100$$

Example

Calculate the gross efficiency for a 22-year-old female whose BW is 65.5 kg. She has ridden a Monark cycle ergometer (flywheel distance = 6 m) at 50 rev·min⁻¹ with a load of 2.5 kp for 15 minutes.

The external output is calculated as work equals force times distance ($W = F \times D$).

$$\begin{aligned} W &= 2.5 \text{ kp} \times (50 \text{ rev} \cdot \text{min}^{-1} \times 6 \text{ m}) \times 15 \text{ min} \\ &= 11,250 \text{ kg} \end{aligned}$$

It takes 426.8 kg of work to equal 1 kcal. Therefore,

$$11,250 \text{ kg} \div 426 \text{ kcal} \cdot \text{kg}^{-1} = 26.36 \text{ kcal of work output}$$

The amount of energy expended is calculated using [Equation 4.4](#) and multiplying by the total time of the exercise. The average oxygen consumption for the ride was 1.73 L·min⁻¹, and the RER was 0.91. At an RER of 0.91, the caloric equivalent ([Table 4.4](#)) is 4.936 kcal·L⁻¹ O₂. Substituting into [Equation 4.4](#), we get

$$\begin{aligned}
 &1.73 \text{ LO}_2 \cdot \text{min}^{-1} \times 4.936 \text{ kcal} \cdot \text{L}^{-1} \text{ O}_2 \\
 &= 29.95 \text{ kcal} \cdot \text{min}^{-1} \times 15 \text{ min} \\
 &= 128.09 \text{ kcal of energy expended}
 \end{aligned}$$

Equation 4.6 can now be used to determine gross efficiency:

$$\text{gross efficiency} = \frac{26.36 \text{ kcal}}{128.09 \text{ kcal}} \times 100 = 20.58\%$$

A slightly more complex method uses net efficiency. In *net efficiency*, the energy expended is corrected for resting metabolic rate.

$$\begin{aligned}
 \text{Net efficiency} &= \frac{\text{work output}}{\text{energy expended-resting} \\ &\quad \text{metabolic rate for} \\ &\quad \text{the same period}} \times 100
 \end{aligned}$$

4.7

Example

Calculate net efficiency using the same example as for gross efficiency; this means that the individual's resting metabolic rate (measured to be $1.11 \text{ kcal} \cdot \text{min}^{-1}$ or 16.6 kcal for the 15 minutes) must be subtracted from the total energy expenditure of 128.09 kcal before computing for efficiency. Thus, substituting in Equation 4.7, we get

$$\text{Net efficiency} = \frac{26.36 \text{ kcal}}{128.09 - 16.6 \text{ kcal}} \times 100 = 23.64\%$$

The third technique for calculating efficiency requires the use of at least two workloads and is based on the difference between the two loads. It is called *delta efficiency*.

$$\text{Delta efficiency} = \frac{\text{difference in work output between two loads}}{\text{difference in energy expenditure between the same two loads}} \times 100$$

Example

Calculate the delta efficiency for the 22-year-old female in the last two examples whose BW is 65.5 kg. This time she has performed two exercise stages on a treadmill, the first at 0% grade and the second at 10% grade. The speed was a constant 94 m·min⁻¹ (3.5 mi·hr⁻¹). The difference in work output is therefore primarily determined by the difference in percent grade—in this case, a 10% difference. Treadmill delta efficiency calculations are usually done on a perminute basis. Therefore, instead of calculating the change in work ($W = F \times D$) as in the first two examples, the change (Δ) in work, rate or power (P), which is work divided by time, is used (Adams et al., 1972; Gaesser and Brooks, 1975):

$$\Delta P \text{ kg} \cdot \text{min}^{-1} = \text{BW (kg)} \times \text{speed (m} \cdot \text{min}^{-1}) \times (\% \text{ slope} / 100)$$

Substituting, we get

$$\begin{aligned} \Delta P &= 65.5 \text{ kg} \times 94 \text{ m} \cdot \text{min}^{-1} \times (10/100) \\ &= 615.7 \text{ kg} \cdot \text{min}^{-1} \end{aligned}$$

Using the conversion of 426.8 kg of work is equal to 1 kcal, we then divide 615.7 kg·min⁻¹ by 426.8 kg for a difference in work output of 1.44 kcal·min⁻¹.

As before, energy expenditure is calculated using Equation 4.4. The average oxygen consumption at 0% grade is 0.91 L·min⁻¹ with an RER of 0.73. The caloric equivalent (Table 4.4) of the 0.73 RER is 4.714 kcal·L⁻¹ O₂. Substituting into Equation 4.4, this becomes

$$0.91 \text{ L O}_2 \cdot \text{min}^{-1} \times 4.714 \text{ kcal} \cdot \text{L}^{-1} \text{ O}_2 = 4.29 \text{ kcal} \cdot \text{min}^{-1}$$

The average oxygen consumption at 10% grade was 1.75 L·min⁻¹ O₂, and the RER was 0.86. The caloric equivalent of an RER of 0.86 is 4.875 kcal·L⁻¹ O₂. Substituting these values into Equation 4.4 leads to

$$1.75 \text{ L O}_2 \cdot \text{min}^{-1} \times 4.875 \text{ kcal} \cdot \text{L}^{-1} \text{ O}_2 = 8.53 \text{ kcal} \cdot \text{min}^{-1}$$

To obtain the difference in energy expended at the two workloads, simply subtract:

$$8.53 \text{ kcal} \cdot \text{min}^{-1} - 4.29 \text{ kcal} \cdot \text{min}^{-1} = 4.24 \text{ kcal} \cdot \text{min}^{-1}$$

Equation 4.8 can now be used to solve for delta efficiency:

$$\text{delta efficiency} = \frac{1.44 \text{ kcal} \cdot \text{min}^{-1}}{4.25 \text{ kcal} \cdot \text{min}^{-1}} \times 100 = 33.9\%$$

When used for the same exercise modality, these different methods of calculating efficiency yield very different results. For example, Gaesser and Brooks (1975) calculated gross efficiencies of 7.5–20.4%, net efficiencies of 9.8–24.1%, and delta efficiencies of 24.4–34% on the bicycle ergometer under the same controlled experimental conditions. Despite these differences, all these techniques are valuable because each technique is best suited for particular uses.

Gross efficiency is most useful for calculating values for specific workloads or speeds and is the most appropriate measure of whole body efficiency (Noordhof et al., 2010). For example, it answers the question: What is the efficiency of cycling into a 15 mi·hr⁻¹ (24 km·hr⁻¹) head wind, and how might that change with body position? Gross efficiency is also important for applications in nutritional studies where gross energy expenditure is a concern for adequate replenishment, such as during the Tour de France, when replacement is essential if a cyclist is to continue hard riding day after day. Furthermore, gross efficiency is the

measure reported most frequently, making it valuable for comparison purposes (Donovan and Brooks, 1977). It is an extremely robust measure and does not exhibit either day-to-day or within-day circadian rhythms for the same individual but can be different between individuals. Gross efficiency increases in a curvilinear fashion with exercise intensity (Ettema and Loras, 2009; Noordhof et al., 2010).

Net efficiency is an indication of the efficiency of work per se because it eliminates resting levels of energy that are not used to perform the work. Net efficiency implies that resting metabolic rate is independent of exercise intensity and isolated from work production. At least in cycling, this is not accurate (Ettema and Loras, 2009). In addition, it is not a realistic value because an individual performing any external work is still expending resting energy.

Delta efficiency is the most accurate means for determining the effect of speed or work rate on efficiency. It indicates the relative energy cost of performing an additional increment of work. Delta efficiency is also the technique of choice for calculating efficiency on a treadmill. Its use is necessary because technically no work (calculated as force \times distance) is done when the treadmill is horizontal (0% grade), despite the fact that energy is used. The reciprocal arm and leg movements cancel each other out, and there is no gain in vertical distance. If the treadmill is elevated so that an individual is walking or running up a grade, then work can be calculated.

All efficiency calculations assume a submaximal steady-state or steady-rate condition ($RER < 1.0$) and require that both work output and energy expenditure be expressed in the same units, typically kilocalories. The calculations may be done for the total time, as in the 15-minute bicycle ergometer ride used in the example for gross efficiency, or per unit of time, as in the per-minute calculations for the treadmill of delta efficiency. However, time can be a factor, since efficiency is generally high when a large amount of work is performed in a short period of time and low when a small amount of work is performed over a long period of time (Stegeman, 1981).

Because the same amount of physical work in any given exercise modality can cause different metabolic effects in

individuals, the energy cost ($\dot{V}O_2$ consumption) of the activity is the deciding factor in determining efficiency (Stegeman, 1981). **Figure 4.8** shows this basic relationship. As the energy cost ($\dot{V}O_2$ mL·kg⁻¹·min⁻¹ on the left Y-axis) increases, efficiency (% efficiency on the right Y-axis) decreases, and vice versa. Some of the factors that have been investigated to try and optimize efficiency for physical activities are described in the following section. Each of these could be plotted on the X-axis in **Figure 4.8**.

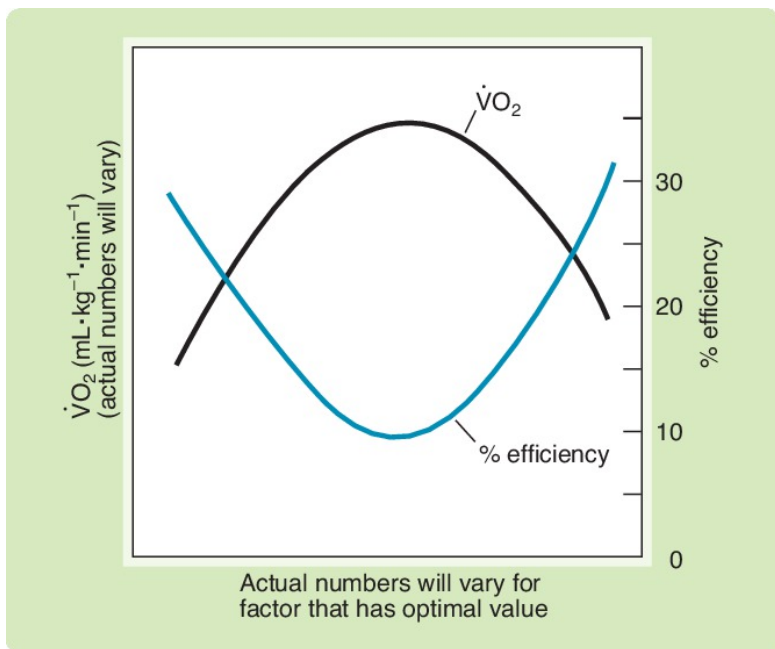


Figure 4.8 Optimizing Efficiency.

Note: Optimization means minimizing energy expenditure and maximizing work output. A variety of factors such as stride length, seat height, or pedaling frequency could be plotted on the X-axis. **Source:** Cavanagh and Kram (1985).

Practical Application of Efficiency Information

Certain factors have been shown to change efficiency and thus can be manipulated by an individual to optimize efficiency and improve performance. For example, the optimal seat height on a bicycle at any given power output has been found to be approximately 109% of leg length. Optimal pedaling frequency at any given power output has been shown to be between 40 and 60 rev·min⁻¹ for trained and untrained individuals, despite the fact that trained cyclists self-select a rate closer to 90 rev·min⁻¹. The reason for this difference is still not certain (Kohler and Boutellier, 2005) but may be due to the individual heart rate response while cycling (Reed et al., 2016). The higher revolutions per minute used by trained cyclists may also optimize muscular forces and lower limb stresses but not metabolic efficiency (Widrick et al., 1992). Maximal power might not be reached at a pedaling rate near the most efficient one. It may be that the optimal pedaling rate is not fixed but depends on race duration (Kohler and Boutellier, 2005). In addition, at high values of mechanical power, the relationship between mechanical efficiency and pedaling frequency is relatively flat, so little efficiency is lost with the higher pedaling frequencies (Lazzer et al., 2011). When the revolutions per minute are kept constant on a cycle ergometer, efficiency tends to increase from low to high workloads (Cavanagh and Kram, 1985; Hagberg et al., 1981; Stegeman, 1981).

The optimal speed for walking efficiency, when distance is held constant, is between 60 and 100 m·min⁻¹ (about 2.25 and 3.75 mi·hr⁻¹, or 16–27 min·mi⁻¹). The optimal grade for efficiency, when speed is held constant, is approximately 5% downhill (25%). The optimal stride length for efficiency, when speed is held constant, varies considerably among individuals. Most runners, however, intuitively select a stride length that is very close to optimal for themselves. Therefore, coaches who attempt to alter stride length may be harming efficiency.

Exercise efficiency values are most frequently reported in the 20–25% range. These values may be slightly higher (20–45%) if they are calculated as delta efficiencies and slightly lower (5–20%) if the activity involves air or wind resistance. Overcoming air resistance requires additional energy at all speeds of running and velocities of wind (including no wind). Therefore, in a running or cycling race, participants often draft behind (or tuck

in behind) the athlete in front of them. The first athlete must work harder than the athlete tucked behind, who does less work, hoping to save sufficient energy to put on a surge at the end of the race and pass the more fatigued front competitor or designated teammates as in an event such as the Tour de France. It has been shown (Broker et al., 1999) that during a team pursuit cycling race, performed at approximately $37 \text{ mi}\cdot\text{hr}^{-1}$ ($60 \text{ km}\cdot\text{hr}^{-1}$), the average mechanical power was equal to 607 W in the lead position, 430 W (-29.2%) in the second position, and 389 W (-36%) in third and fourth position.

The extra energy cost due to air resistance is proportional to the velocity at which the runner is moving raised to the second power, or $\text{m}\cdot\text{sec}^2$. Assuming there is no head wind, this amounts to approximately 2% extra energy expenditure for the marathon distance, 4–8% for middle-distance events, and 8–16% for sprints at world-class speeds. Although these percentages may not sound like much, they can be the equivalent of 5 minutes for a marathoner. At high levels of competition, this is a tremendous amount of time and would make a considerable difference in the results (Åstrand, 1952; Davies, 1980; Donovan and Brooks, 1977; Gaesser and Brooks, 1975; Pendergast et al., 1977; Pugh, 1970). Although running against a head wind can require additional energy, and thus decrease efficiency, the reverse is not true. A tailwind never assists the runner by decreasing the energy cost proportionally.

The mechanical efficiency of cycling, whether calculated as net efficiency or delta efficiency, is similar in prepubertal children, postpubertal adolescents, and adults (Klausen et al., 1985; Rowland, 1990). It may be slightly reduced in older adults such that a lower pedaling frequency is more efficient for older cyclists than younger ones (Sacchetti et al., 2010). These similarities are true whether the calculations are based on

relative ($\%\dot{V}\text{O}_2 \text{ max}$) or on absolute ($\text{kg}\cdot\text{min}^{-1}$ or W) work rates. The similarities also hold for males and females although, if anything, females may be more efficient than males at the same cycling power outputs (Bal et al., 1953; Girandola et al., 1981; Hopker et al., 2010; Sidney and Shephard, 1977; Taylor et al., 1950). Because of the relative constancy of efficiency, cycling is a good family activity.

FOCUS ON RESEARCH

The Impact of Body Posture on Cycling Efficiency

Eight junior, national, and professional male competitive cyclists performed two sets of trials after determination of their peak power output (PPO). The first set of trials consisted of three Wingate anaerobic 30-second leg tests: one seated on a Monark ergometer, one seated on his personal bike on an ascending road, and one standing on his personal bike on an ascending road. The second set of trials consisted of five sessions of 6-minute duration at 75% PPO: two seated-level cycling, two seated-uphill cycling (5.3% grade), and one standing-uphill cycling.

The data for the 30-second cycling sprints revealed that power output was lowest (603 ± 81 W) when seated on the Monark ergometer, slightly higher when seated on personal bikes (635 ± 123 W), but highest in the standing position (803 ± 103 W)—a difference of 20–30%, respectively, over the two seated power outputs. These results clearly show an advantage in short-term power output in short sprints for the standing position.

Data from the second set of trials are presented in the table below.

Remember that the power output was held constant at 75% PPO. Despite this constancy, both velocity and cadence were significantly lower in both uphill conditions. However, neither gross efficiency nor economy changed by either terrain (level or uphill) or body position (seated or standing).

The researchers concluded that there is an advantage for the cyclist to stand during short high-intensity hill climbs but to remain seated in longer steady-state climbs. However, which combination of speed and hill gradient favors climbing in the standing position remains to be determined.



Source: Millet, G. P., C. Tronche, N. Fuster, & R. Candau: Level ground and uphill cycling efficiency in seated and standing positions. *Medicine & Science in Sports & Exercise*. 34(10): 1645–1652 (2002).

| Condition | Power (W) | Velocity (km·hr ⁻¹) | Cadence (rev·min ⁻¹) | Gross Efficiency (%) | Economy (kJ·L ⁻¹) |
|-----------------|--------------|---------------------------------|----------------------------------|----------------------|-------------------------------|
| Seated level | 279.6 ± 34.7 | 35.8 ± 1.7 | 90.5 ± 6.5 | 22.4 ± 0.8 | 4.7 ± 0.2 |
| Seated uphill | 286.6 ± 35.3 | 18.3 ± 1.3* | 58.9 ± 4.1* | 22.2 ± 1.3 | 4.8 ± 0.3 |
| Standing uphill | 292.1 ± 34.6 | 17.9 ± 1.2* | 58.5 ± 4.1* | 22.5 ± 1.9 | 4.7 ± 0.5 |

*P < 0.001 for differences with the seated-level condition.

Economy of Walking and Running

As discussed earlier, the calculation of efficiency is a ratio of work output to energy expenditure input. However, measuring external work output may be impossible in many activities such as horizontal walking and running on the treadmill. Consequently, the energy expended to move mass or oxygen cost is often used alone for walking and running as a measure of economy, not efficiency.

Economy is the oxygen cost of any activity, but particularly walking or running at varying speeds. The relationship between oxygen cost and economy is inverse. That is, the more oxygen utilized in an activity, the lower the economy of that activity. This is similar to the concept that the more gas used to go any given distance at any given speed, the less economical the car.

Economy The oxygen cost of any activity, but particularly walking or running at varying speeds.

The most basic generalization about economy is that, over a wide range of velocities of walking or running, the energy cost (in $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of O_2) is rectilinearly related to the speed (in $\text{m}\cdot\text{min}^{-1}$). That is, higher speeds are less economical, albeit only slightly so (Lacour and Bourdin, 2015). This generalization is true at least at 0% grade on a treadmill when the speed can be accomplished at a submaximal steady-state level (Costill, 1986; Lacour and Bourdin, 2015). **Figure 4.9** shows this fundamental relationship. The black lines were computed from the equations recommended by the ACSM (2022). These equations, in turn, are based on a compilation of research. Note that the relationship between speed and oxygen consumption is not a continuous straight line encompassing both walking (**Figure 4.9A**) and running (**Figure 4.9B**). For walking speeds between approximately 50 and 100 $\text{m}\cdot\text{min}^{-1}$, each $\text{m}\cdot\text{min}^{-1}$ adds 0.1 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ above resting to the cost of the walk; for running speeds above 134 $\text{m}\cdot\text{min}^{-1}$, the increment is 0.2 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. Speeds between 100 and 134 $\text{m}\cdot\text{min}^{-1}$ are awkward for most people because they are too fast to walk but too slow to run. Additionally, outdoor running over ground is probably more demanding in terms of oxygen cost than indoor running on the treadmill at any given velocity, although overground inclined running equals the oxygen cost of treadmill-grade running (Daniels, 1985; Morgan et al., 1989b).

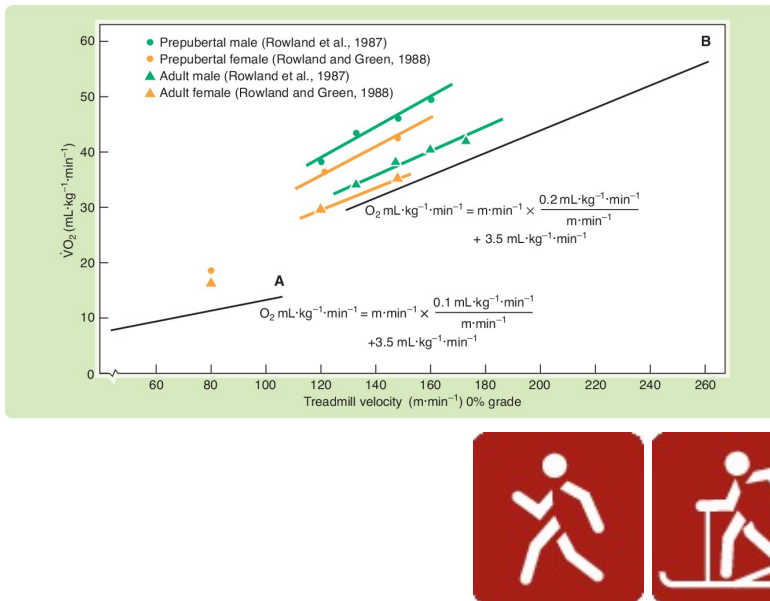


Figure 4.9 The Economy of Walking and Running for Children versus Adults.

The oxygen cost (expressed as $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) of walking (A) and running (B) increases rectilinearly as velocity increases. However, the slopes of the two lines are different, and the lines are not continuous from slow walking to fast running. Prepubertal males and prepubertal females are less economical than adult males and females as indicated by the higher oxygen cost at each and every velocity. **Sources:** ACSM (2022); Rowland et al. (1987); Rowland and Green (1988).

For any given individual, running economy appears to be relatively stable if environmental, equipment, and testing factors (such as body temperature, air resistance, footwear, time of day, and training status) are controlled (Morgan et al., 1989b). Day-to-day variations from 1.6% to 11% have been reported in the literature for welltrained and elite male runners. On the other hand, differences among individuals in terms of running economy are often extensive, ranging from 20% to 30%, in subjects of equal training and performance status. Highly trained individuals,

however, do exhibit better running economy than do untrained or lesser trained individuals. The precise reason for these observations has not been determined (Conley and Krahenbuhl, 1980; Daniels, 1985; McCann and Higginson, 2008), but a complex interplay between physiological and biomechanical factors is probably responsible (Barnes and Kilding, 2015; Lacour and Bourdin, 2015; Moore et al., 2012; Santos-Concejero et al., 2014; Tartaruga et al., 2012).

Male-female Differences in Economy

The influence of sex on variations among individuals in economy is unclear (Lacour and Bourdin, 2015). Different studies report that males expend more energy (Figure 4.9, green lines), less energy, or the same amount of energy in walking and running as do females (Figure 4.9, gold lines) when values are expressed relative to body mass (per kilogram of body weight). What is clear is that even if the oxygen cost is equal at any given speed, females, who typically have a lower $\dot{V}O_{2\max}$, will be working at a higher % $\dot{V}O_{2\max}$ than will males (Åstrand, 1956; Barnes et al., 2014; Bhambhani and Singh, 1985; Bransford and Howley, 1977; Bunc and Heller, 1989; Cunningham, 1990; Daniels, 1985; Morgan et al., 1989b). This fact has implications for the pace at which long-distance events can be run.

Studies on sex differences in economy in children and adolescents are more or less equally divided between those showing no sex differences and those showing girls with a higher economy (lower $\dot{V}O_2$ cost). No study has indicated that boys have a higher economy than do girls (Rowland, 2005).

FOCUS ON RESEARCH | Clinically Relevant

Wheelchair Propulsion Economy

Twelve able-bodied male participants (without prior preferences for frequency or strategy for wheelchair propulsion) completed four 5-minute exercise bouts at 32 W

(52%) in a randomized design on a basketball wheelchair ergometer. The purpose was to examine physiological responses to two selected push frequencies (40 and 70 push·min⁻¹) completed using either a synchronous (SYN) push strategy (both arms working simultaneously) or asynchronous (ASY) strategy (arms working alternately).

Results indicated that the SYN 40 condition was 9% more economical than the ASY 70, 12% more economical than the SYN 70 ($P < 0.01$), and 13% more economical than ASY 40 ($P < 0.01$). Heart rate values showed that the SYN 40 condition was significantly less stressful than all other conditions, and lactate concentrations were lower in both 40-push conditions as opposed to 70-push conditions. It was concluded that low push frequency combined with SYN arm motion provides the most economical form of wheelchair propulsion.



Source: Goosey-Tolfrey, V. L., & J. H. Kirk: Effect of push frequency and strategy variations on economy and perceived exertion during wheelchair propulsion. *European Journal of Applied Physiology*. 90:154–158 (2003).

Economy in Children and Adolescents

Age has a clear-cut effect on economy in youths. Ample evidence

shows that children and adolescents are less economical than adults when running/walking over a wide range of set external speeds, when economy is expressed as $\dot{V}O_2$ mL·kg⁻¹·min⁻¹ (Asmussen, 1981; Daniels, 1985; Girandola et al., 1981; Lacour and Bourdin, 2015; Larish et al., 1987; Morgan et al., 1989b; Robinson et al., 1976; Rowland, 2005; Rowland and Green, 1988; Rowland et al., 1987, 1988; Van de Walle et al., 2010).

Figure 4.9A and B shows the results of two studies conducted in the same laboratory in which male prepubertal children (aged 9–13 years) were compared with adult males (aged 23–33 years) (Rowland et al., 1987) and female prepubertal children (aged 8–12 years) were compared with adult females (aged 22–35 years) (Rowland et al., 1988) and how the four groups compared to the theoretical computed oxygen consumption values. In both sexes, the energy cost at all speeds of running was higher for children than for adults relative to body mass. Males were not tested for walking, but in females, at 80 m·min⁻¹, there was no apparent walking economy difference with age. The differences at the running speeds held true whether expressed as an absolute load (m·min⁻¹) or relative load. The adult values were much closer to the calculated values than the children's, indicating that the equations should not be used for children.

Figure 4.10A and B shows that children and adolescents differ in economy of running not only from adults but also from each other by sex and age (Åstrand, 1952). Indeed, there is a progressive decline in oxygen cost (progressive increase in economy) from the youngest (4–6 years) to the oldest (16–17 years) age groups in both boys and girls. A compilation of studies shows the decline in oxygen cost to be about 2% per year from 8 to 18 years when the same work is performed (Morgan et al., 1989b). Similar data are available for both boys and girls walking at 80–100 m·min⁻¹ at increasing grades. These changes are evident from cross-sectional studies in which different individuals are tested at each age and in longitudinal studies of the same individuals at different ages (Bar-Or, 1983; Costill, 1986; Daniels et al., 1978). The available data indicate that the oxygen cost of walking/running at any given speed decreases on average by 1.0 mL·kg⁻¹·min⁻¹ per year (Rowland, 2005).

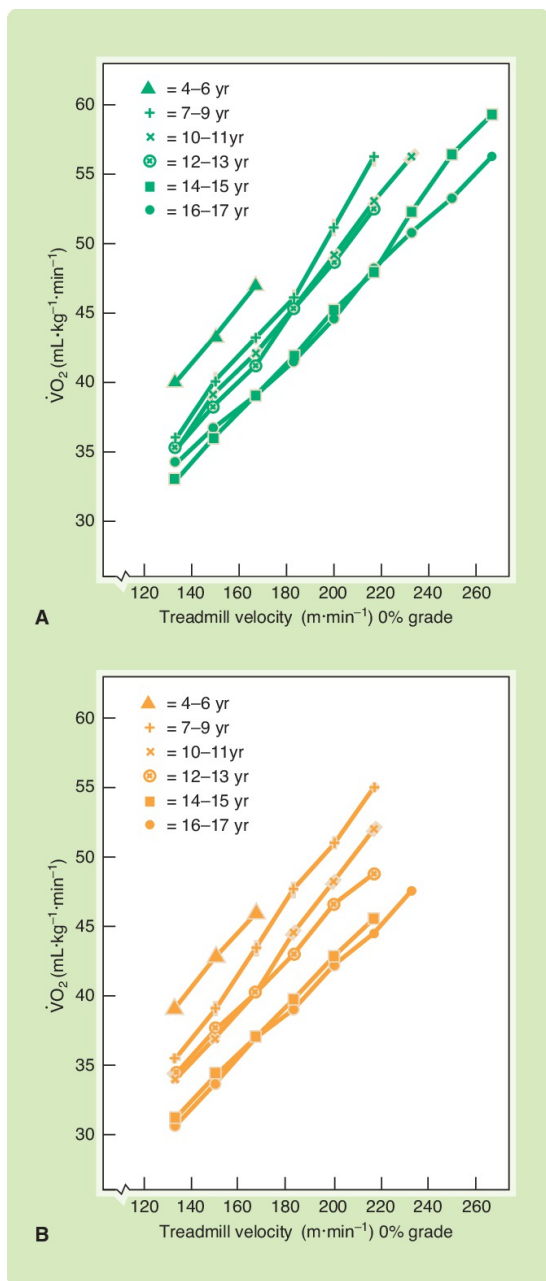


Figure 4.10 Running Economy of Children and Adolescents.

Running economy improves as both male (A) and female

(B) children age. This can be seen by the almost parallel and successively lower $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ values across the tested velocities of running. **Source:** Modified from Åstrand, P. O.: *Experimental Studies of Physical Working Capacity in Relation to Sex and Age*. Copenhagen, Denmark: Munksgaard (1952). Copyright © 1952 E. Munksgaard. Reprinted by permission of John Wiley & Sons, Inc.

There has been much speculation about the cause of this low economy in children. Five factors known to be affected by growth may offer at least a partial explanation.

1. *High basal metabolic rate.* Basal metabolic rate (BMR) is the minimum level of energy required to sustain the body's vital functions in a waking state, as measured by oxygen consumption. BMR is highest in young children and progressively declines to adulthood, where it stabilizes until it again declines in old age (Rowland, 1990) (see **Figures 4.11 and 8.4** in **Chapter 8**). Thus, gross exercise oxygen consumption values may be higher in children than in adults because resting metabolic rates are higher (Lacour and Bourdin, 2015). Bar-Or (1983) has pointed out that this difference in resting metabolism is only $1\text{--}2\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, and although this value represents a 25–35% greater BMR in children than in adults, it alone is unlikely to account for all of the difference in submaximal exercise values. Indeed, when Åstrand (1952) used net oxygen values instead of gross (by subtracting the BMR), the difference between the age groups was reduced but not eliminated. Thus, the use of net, not gross, values is better when interpreting oxygen consumption of walking/running for children and adolescents (Van de Walle et al., 2010).

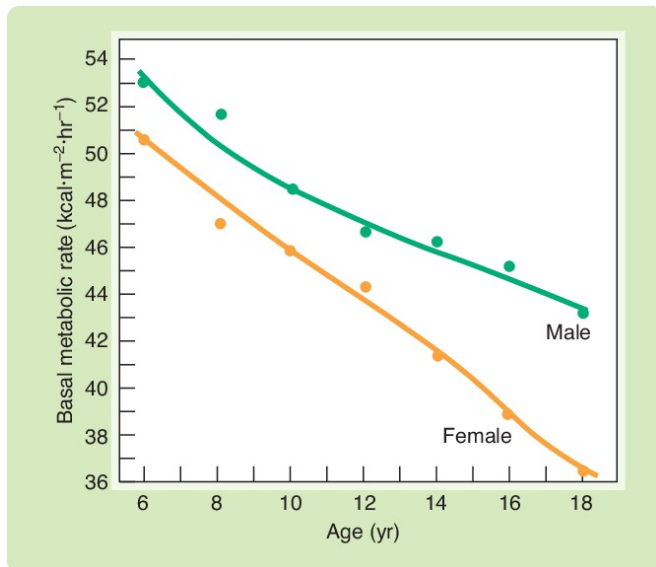


Figure 4.11 Changes in BMR with Age.

Source: From Rowland, T. W.: *Exercise and Children's Health*. Champaign, IL: Human Kinetics (1990).

Reprinted by permission of Dr. Thomas Rowland. Data from Knoebel (1963).

2. *Large surface area/mass ratio.* Throughout the animal kingdom, smaller animals (such as mice, squirrels, rabbits, or the young of any species) have higher resting metabolic rates per unit of body mass than do larger animals (dogs, horses, elephants, or the adults of any species). However, when the unit of comparison is not body mass (per kilogram) but body surface area (per square meter), metabolic rates are similar. This is called the *surface law*. The surface law appears to be important for the maintenance of normal core temperatures, as body heat loss is directly related to surface area: The larger the surface area, the greater the heat loss. Smaller individuals, as is typically true of the young, have greater surface areas per unit of mass than do larger individuals. Therefore, a higher resting metabolic rate is necessary to maintain body temperature in a smaller (younger) person. This phenomenon may be the reason for the elevated BMR in

the young (Rowland, 1990; Rowland and Green, 1988; Rowland et al., 1987). When Rowland and colleagues (Rowland, 1987, 1988, 2005) changed the unit of comparison from $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ to $\text{mL}\cdot\text{m}^2\cdot\text{min}^{-1}$, the differences between the prepubertal and adult subjects of both sexes were no longer significant and were almost eliminated.

3. *Immature running mechanics.* Watching a young child and an adult run together reveals obvious differences in motor skill (Rowland, 1990; Rowland et al., 1987). Young children seem to take numerous small, choppy steps involving lots of extraneous movements. At the same speed as an adult, the child has a higher stride frequency; a shorter stride length; a greater vertical displacement; increased ankle, knee, and hip extension at takeoff; increased cocontraction of antagonistic muscles; a longer nonsupport phase; and a shorter placement of the support foot in front of the center of gravity—all costly in terms of oxygen.
4. *Body size.* The variable that seems to have the most direct effect on the oxygen cost of running is stride frequency, and stride frequency is related to body size. Each stride requires energy to accelerate and decelerate the body's mass. Per stride, the oxygen cost is the same in children and adults. Similarly, when speed is adjusted for body size using a leg length per second ($\text{LL}\cdot\text{sec}^{-1}$) unit, children and adults are equally economical (Maliszewski and Freedson, 1996). However, at any given pace, the child has a greater frequency of strides simply because he or she is anatomically smaller. Thus, more oxygen is utilized (indicating lower economy) to provide the greater energy need (Rowland, 2005; Rowland et al., 1987). During typical daily physical activities, children and adults do not exercise at the same workload, but at a metabolic rate that is scaled to their body size. In these daily self-paced physical activities, unlike identical external loads such as miles per hour speed, children are not less energy economical than adults (Rowland, 2012).

Remember that each individual intuitively selects the most economical combination of stride frequency and length

for running. Therefore, one should not attempt to “make” children more economical by training them to lengthen their strides. Stride frequency decreases and stride length increases naturally as the child grows.

5. *Less efficient ventilation.* The volume of air breathed in to consume 1 L of oxygen is called the *ventilatory equivalent* (VE). Children have a higher VE than do adults. The processing of this additional air requires an added expenditure of energy. This increased metabolic cost of respiration may account for part of the increased oxygen cost at submaximal work in children, although not all research evidence agrees (Allor et al., 2000; Rowland, 1990; Rowland and Green, 1988; Rowland et al., 1987, 1988).
6. *Lower anaerobic capacity.* As discussed in the preceding chapter, children are less able to generate ATP anaerobically than are adults. The measurement of economy by oxygen consumption evaluates only the aerobic energy contribution. It is possible that at higher, but still submaximal, workloads, adults provide more of the required energy anaerobically and thus exhibit artificially low oxygen cost values (Armstrong et al., 2015; Rowland, 1990; Rowland et al., 1987). Evidence from RER values supports this contention. Children typically show lower RERs during submaximal exercise than do adults. Direct measurement of free fatty acid and glycerol levels shows, however, that the lower RERs are not the result of an increased utilization of fat as a fuel as might be expected. Instead, the lower RERs are thought to result from lower amounts of nonmetabolic “excess CO₂,” that is, the CO₂ generated from the buffering of lactic acid.

Economy in Older Adults

Economy in older adults appears to be higher than that in children, approximately equal to that in adolescents, and lower than that in young adults (McCann and Adams, 2002). The difference from young adults in oxygen cost has been observed to be anywhere from 8% to 31% (Malatesta et al., 2003; Martin et al., 1992; McCann and Adams, 2002; Mian et al., 2006; Ortega and Farley, 2007).

As with children, the cause of the increased oxygen cost

(lower economy) at any given speed of walking is unknown, although, unlike with children, there does not appear to be a size component ([McCann and Adams, 2002](#)). Mechanistic ideas about the difference include the following:

1. *Recruitment of additional motor units.* Aging brings a decline in the force-generating capacity of muscle. This may require the recruitment of additional motor units, including a higher proportion of less economical fast-twitch muscle fibers. More motor units require more oxygen to produce ATP ([Martin et al., 1992](#)).
2. *Gait instability.* Increased gait instability (based on stride time) and a greater energy expenditure needed to maintain balance during such instability have both been suggested as causes for the greater oxygen cost of walking in older adults. However, although [Malatesta et al. \(2003\)](#) found both greater stride time variability in 65-year-old and 80-year-old individuals and lower economy compared with 25-year-old individuals, these two variables were not significantly correlated. They concluded that the metabolic cost of the static contraction involved in maintaining balance was negligible.
3. *Antagonistic cocontraction.* Cocontraction or coactivation means that there is contraction or activation of antagonist muscles during agonist action. Excessive coactivation causes joint stiffening, limits range of movement, and may partly explain the elevated oxygen cost of walking in older adults. Again, more muscles require more oxygen for the production of ATP ([Malatesta et al., 2003](#); [Mian et al., 2006](#)).

Practical Application of Economy Information

Economy and efficiency probably matter little in high-intensity, short-term activities such as maximal weight lifts and sprints or in skilled movements such as golf. However, economy is extremely important in endurance events. Among those factors critical for success in endurance performances are a high $\dot{V}O_2\text{max}$, the ability to work at a high $\dot{V}O_2\text{max}$ aerobically

(sometimes measured as a high $\dot{V}O_{2\max}$ at LT1 [first lactate threshold]), and a high economy measured as a low submaximal oxygen cost at high velocities (Almarwaey et al., 2003; Conley and Krahenbuhl, 1980; Costill, 1986; Costill et al., 1973; Cunningham, 1990; Morgan et al., 1989a, 1989b; Saunders et al., 2004). What may matter most to competitive runners is a combination of economy and $\dot{V}O_{2\max}$ known as velocity at $\dot{V}O_{2\max}$ ($v\dot{V}O_{2\max}$).

Although a high $\dot{V}O_{2\max}$ is considered to be a prerequisite for success in distance or endurance events, it generally does not determine performance when participants are relatively homogeneous in that trait. For example, Costill (1986) presents data from two runners (Ted Corbitt and Jim McDonagh) who had similar $\dot{V}O_{2\max}$ values (64 and 65 mL·kg⁻¹·min⁻¹). Over a period of 2 years, they competed against each other in 15 major races, and the same runner (McDonagh) won each and every time! Obviously, the physiological explanation did not lie in $\dot{V}O_{2\max}$.

The importance of the ability to sustain a high percentage of $\dot{V}O_{2\max}$ aerobically for long periods of time is exemplified by the success of such runners as Grete Waitz (first woman to run a marathon in under 2:30, winner of New York City Marathon 9 times), Frank Shorter (1972 Olympic Marathon gold medallist and 1976 Olympic Marathon silver medallist), and Derek Clayton (first marathoner to run under 2:10) (Costill, 1986). These world-class runners were estimated to use 85–90% $\dot{V}O_{2\max}$ when they competed in marathons, whereas less successful marathoners average about 75–80% $\dot{V}O_{2\max}$. Typically, such a high percentage (85–90%) can be maintained only for a shorter (10 mi or less) distance. By continuing at that rate over twice as long as normal, these individuals gained a competitive edge. A 2010 study (McLaughlin et al., 2010) showed that when there was little variability in the % $\dot{V}O_{2\max}$ at LT1 during a 16-km race, it (% $\dot{V}O_{2\max}$ at LT1) did not explain distance running ability, and similar to the situation that when $\dot{V}O_{2\max}$ was homogeneous, it ($\dot{V}O_{2\max}$) did not explain endurance performance. However, the velocity (m·min⁻¹) at LT1 was highly correlated with the 16-

km run time.

The importance of a high running economy is seen in several situations. If two runners of widely varying $\dot{V}O_{2\max}$ (runner A = 65 mL·kg⁻¹·min⁻¹; runner B = 50 mL·kg⁻¹·min⁻¹) but similar economy attempt to train together, say at an 8-minute pace ($\dot{V}O_2$ cost = 40 mL·kg⁻¹·min⁻¹), runner B would be working much harder (80% $\dot{V}O_{2\max}$) than runner A (62% $\dot{V}O_{2\max}$). This variation in effort often occurs when females (average lower $\dot{V}O_{2\max}$, equal economy) run with males.

Differences in economy have a similar effect on performance. If, for example, runner A and runner B had the same $\dot{V}O_{2\max}$ (65 mL·kg⁻¹·min⁻¹) but widely varying economies at an 8-minute pace (runner A = 55 mL·kg⁻¹·min⁻¹; runner B = 40 mL·kg⁻¹·min⁻¹), then runner A would be working much harder (85% $\dot{V}O_{2\max}$) than runner B (62% $\dot{V}O_{2\max}$). This is the likely reason why McDonagh (above) continually beat Corbitt in their races. At all velocities faster than 8 min·mi⁻¹, Corbitt used significantly more oxygen than did McDonagh (Costill, 1986). Corbitt could have won only by making up the difference by running at a higher % $\dot{V}O_{2\max}$.

A similar situation occurs when a child (on average, equal in $\dot{V}O_{2\max}$ [in mL·kg⁻¹·min⁻¹] to an adult but lower in economy) runs with an adult as seen in **Figure 4.12**. At any given speed, the child (unless there is a great disparity in his or her favor for $\dot{V}O_{2\max}$) will be working harder than the adult. Although it is important for parents and teachers to encourage children to be active and for adults to do activities with children, the fact that the child will be working proportionally harder at any given running pace must be taken into account. The parent or teacher should match the child's pace or encourage just a slightly faster pace—not try to make the child keep up with what the adult finds comfortable. As a consequence, the adult will have to do his or her hard training without the child along, but the child will enjoy the experience much more. Since there appears to be little difference in efficiency in cycling as a function of sex or age as long as the wheel sizes are equal (allowing the same revolutions per minute), cycling may be a better family activity than running. Work through the [Check Your Comprehension 6—Case Study 3](#)

box. Check your answer in [Appendix C](#).



Figure 4.12 Children and Adults Exercising Together.

Because of the differences in running economy at any given speed when children/adolescents and adults run together, the youngest individual should set the pace.

CHECK YOUR COMPREHENSION 6—CASE STUDY 3

The Fitt family plans to run the Cornfest 10-km (6.2 mi) race together. Dr. Phyllis Fitt, the mother, is an exercise physiologist and recently tested all family members in her lab. Given the following information, what is the fastest time they can run with a reasonable chance of everyone finishing in good shape together?

| | Daughter | Son | Mother | Father | Grand-mother |
|--|----------|-----|--------|--------|--------------|
| Age (y) | 12 | 8 | 37 | 45 | 57 |
| $\dot{V}O_2$ max (mL·kg ⁻¹ ·min ⁻¹) | 48 | 50 | 50 | 52 | 40 |
| $\dot{V}O_2$ (mL·kg ⁻¹ ·min ⁻¹) | | | | | |
| 10 min·mi ⁻¹ | 37 | 39 | 30 | 32 | 33 |
| 9 min·mi ⁻¹ | 40 | 43 | 32 | 34 | 36 |
| 8 min·mi ⁻¹ | 45 | 48 | 38 | 41 | 40 |
| 7 min·mi ⁻¹ | 48 | 50 | 43 | 46 | |

Hint: Competitive runners typically average 80–90%

$\dot{V}O_2$ max during distance races of 5–10 mi; fun runners might be expected to be at the lower end of this value.

As stated previously, what may matter most to competitive runners is the combination of economy and $\dot{V}O_{2\text{max}}$ known as velocity at $\dot{V}O_{2\text{max}}$ ($v\dot{V}O_{2\text{max}}$). **Velocity at $\dot{V}O_{2\text{max}}$** is the speed at which an individual can run when working at his or her $\dot{V}O_{2\text{max}}$ based on both submaximal running economy and $\dot{V}O_{2\text{max}}$. **Table 4.9** and **Figure 4.13** show how this value is calculated and what it means. To calculate $v\dot{V}O_{2\text{max}}$, $\dot{V}O_2$ values (Y-axis) are plotted at several submaximal speeds (X-axis) for each individual, and a regression line—a line that best fits the $\dot{V}O_2$ values—is established. The line is extended to reach the individual's measured $\dot{V}O_{2\text{max}}$. The speed (or velocity) at which the $\dot{V}O_{2\text{max}}$ occurs is then determined by dropping a perpendicular line from the $\dot{V}O_{2\text{max}}$ point to the X-axis. This velocity is taken as the velocity at $\dot{V}O_{2\text{max}}$ ($v\dot{V}O_{2\text{max}}$). As the figure shows, it is possible to achieve the same $v\dot{V}O_{2\text{max}}$ with a high economy (low energy cost at submaximal speeds) and a low $\dot{V}O_{2\text{max}}$ (subject A) or with a

low economy (high energy cost at submaximal speeds) and a high $\dot{V}O_2 \text{ max}$ (subject B). Individuals such as these two with a similar $\dot{V}O_2 \text{ max}$ would be expected to have similar performances in endurance running events (Bird, 1991; Morgan et al., 1989a). All other factors (such as $\% \dot{V}O_2 \text{ max}$) being relatively equal, an individual with a high $\dot{V}O_2 \text{ max}$ would be expected to perform better than an individual with a low $\dot{V}O_2 \text{ max}$ (Almarwaey et al., 2003; Kilding et al., 2006; McLaughlin et al., 2010; Sirotic and Coutts, 2007).

Velocity at $\dot{V}O_2 \text{ max}$ The speed at which an individual can run when working at his or her maximal oxygen consumption, based on both submaximal running economy and $\dot{V}O_2 \text{ max}$.

TABLE 4.9 Information for Predicted Velocity at $\dot{V}O_2 \text{ max}$ for Subjects A and B (Figure 4.13)

| Subject | $\dot{V}O_2$, mL·kg ⁻¹ ·min ⁻¹ at Indicated Speed (m·min ⁻¹) | | | | | $\dot{V}O_2 \text{ max}$ (mL·kg ⁻¹ ·min ⁻¹) | $\dot{V}O_2 \text{ max}$ (m·min ⁻¹) | 10-km run time | $\dot{V}O_2 \text{ max}$ for 10 km |
|---------|---|-------|-------|-------|-------|---|--|-------------------|---------------------------------------|
| | 196 | 215 | 230 | 248 | 268 | | | | |
| A | 38.03 | 40.58 | 44.30 | 45.10 | — | 54.68 | 304.56 | 38:21 | 83.2 |
| B | — | 46.13 | 48.28 | 50.54 | 54.88 | 60.88 | 306.50 | 38:09 | 83.1 |

Source: Based on Bird (1991).

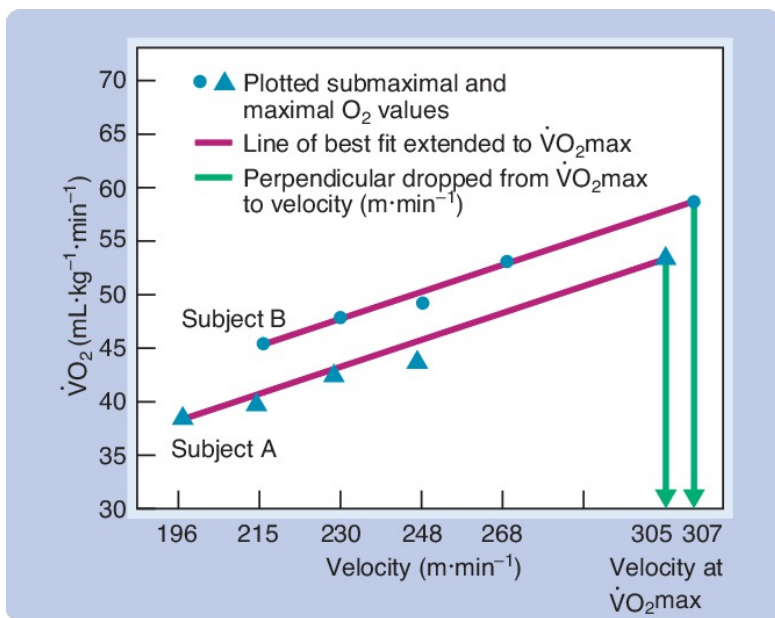


Figure 4.13 Predicted Velocity at O₂max.

Source: Based on Bird (1991).

The highest speed attained at the end of a graded exercise test with horizontal running can be used to predict performance, especially in a marathon, because it appears to reflect the physiological variables involved in $\dot{V}O_2 \text{ max}$ (McLaughlin et al., 2010). A recent study has shown that it is possible to get a

good estimate of $\dot{V}O_2 \text{ max}$ in adult males and females from performance of the PACER 20-m shuttle test. The equation is $\dot{V}O_2 \text{ max} = 0.093x + 6.89$, where x = number of shuttles completed.

This equation accounts for 87% of the variance between measured and predicted values (Paradis et al., 2014).

The low economy of children at any given external pace may help explain another phenomenon. Typically, endurance performance as measured by treadmill time to fatigue, PACER distance, mile-run time, and so on, improves in children with age despite a constant $\dot{V}O_2 \text{ max}$. Furthermore, although young

athletes generally have $\dot{V}O_{2\max}$ values higher than those of nonathletes, when training programs are undertaken, endurance performance often improves exclusively—or at least more than does $\dot{V}O_{2\max}$. Increases in body size that allows longer stride length and hence improved skill; age improvements in qualitative changes in oxygen delivery (not indicated by $\dot{V}O_{2\max}$); and/or the improvement of anaerobic strength and neurological function in the long term probably account for the enhanced endurance performances regardless of what occurs with $\dot{V}O_{2\max}$ (Rowland, 2013). Submaximal running economy in relation to a given external pace has been found to be statistically related to and to account for much of the variance in endurance performance in children and adolescents in most (but not all) studies. Since $\dot{V}O_{2\max}$ remains relatively stable and the O₂ cost of any given speed decreases, the child is working at a progressively lower percentage of $\dot{V}O_{2\max}$, as he or she ages. The individual is therefore expected to perform (endure) at the same speed for a longer period of time, cover more ground in the same time span, and/or increase the speed at which any given distance is covered (Bar-Or, 1983; Burkett et al., 1985; Daniels and Oldridge, 1971; Daniels et al., 1978; Krahenbuhl et al., 1989; Mayers and Gutin, 1979; McCormack et al., 1991; Rowland, 1990; Rowland et al., 1988).

Finally, for all the reasons discussed here, one cannot use adult prediction equations based on $\dot{V}O_2$ /speed relationships to predict $\dot{V}O_{2\max}$ in children.

Summary

1. Aerobic metabolism can be measured directly by calorimetry (the measurement of heat production) or indirectly by spirometry (the measurement of air breathed and the analysis of oxygen and carbon dioxide gases). Typically, open-circuit indirect spirometry/open-circuit indirect calorimetry is used.

2. During aerobic exercise, the amount of oxygen consumed and carbon dioxide produced increases. In a short-term submaximal activity, the metabolic costs level off where the energy requirements are met. This activity is called steady-state or steady-rate submaximal exercise.
3. If a submaximal exercise lasts for a long time, is above approximately 70% $\dot{V}O_{2max}$, or takes place under hot and humid conditions, oxygen drift occurs. The oxygen consumption drifts upward owing to rising levels of catecholamines, lactate, and body temperature as well as an increasing cost of ventilation and a shift in substrate utilization.
4. During incremental exercise to maximum, oxygen consumption rises in a rectilinear pattern proportional to the workload increments until the individual can increase oxygen utilization no more and a plateau occurs. The highest amount of oxygen an individual can take in, transport, and utilize during heavy exercise is called maximal oxygen consumption ($\dot{V}O_{2max}$).
5. Very-short, high-intensity anaerobic exercise and static and dynamic resistance activity are predominantly anaerobic activities. The oxygen contribution to the energy cost rarely exceeds one fourth to one half of the total energy cost but is a meaningful amount.
6. On the cellular level, the ratio of carbon dioxide produced to oxygen consumed is termed the respiratory quotient; as analyzed from expired air, it is termed the respiratory exchange ratio. Nonprotein RER is used most frequently in determining energy substrate utilization, with 0.7 being interpreted as fat, 1.0 as carbohydrate, and 0.85 as an approximate 50–50 mixture of both.
7. During exercise, the higher the intensity, the higher the RER value. During long-duration submaximal exercise, RER may decrease as the carbohydrate stores are depleted. During incremental exercise to maximum, RER may increase above 1.0, reflecting carbon dioxide increases from nonmetabolic sources. RERs for high-intensity static exercise and dynamic resistance exercise are between 0.8 and 1.0+, reflecting a mixed fuel supply during the static activity followed by

hyperventilation and a reliance on glycogen during the dynamic resistance activity.

8. The ability to transition from relying primarily on fat use for ATP production to carbohydrates use for ATP production as exercise intensity increases is called metabolic flexibility. When someone is metabolically inflexible (such as someone with obesity or diabetes), energy production can be impaired during exercise.
9. Given the amount of oxygen consumed, the RER during the same time span, and the caloric equivalent, caloric expenditure ($\text{kcal} \cdot \text{min}^{-1}$) and metabolic equivalents (METs) can be calculated.
10. Energy expenditure can be estimated in field settings using a series of metabolic formulas, motion sensors/accelerometers, or activity recall questionnaires and previously established caloric cost charts with varying degrees of accuracy.
11. Mechanical efficiency (gross, net, or delta) is some ratio of work output to energy expended. Economy is simply a measure of energy expended (oxygen consumed); it is used when measuring work output is difficult. Energy cost and economy are inversely related. Because the energy cost of walking and running is rectilinear over a wide range of speed, oxygen cost or MET values can be estimated when speed is known.
12. Variation in running economy is high between individuals but is low within the same individual. No clear-cut distinction in running economy has been shown between females and males. Children and adolescents are less economical than adults when running over a wide range of set speeds but not in self-paced activity. Older individuals are less economical when walking than young and middle-aged adults.
13. The exact reason why children are less economical than adults is unknown, but one or more of the following characteristics of children may be involved:
 - a. A higher basal metabolic rate
 - b. A larger surface area/mass ratio
 - c. Immature running mechanics

- d. Smaller bodies that result in shorter stride length and higher stride frequency
 - e. Less efficient ventilation
 - f. Lower anaerobic capacity
14. The exact reason why older adults are less economical than younger adults is unknown, but the following factors have been considered possibilities:
- a. Recruitment of additional motor units
 - b. Gait instability
 - c. Antagonistic cocontraction
15. A high economy (low oxygen cost) at any given velocity of running is beneficial to performance, especially when combined with a high $\dot{V}O_{2\max}$ and the ability to work consistently at a high percentage of $\dot{V}O_{2\max}$ aerobically (high percentage of $\dot{V}O_{2\max}$ at LT1). The higher the velocity at $\dot{V}O_{2\max}$ and the greater the percentage of $\dot{V}O_{2\max}$ an individual can maintain, the better his or her performance will be. Improvements in size and set pace running economy may explain why, as they grow and/or train, children typically improve in endurance-run performance (such as the mile run) but do not show as much improvement in $\dot{V}O_{2\max}$ as adults.

Review Questions

1. List and define the variables used to describe aerobic metabolic responses to exercise. Describe how each is obtained from laboratory or field tests.
2. Diagram the oxygen consumption response during (a) short-term, light- to moderate-intensity submaximal aerobic exercise; (b) long-term, moderate to heavy submaximal aerobic exercise; (c) incremental aerobic exercise to maximum; (d) static exercise; and (e) dynamic resistance

exercise.

3. Describe the relationship between the oxygen cost of breathing and the exercise intensity.
4. Explain the respiratory quotient and the respiratory exchange ratio. Relate them to the determination of energy substrate utilization, theoretically and numerically according to exercise intensity, duration, and type.
5. Describe metabolic flexibility and how it changes with exercise training and certain disease states.
6. Explain the similarities and differences between describing activity by kilocalories and describing activity by MET levels. How can both be practically applied?
7. Differentiate, in terms of definition, calculation, and application, among gross efficiency, net efficiency, and delta efficiency. How can cyclists maximize efficiency?
8. Compare running economy by sex and age. Discuss possible reasons for any observed differences. Give three situations where observed differences could have significant practical meaning.
9. Show how efficiency or economy can affect exercise performance.

Literature Search

We discussed metabolic flexibility in this chapter. Metabolic flexibility is critical to energy production during exercise of different intensities. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search metabolic flexibility, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Metabolic flexibility and ketogenic diet
 - ii. Metabolic flexibility and obesity
 - iii. Metabolic flexibility and diabetes

- iv. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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5 Metabolic Training Principles and Adaptations



CHAPTER OUTLINE

Introduction

Application of the Training Principles for Metabolic Enhancement

Specificity

Overload

Rest/Recovery/Adaptation

Progression

Individualization

Maintenance

Retrogression/Plateau/Reversibility

| |
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| Warm-up and Cooldown |
| Metabolic Adaptations to Exercise Training |
| Substrate or Fuel Supply |
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| Oxygen Utilization (7) |
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| Adaptations in Children and Adolescents |
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OBJECTIVES

After studying the chapter, you should be able to:

- Name and apply the training principles for metabolic enhancement.
- Describe and explain the metabolic adaptations that normally occur as a result of a well-designed and carefully followed training program.
- Discuss the influence of age and sex on the metabolic training adaptations.

Introduction

To provide a training program that meets an individual's metabolic goals, the training principles must be systematically applied. How these principles are applied will determine the extent to which the body uses the aerobic and/or anaerobic systems of energy production. Which energy systems are

emphasized will, in turn, determine the training adaptations that occur. Individualized training programs designed to bring about metabolic adaptations may be used to enhance performance in aerobic endurance or anaerobic events or health-related fitness.

Application of the Training Principles for Metabolic Enhancement

The training principles were described generically in [Chapter 1](#). Each training principle can be applied specifically in relation to the metabolic production of energy to support exercise.

Specificity

In order to be specific and match the demands of the event, a training program must begin with determining the goal. For example, a 50-year-old male who is enrolled in a fitness program and wants to break 60 minutes in a local 10-km race will have a very different training program from a 16-year-old high school student competing in the 400- and 800-m distances.

With the goal established, the relative contributions of the major energy systems can be estimated using a graph such as the one shown in [Figure 3.3](#). For the 50-year-old male described earlier, approximately 98% of the energy for his 60-minute, 10-km run is derived from the O₂ system, with the remaining 2% from the adenosine triphosphate-phosphocreatine (ATP-PC) and lactic acid (LA) systems. These percentages vary little even if an individual's time is considerably slower or even somewhat faster than 60 minutes. Thus, this individual's training regimen should emphasize the O₂ system.

For the high school middle-distance runner, it is a different story. Planning her training program requires knowing her typical times at those distances. In general, she would be expected to be in the 1:00–3:00-minute range for both distances. (The Track & Field News 2020 record for a high school female is 0:50.69 for the 400-m run [set in 2002] and 1:59.51 for the 800-m run [set in 2013].) Events in this range rely on approximately 60%

anaerobic and 40% aerobic metabolism, with a heavier reliance on the ATP-PC and LA anaerobic systems as performance speed increases. Because a faster time is the goal, her training should emphasize the anaerobic systems without neglecting the O₂ system.

In general, only by stressing the primary energy system or systems used in the activity can improvement be expected. The one exception to this rule appears to be the development of at least a minimal level of cardiorespiratory fitness, often termed an *aerobic base* for all sports. An aerobic base does not need to be obtained through long-distance running for all athletes. Other programs such as interval work may be more appropriate for sports that are basically anaerobic such as football (Kraemer and Gómez, 2001). Such a base should be achieved in the general preparation phase (off-season) of a periodization schedule (see **Figure 1.7**). This base prepares the athletes for more intense and specific training for anaerobic sports and aids in recovery from anaerobic work (Hoffmann et al., 2014). The most specific training for metabolic improvement should occur in the specific preparation phase (preseason) (see **Figure 1.7**).

For those sports in which performance is not measured in time (such as basketball, football, ice and field hockey, lacrosse, soccer, softball, rugby, tennis, and volleyball), the sport's separate components must be analyzed to determine which energy system supports it. For example, the average football play lasts 4–7 seconds and the total action in a 60-minute game (although perhaps spread over 3 hours) may be only 12 minutes. Thus, football training must emphasize the ATP-PC system (the 4- to 7-second range), not the O₂ system (the 60-minute time). Conversely, soccer matches last (without added time) 90 minutes. During this time, soccer players may cover 6–7.5 mi interspersed with 20–60 sprints of 10–20 m lasting 2–3 seconds each. Obviously, the aerobic load is much higher here, but the anaerobic requirement is high as well. In both cases, the ability to perform repeated short anaerobic bouts is very important and must be included in the training.

The time-honored technique of using fewer players on smaller fields, now called small-sided games (SSGs), is becoming more formalized by team sport coaches and investigated by

researchers. SSGs involve a brief period (seconds to a few minutes) of high-intensity movement followed by a predetermined period of rest. For example, in soccer, this might involve 3 on 3 play (+goalie) for 5×3 minute games focusing on some aspect the coach wishes to improve progressing over a period of 6 weeks to 10×3 minute games. All that is required to modify the intensity of the training is for the coach to manipulate the field dimensions, number of players on each side, time of play, and time of rest. SSGs make excellent activities for the specific preparation phase (in-season), competitive phase, and/or tapering macro or micro cycles to enhance conditioning, continue tactical development, and reduce training volume. Acute physiological responses and training adaptations to SSGs have been found to be similar to high-intensity interval training when structured with that intent, while also reporting more enjoyment during SSGs than high-intensity interval training (Hoffmann et al., 2014; Kunz et al., 2019; Owen et al., 2012; Selmi et al., 2020).

Specificity also applies to the major muscle groups and exercise modality involved. Most biochemical training adaptations occur only in the muscles that have been trained repeatedly in the way in which they will be used. Thus, a would-be triathlete who emphasizes bicycling and running in his or her program but spends little time on swimming should be more successful (in terms of individual potential) competing in duathlons instead.

Overload

Overload of the metabolic systems is typically achieved in one of two ways: first, by manipulating time and distance and second, by monitoring lactate levels and adjusting work intensity

accordingly. Maximal oxygen uptake ($\dot{V}O_{2\max}$), although a measure of aerobic power and a means of quantifying training load, is more a cardiovascular than a metabolic variable. Factors

contributing to the improvement of $\dot{V}O_{2\max}$ and the use of % $\dot{V}O_{2\max}$ reserve as an overload technique are therefore primarily discussed in the section on application of the cardiorespiratory training principles (see Chapter 13).

The Time or Distance Technique

The *time or distance technique* involves performing continuous and/or interval training. As the name implies, continuous training occurs when an individual selects a distance or a time to be active and continues uninterrupted to the end, typically at a steady pace. A runner who completes an 8-mi training run at a 7:30 min-mi-1 pace has done a continuous workout. The interaction of intensity (speed) and duration is important in continuous endurance work with intensity being more important until an individual threshold level is reached and then duration at or above that intensity becomes paramount (Green et al., 2013). If a continuous steady-state aerobic training session is maintained for an extended period of time or distance, it is sometimes called a **long slow distance (LSD) workout**. If several periods of increased speed are randomly interspersed in a continuous aerobic workout, the term *fartlek* is used. Thus, a **fartlek workout**, named from the Swedish word meaning “speed play,” combines the aerobic demands of a continuous run with the anaerobic demands of sporadic speed intervals. The distance, pace, and frequency of the speed intervals can vary depending on what the individual wishes to accomplish that day.

Long Slow Distance (LSD) Workout A continuous aerobic training session performed at a steady-state pace for an extended time or distance.

Fartlek Workout A type of training session, named from the Swedish word meaning “speed play,” that combines the aerobic demands of a continuous run with the anaerobic demands of sporadic speed intervals.

Interval training is an aerobic and/or anaerobic workout that consists of three elements: a selected work interval (usually a distance), a target time for that distance, and a predetermined recovery or relief period before the next work interval. The target

time for any given distance should be based on the time trials or past performance of the individual at that distance. The time period of the work interval determines the energy system that is stressed. A work time of less than 30 seconds stresses the ATP-PC system; one between 30 seconds and 2 minutes stresses the LA system. Anything over 2–5 minutes primarily stresses the O₂ system. The choice of length and type of recovery period also depends on the energy system to be stressed. Its length is typically between 30 seconds and 6 minutes, and the type may be rest-relief (which can include light aerobic activity and flexibility exercises) or work-relief (moderate aerobic activity). Examples of ATP-PC, LA, and O₂ interval sets are presented in **Table 5.1** (Fox and Mathews, 1974). Note that the three sets are not intended to be combined. In addition, other techniques for determining interval times can, of course, result in different target and recovery times for the same distance and ability level.

Interval Training An aerobic and/or anaerobic workout that consists of three elements: a selected work interval (usually a distance), a target time for that distance, and a predetermined recovery period before the next repetition of the work interval.

TABLE 5.1 Examples of Time-Distance Interval Training for Runners

| Energy System | Competitive Distance | Best Time | Training Distance | Training Time | Repetitions | Recovery Time | Recovery Type |
|----------------|----------------------|-----------|-------------------|---------------|-------------|---------------|---------------|
| ATP-PC | 100 (m) | 0:15 | 100 (m) | 0:18 | 8 | (1:3) 0:54 | Rest |
| LA | 1,500 (m) | 5:16 | 400 (m) | 1:20† | 5 | (1:2) 2:40 | Work |
| O ₂ | 1,500 (m) | 5:16 | 1,200 (m) | 4:24‡ | 3 | (1:1/2) 2:12 | Rest |

*This is not intended to be one workout, although the 100- and 400-m training sets could constitute one workout and the 1,200-m repeats another. Each would then total approximately 2 mi of intervals.

†Based on 1–4 sec faster than average 400 m during the 1,500–1,600-m race. ($1,500\text{ m} \div 100\text{ m} = 15$; $5:16 = 316\text{ sec} \div 15 = 0:21$; $100\text{ m} - 1 \times 4 = 1:24$; $400\text{ m} - 1; 1:24 - 0:04 = 1:20$.)

‡Based on 1–4 sec slower than average 400 m during 1,500–600-m-1 race. ($0:21 \cdot 100\text{ m} - 1 \times 12 = 252\text{ sec}$; $1,200\text{ m} - 1 =$

$$4:12 + 0:12 = 4:24.)$$

ATP-PC SYSTEM In this example of the ATP-PC set, the runner is doing 100-m sprints. Each repetition is to be run at 3 seconds slower (0:18) than her best time (0:15). A total of eight repetitions are to be completed with 0:54 of rest recovery (which may involve no or mild activity) between repetitions.

The amount of time required to restore half of the ATP-PC used—that is, the half-life restoration period for ATP-PC—is approximately 20–30 seconds, with full restoration taking at least 2 minutes to possibly 8 minutes (Fox and Mathews, 1974; Harris et al., 1976; Hultman et al., 1967). Thus, this individual should restore over half her ATP-PC while she rests.

During the same recovery time, myoglobin O₂ replenishment is also taking place. The amounts replenished and restored are influenced by the individual's activity during the recovery phase, with the greatest restoration occurring with rest during recovery (Dupont et al., 2004).

Because the ATP-PC stores recover so quickly, they can be called upon repeatedly to provide energy. Repeatedly stimulating the ATP-PC system should bring about an increase in its capacity. Any major involvement of the LA system is avoided by keeping the work intervals short so that little lactate accumulation occurs.

LA SYSTEM Stressing the LA system requires work durations of 30 seconds to 2 minutes. In this example, the runner is asked to perform five repetitions of 400 m each at a speed of 1:20, with a work relief recovery (which should include mild to moderate exercise) of 2:40 between repetitions. Lactate is produced in excess of clearance amounts during heavy work of this duration, resulting in an accumulation in the blood. Because lactate has a half-life clearance time of 15–25 minutes, with full clearance taking almost an hour, it is neither practical nor beneficial to allow for clearance of even half the accumulated lactate between repetitions.

Tolerance to lactate is increased by incomplete recovery periods of 1 minute 30 seconds to 3 minutes. This amount of rest allows the replenishment of myoglobin O₂ as well as most of the ATP-PC, thus allowing the high-intensity work in the next work

interval to be partially supplied by the ATP-PC energy system before stressing the LA system again (Fox et al., 1969). Of the three overload factors of frequency, intensity, and duration, intensity is most important for improving the capacity of the LA system. Work relief recovery is typically used at these work times, since active recovery does speed up lactate clearance.

O2 SYSTEM Long work bouts that are a portion of the competitive event (e.g., 0.5–1-mi [800–1,600 m] repeats for a 10-km runner) can be performed to stress the O2 system. The pace is typically close to average pace during competition and may exceed it. The smaller the proportion of the competitive distance that is performed with each repetition, the faster the pace and the more repetitions performed. The intent is for the intervals to be done primarily aerobically, however. The example in **Table 5.1** is for 1,200 m. Note that the time is longer than simply triple the 400-m time (4:12 vs. 4:24) and that the recovery time is proportionally very short. The 2:12 recovery allows for a large portion of ATP-PC restoration before the start of the next repetition. Because this pace is already relatively low-intensity work, a rest or walking recovery (as opposed to jogging) is best.

The distance for an interval workout (excluding warm-up and cooldown) should rarely exceed 2–5 mi (3.2–8 km) with a frequency of 1–3 d·wk⁻¹ (Costill, 1986; Rennie, 2007). High-intensity interval training taxes the muscles and joints, and one must be careful to avoid injury or overtraining. (Chapter 22 lists the signs and symptoms of overtraining.) Continuous work at lower intensities allows for greater frequency and longer durations, both of which lead to a greater volume of training. A high training volume is particularly important to endurance athletes.

Although originally used primarily by athletes, interval training in the format of low-volume high-intensity workouts is now becoming popular for fitness and rehabilitation training. These workouts are popular in part because the time commitment can be lower than with continuous endurance work and studies have shown that results are similar to continuous endurance work (Gist et al., 2014; Kessler et al., 2012; Nalcakan, 2014; Weston et al., 2014). Recently, it has been shown that time trial performance and VO₂max are improved more by high-intensity

interval training compared to moderate-intensity continuous training when volume is equated (MacInnis and Gibala, 2017; Rosenblat et al., 2020).

The Lactate Monitoring Technique

Assessing blood lactate concentration ([La⁻]) is the second common technique for monitoring overload. Ideally, this technique involves the direct measurement of blood lactate levels from a given workout. Currently, there is no general agreement about how best to use blood lactate values to design and monitor training programs. Nomenclature also varies greatly. Generally, however, six categories of workouts or training zones are useful (Table 5.2). These training zones are based on the lactate thresholds (LT1 and LT2) obtained during incremental exercise to maximum. The zones overlap considerably. The three lower zones (recovery, extensive aerobic, and intensive aerobic) involve predominantly low- to moderate-intensity aerobic activity, whereas the three higher zones (threshold, $\dot{V}O_{2\max}$ and anaerobic) represent the transition from aerobic to anaerobic energy supply at progressively higher intensities until both aerobic and anaerobic energy productions are maximized (Anderson, 1998; Bourdon, 2000).

TABLE 5.2 Training Zones Based on Lactate Thresholds and Values

| | Recovery | Extensive Aerobic | Intensive Aerobic | Threshold | $\dot{V}O_{2\max}$ | Anaerobic |
|--|--|---|--|------------------------------------|---|--|
| Relation to LT1 and LT2 | <LT1 | LT1 to halfway to LT2 | >LT1 but <LT2 | LT2 | >LT2 | Maximal |
| Lactate values (mmol·L ⁻¹) | <2.0 | 1.0–3.0 | 1.5–4.0 | 2.5–5.5 | >5.0 | >7.0 |
| RPE | <11–12 | 11–15 | 12–15 | 14–17 | 17–20 | 17–20 |
| Workout example | Low-intensity aerobic; for example, 20–30 min continuous | LSD; for example, 30 min to 2 hr continuous | Tempo runs; for example, 10–12 sec slower than 10-km race pace | Fartlek; for example, 1-min bursts | High-intensity intervals; for example, 6–8 reps 0:30–3:00 | Interval repetitions at maximum; for example, 2–4 reps 0:45–1:30 |

Sources: Based on information from Anderson (1998) and Bourdon (2000).

Although it is done for elite athletes, and small lactate monitors that can be easily used outside of the laboratory are

available, direct measurement of $[La-]$ during training for others is not very practical because of the necessity and cost of taking multiple blood samples. Some studies (Irving et al., 2006; Scherr et al., 2013) have demonstrated a reasonably stable relationship between blood lactate values and Borg's rating of perceived exertion (RPE) 6–20 scale (Borg, 1998). Borg's RPE scale is described fully in Chapter 13. The range of RPE values corresponding approximately to lactate values for each training zone is presented in Table 5.2.

A better method to individualize the use of RPE is to test the individual in a laboratory and record both RPE and lactate values at each progressive work rate. Figure 5.1 presents an example of such results. This individual reached his LT1 at 220 m·min⁻¹. At that speed, he reported an RPE of 12. He reached LT2 at a speed of 260 m·min⁻¹ with an RPE of 14. Combining these results with the guidelines from Table 5.2 means that his recovery workouts should be performed at an RPE of 9–12, extensive aerobic workouts at 12 or 13, intensive aerobic at 13–14, threshold workouts at 14, and $\dot{V}O_{2max}$ at an RPE of at least 15. The test results presented in Figure 5.1 do not represent a maximal test for this individual. Maximal workouts should elicit at least an RPE of 18 for everyone. Thus, an individual can be given a workout and an RPE value and can adjust his or her intensity accordingly. An alternative method, when a laboratory testing facility is available, is to determine the relationship between $[La-]$ and HR values. Then, HR can be used to estimate the $[La-]$ level during training sessions, and the intensity is modified accordingly (Dwyer and Bybee, 1983; Gilman and Wells, 1993).

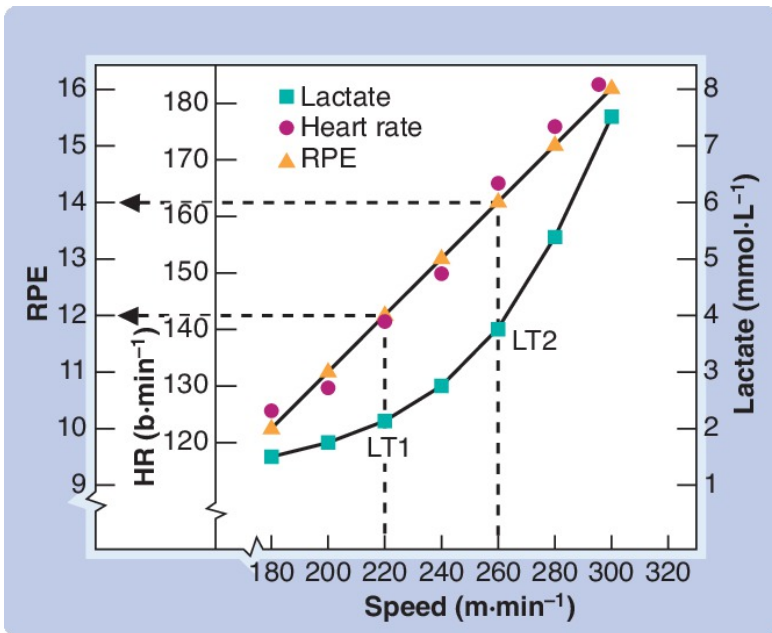


Figure 4.13 Heart Rate, Lactate, and Perceived Exertion Responses to Incremental Exercise as a Basis for Exercise Prescription.

LT1 occurred at 220 m·min⁻¹ and LT2 at 260 m·min⁻¹ for the individual whose data are plotted. See text for the explanation of how these points are used to prepare an exercise prescription.

In our example, now reading the rectilinear plot as heart rate, the individual would perform recovery activity between approximately 110 and 140 b·min⁻¹; extensive aerobic exercise bouts between 110 and 155 b·min⁻¹; intensive aerobic exercise between 120 and 165 b·min⁻¹; threshold workouts between 157 and 173 b·min⁻¹; $\dot{V}O_2$ max workouts close to 170 b·min⁻¹; and maximal activity at least at 185 b·min⁻¹.

Heart rate reflects primarily the functioning of the cardiovascular system, but lactate levels reflect the metabolic energy system. [Fox et al. \(1988\)](#) estimate that if the HR-[La-] relationship is not individually determined to ensure that all individuals are working at or above their “anaerobic threshold,” heart rate would have to be greater than 90% of the maximal heart rate or equal to or greater than 85% of the heart rate reserve in order to use it for anaerobic exercise prescription. Experimental data reported by [Weltman \(1995\)](#) confirm that techniques for exercise prescription involving percentages of heart rate maximum or heart rate reserve do not reflect specific blood lactate concentrations. Thus, unless individually correlated with lactate values, heart rate cannot be used for anaerobic exercise prescriptions. Regardless of the system used to prescribe an individual’s training session, a mixture of workout types in a periodized format ([Chapter 1](#)) should be used to maximize the possibility for improvement, minimize the possibility of overtraining, and prevent boredom.

Complete the [Check Your Comprehension 1](#) box to evaluate your understanding of intensity overload.

CHECK YOUR COMPREHENSION 1

Check your comprehension of prescribing exercise intensity in a periodization schedule for soccer players by matching the workout example in Column B with the most appropriate phase of periodization in Column A. Check your answers in [Appendix C](#).



| Periodization Phase | Training Workout |
|----------------------------------|---|
| 1. General preparation | A. Small-sided games (SSGs): 7 × 3 min 3 on 3 + goalie at an RPE of 8 |
| 2. Specific preparation | B. Bike or Swim 30 min <LT1 |
| 3. Competition phase maintenance | C. Run off-road, hilly terrain; long slow distance; 2 hr ~LT1 HR |
| 4. Transition phase | D. 2 × 5 min; 10 sec sprint/10 sec recovery at near maximal speeds OR 2 × 5 min high-intensity technical running—with ball (weaves, repeated passes, shots) |

Rest/Recovery/Adaptation

Adaptation is evident when a given distance or workload can be covered in a faster time with an equal or lower perception of fatigue or exertion and/or in the same time span with less physiological disruption (lower [La⁻] values) and faster recovery. The key to adaptation for energy production in muscles appears to be allowing for sufficient recovery time between hard-intensity workouts. Periodized training programs that alternately stress the desired specific energy system on a hard day and allow it to recover on an easy day lead to optimal adaptation ([Evans, 2019](#); [Judge and Burke, 2010](#); [Turner, 2011](#)). Too many successive hard days working the same muscles and same energy system can lead to a lack of adaptation because of overtraining, and too many successive easy days can lead to a lack of adaptation because of undertraining.

Recently, there has been considerable interest in the use of modalities to enhance recovery between training sessions or within tournament structures requiring multiple competitions. Overall, studies have not found massage, hyperbaric oxygen therapy (exposure to wholebody pressure greater than 1

atmosphere while breathing 100% oxygen), stretching, or electromyostimulation (the transmission of electrical impulses through surface electrodes to stimulate motor neurons and induce muscle contraction) advantageous. Research results for using water immersion depend on the temperature of the water. Cold water ($\leq 20^{\circ}\text{C}$) and contrast water therapy [alteration of cold and hot ($36\text{--}38^{\circ}\text{C}$)] offer more benefits than does either hot water immersion alone or thermoneutral immersion for decreasing acute inflammation. While this may be beneficial for in-season recovery for sport, chronically it may have negative impacts on adaptation. Exercise-induced inflammation leads to signaling to increase muscle protein synthesis, and if inflammation is blunted, there could be impairments to muscle growth. Further, decreases in dietary amino acid incorporation into muscle, decreased blood flow due to vasoconstriction, and decreased ribosome biogenesis could occur if cold water immersion is done regularly. Nonsteroidal anti-inflammatory drugs have potential negative health outcomes (cardiovascular, gastrointestinal, and renal) and may negatively affect muscle repair and adaptation to training. Cupping as a modality to enhance of recovery has resulted in no change to objective markers such as creatine kinase; however, subjective results seem to show improvements. Additionally, improved range of motion is typically observed with the utilization of cupping. Insufficient data are available to evaluate compression garments (stockings, sleeves, tights, and tops) (Barnett, 2006; Bridgett et al., 2018; Chaillou and Treigyte, 2020; King and Duffield, 2009; Malone et al., 2014; Montgomery et al., 2008; Poppendieck et al., 2013; Versey et al., 2013; Wilcock et al., 2006).

Progression

Once adaptation occurs, the workload should be progressed if maximizing improvement is desired (McNicol et al., 2009). Progression can involve increasing the distance, speed, workload, number of repetitions, or sessions; decreasing the length of the relief interval; or changing the frequency of the various types of workouts per week. The key to successful progression is an increase in intensity and total **training volume**. The progression should be gradual. A general rule of thumb is that training

volume—the total amount of work done, usually expressed as mileage or load—should not increase more than 10% per week. For example, for an individual currently cycling 60 mi·wk⁻¹, the distance should not be increased by more than 6 mi the following week. Step loading, as described in [Chapter 1](#), should be used.

Training Volume The total amount of work done, usually expressed as mileage or load.

Often in fitness work, the challenge is to prevent an individual from doing too much too soon. For example, a 50-year-old man remembers being a high school star athlete and wants to regain that feeling and physique—now! Fitness leaders must gently help such participants be more realistic and should err, if at all, on the side of caution in exercise prescription and progression.

Metabolic adaptation appears to plateau in approximately 10 days to 3 weeks if training is not progressed ([Hickson et al., 1981](#)). The ultimate limit may be set by genetics.

Individualization

The first step in individualizing training is to match the sport, event, fitness, or health goal of the participant with the specific mix of energy system demands. The second step is to evaluate the individual. The third step is to develop a periodization sequence for general preparation, specific preparation, competition, and transition phases. The fourth step is to develop a format: the number of days per week for each type of training or energy system to be stressed. The fifth step is to determine the training load (distance, workload, repetitions, or the like) based on the individual's evaluation and adjusted according to how he or she responds and adapts to the program. Interpreting and adjusting to an individual's response is the art of being a coach or fitness leader.

Maintenance

Once a specific level of endurance adaptation has been achieved, it can be maintained by the same or a reduced volume of work. How the volume is reduced is critical. When training intensity is maintained (i.e., exercising heart rate is maintained), training volume can be reduced by 33–66% (with just 13–26 minutes per session) or just 2 training session per week (such as during a 1-week vacation), and endurance performance can be maintained for up to 15 weeks (Spiering et al., 2021). Likewise, others have shown that if intensity is maintained, reductions of one third to two thirds in frequency and duration have been shown to maintain aerobic power ($\dot{V}O_{2max}$), endurance performance (at a given absolute or relative submaximal workload), and lactate accumulation levels at submaximal loads (Chaloupka and Fox, 1975; Neufer, 1989; Weltman, 1995). Conversely, a reduction in intensity brings about a decrease in training adaptation.

It also seems to be important that the mode of exercise is consistent with or closely simulates an athlete's activity because many metabolic training adaptations are specific to the muscles involved. Thus, cross-training—the use of different modalities to reduce localized stress but increase the overall training volume—is likely to be more beneficial for the cardiovascular system than for the metabolic system.

The level of maintenance training necessary for the anaerobic energy systems to keep operating at maximal levels is largely unknown. Sprint performances are known to deteriorate less quickly than endurance performances, with a decrease in training (Wilmore and Costill, 1988).

Many fitness participants are primarily in a maintenance mode after the initial several months or the first year of participation. The appropriate level for maintenance should be based on the individual's goals. For athletes, maintenance should occur primarily during the competition training cycle.

A special kind of maintenance called tapering is often used by athletes in individual sports such as swimming, cycling, and running. A **training taper** is a reduction in training load before an important competition that is intended to help the athlete recover from previous hard training, maintain physiological conditioning, and improve performance. Athletes often fear that if they taper more than just a few days, their competitive fitness

and performance will suffer. However, studies consistently show that if intensity is maintained while training volume is reduced, physiological adaptations are retained and performance either stays the same or improves after a taper (Bosquet et al., 2007; Costill et al., 1985; Houmard et al., 1990; Johns et al., 1992; Mujika and Padilla, 2003; Shepley et al., 1992; Travis et al. 2020; Zehsaz et al., 2011). **Figure 5.2** presents a schematic of the four different possible types of taper. Two of the variations are linear—one a gradual decrease in training load and the other making one large step down in training load and remaining at that level throughout the taper. Two of the variations are exponential, with either a large initial drop (fast decay) or slow initial drop (slow decay). Exponential techniques have been shown to be more effective than linear ones and fast decay more beneficial than slow decay. Optimal tapering strategies include the following:

Training Taper A reduction in training load before an important competition that is intended to allow the athlete to recover from previous hard training, maintain physiological conditioning, and improve performance.

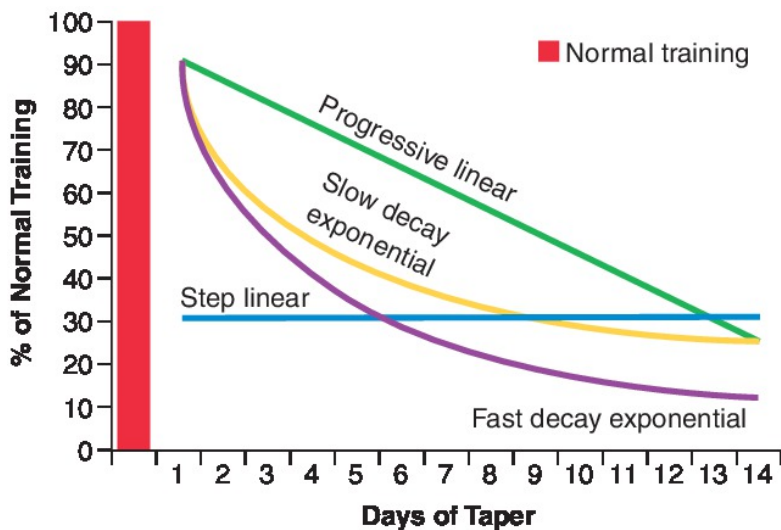


Figure 5.2 Representation of Taper Types: Progressive

Linear, Step Linear, Slow Decay Exponential, and Fast Decay Exponential.

Source: Modified with permission from Mujika, I., & S. Padilla: Scientific bases for precompetition tapering strategies. *Medicine & Science in Sports & Exercise*. 35(7):1182–1187 (2003). Copyright ©2003 The American College of Sports Medicine.

1. Using a progressive, fast decay exponential taper
2. Maintaining or even increasing training intensity
3. Reducing training volume by 41–60%, preferably by a decrease in the duration of each training session
4. Maintaining training frequency in highly trained athletes
5. Continuing the tapering intervention for 2 weeks although effective tapers have varied from 4 to 28 days
6. Ingesting only enough energy (kcal) to match the decrease in energy expenditure to avoid weight gain

The art of tapering involves balancing the goals of minimizing fatigue but not compromising previously acquired adaptations and fitness level. Tapering appears to be beneficial to both male and female athletes. Done properly, tapering strategies can result in an improved performance of approximately 2–3% (range 0.5–6.0%) ([Bosquet et al., 2007](#); [Mujika and Padilla, 2003](#)).

Retrogression/Plateau/Reversibility

Coaches and fitness leaders anticipate and react to, rather than apply, the training principles of retrogression, plateau, and reversibility. At one or more times in the process of training, an individual may fail to improve with progression and will either show a performance or physiological decrement (retrogression) or stay at the same level (plateau). When such a pattern of nonimprovement occurs, it is important to check for other signs of overtraining. Changing the training emphasis or including more easy days may be warranted. Remember that reducing training load does not necessarily lead to detraining. Of course, not all training plateaus can be explained as overtraining; sometimes there is no explanation.

If an individual ceases training completely, for whatever reason, detraining or reversibility of the achieved adaptations will occur.

Warm-up and Cooldown

Information about the effects of warm-up on metabolic function is sparse, but several generalizations can be made. An elevated body temperature—and more specifically an elevated muscle temperature (T_m)—increases the rate of metabolic processes in the cells. This increase occurs largely because enzyme activity is temperature dependent, exhibiting a steady rise from 0°C to approximately 40°C before plateauing and ultimately declining. At the same time, at elevated temperatures, oxygen is more readily released from the red blood cells and transported into the mitochondria. Therefore, one consequence of an increased T_m is a greater availability of oxygen to the muscles during work. When more oxygen is available sooner, there is less reliance on anaerobic metabolism, and less lactate accumulates at any given heavy workload. At lighter endurance workloads, a greater utilization of fat for energy is possible earlier in the activity. This early use of fat spares carbohydrate and allows a high-intensity effort to be continued longer. Other metabolic benefits of an increased T_m include increased glycogenolysis, glycolysis, and ATP-PC degradation (Bishop, 2003; McGowan et al. 2015). These beneficial metabolic effects of a warmup appear to occur in children and adolescents as well as adults (Bar-Or, 1983).

As with the other training principles, to be effective, the warm-up needs to match the intensity and duration of the intended activity. In general, a warm-up should consist of three elements in this order: a period of aerobic exercise, stretching, and a period of activity similar to the event to be performed (Fradkin et al., 2010). In addition, the structure of the warm-up should depend on the participant's fitness level, the environmental conditions (see Chapter 3, Focus on Research: The Effect of Cold Ambient Temperature on Lactate Kinetics), and specific constraints of the situation in which the warm-up will occur. Several guidelines are available for devising a warm-up to achieve these metabolic benefits and possibly improved performance (Bishop, 2003; McGowan et al., 2015).

1. For a short-term (<10 seconds) high-intensity activity, the goal is to increase T_m but allow time for resynthesis of ATP-PC immediately before the activity. Research suggests a warm-up performed at approximately 40–60% $\dot{V}O_{2\max}$ (~60–70% HRmax) for 5–10 minutes followed by a 5-minute recovery period is optimal.
2. Explosive tasks (such as long or high jumping) at full speed should be used sparingly during warm-up even if they are to be performed during exercise or competition. However, a brief, task-specific burst of activity may be beneficial, or the action can be patterned at lower levels.
3. For an intermediate or long-term high-intensity activity, the goal is to elevate baseline oxygen consumption without either causing fatigue or imposing a high thermal load. Research suggests a warm-up performed at approximately 60–70% $\dot{V}O_{2\max}$ (~70–80% HRmax) for 5–10 minutes, followed by approximately 5 minutes of recovery for moderately trained individuals.
4. The warm-up for endurance activities generally can occur at a lower intensity (25–30% $\dot{V}O_{2\max}$ or <35% HRmax) than for short or intermediate high-intensity activities. The warm-up may be built into an endurance workout if the participant begins at a low intensity and progresses nonstop into higher levels of work.
5. Very fit individuals can use longer, more intense warm-ups than can less fit individuals. Higher intensity may be needed by well-conditioned athletes to elevate their core temperature, but fatigue should be avoided. It is probably best to stay below the first lactate threshold and to avoid impacting glycogen stores negatively.
6. An intermittent or interval-type warm-up has been found to be more beneficial for children than a continuous warm-up.

Complete the [Check Your Comprehension 2](#) box to assess your understanding of the benefits of warm-up on metabolism.

CHECK YOUR COMPREHENSION 2

The metabolic changes described in the Focus on Application: Clinically Relevant box are considered beneficial. Why?

Check your answer in [Appendix C](#).

The primary metabolic value of a cooldown lies in the fact that lactate is dissipated faster during an active recovery. As described in [Chapter 3](#), the lactate clearance rate is maximized if the cooldown activity is of moderate intensity (a little higher than an individual tends to self-select) and continues for approximately 20 minutes.

In general, all training principles appear to apply to both sexes and, except where noted, to all ages. At the very least, there is insufficient evidence for modifying any of the general concepts based on age or sex, although individual differences should always be kept in mind.

FOCUS ON APPLICATION | *Clinically Relevant*

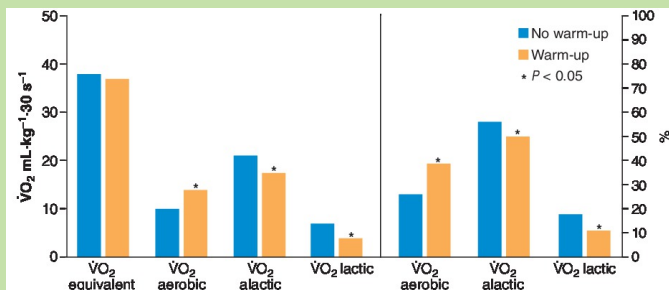
The Effect of Warm-Up on Metabolism

Twelve female ballet dancers (age = 13.7 ± 10 years; HT = 1.61 ± 0.05 m; WT = 45 ± 7 kg; and $\dot{V}O_{2\max} = 46 \pm 2$ mL·kg⁻¹·min⁻¹) at the Italian National Academy of Dance were tested to determine the effects of active warm-up on energy cost and energy source. As in classical dance practice, the warm-up consisted of 3 minutes of free jogging (25–35% $\dot{V}O_{2\max}$), 15 minutes of stretching (10–20% $\dot{V}O_{2\max}$), 2 minutes of *pre barre*, and 2:40 of *plié* dance exercises. The ballet dance exercise consisted of a sequence of 25 *tours piques* on full *pointe* performed on diagonal continuously for 30 seconds to music.

The total energy requirement ($\dot{V}O_2$ equivalent) was determined by adding the amount of measured $\dot{V}O_2$ during

exercise above resting ($\dot{V}O_2$ aerobic) to the $\dot{V}O_2$ from the fast component of recovery ($\dot{V}O_2$ alactic) and the energy equivalent of peak lactate accumulation ($\dot{V}O_2$ lactic).

The metabolic demand was 1.6 times the dancers' $\dot{V}O_{2\max}$ both with and without warm-up, so the energy cost of the activity (graph below, $\dot{V}O_2$ equivalent) did not change. The anaerobic systems were taxed more in both conditions. Relatively (right side of the graph), the anaerobic systems ($\dot{V}O_2$ alactic + $\dot{V}O_2$ lactic) provided 74% of the energy without warm-up and 61% with warm-up. The decreases in anaerobic energy sources (both alactic and lactic, absolute and relative) following warm-up were significant. Conversely, the aerobic component increased significantly.



Source: Source: Guidetti et al. (2007).

Metabolic Adaptations to Exercise Training

When the training principles discussed earlier are systematically applied and rigorously followed, a number of adaptations occur relative to the production and utilization of energy. The extent to which adaptations occur depends on the individual's initial fitness level and genetic potential. **Figure 5.3** is an expanded

version of **Figure 2.4**, showing the metabolic pathways you studied earlier. Numbers have been inserted on the figure to indicate sites where these adaptations occur. The following discussion will follow that numerical sequence. Refer to **Figure 5.3** as you read.

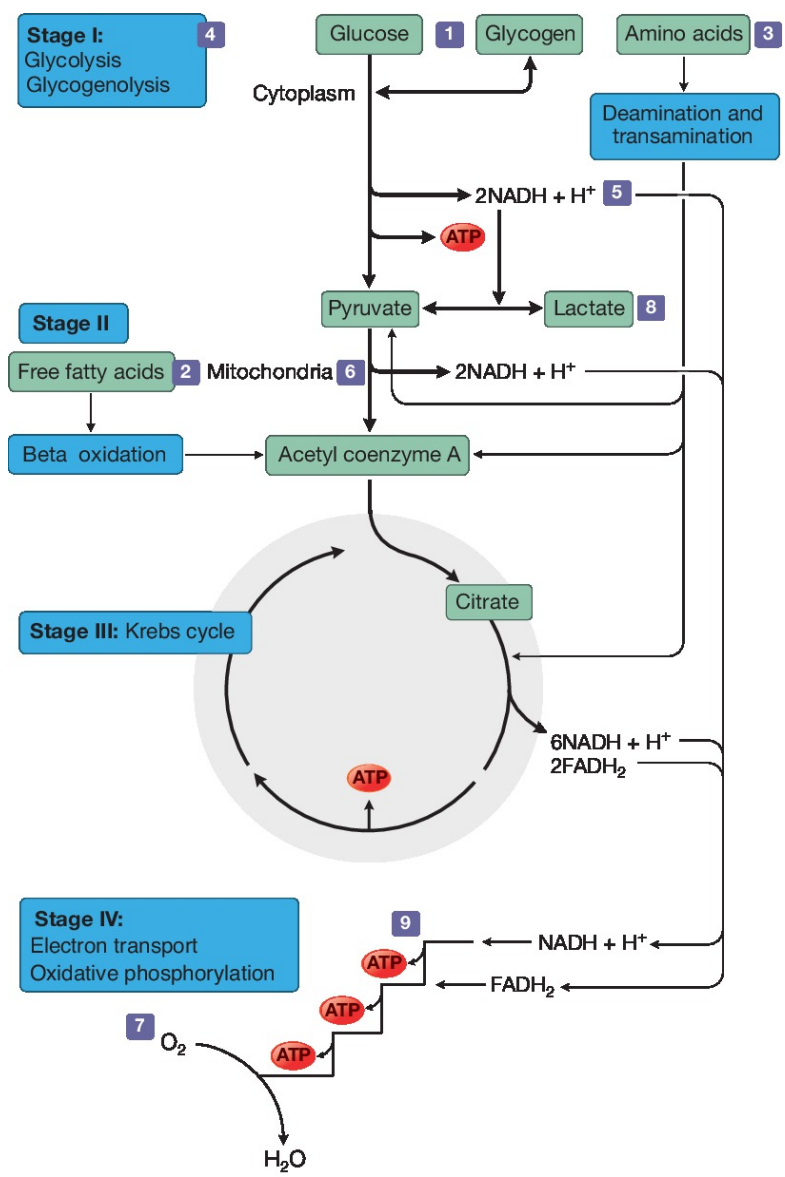


Figure 5.3 Metabolic Training Adaptations.

The numbers in *squares* indicate sites where training changes occur. The *blue* boxes indicate processes. The *green* boxes indicate important substrates.

Substrate or Fuel Supply

Regulatory Hormones

Primary among the metabolic adaptations to a training program are changes that occur in the hormones responsible for the regulation of metabolism (see [Chapters 2 and 21](#), [Figure 2.17](#), and [Table 2.3](#)). Although little is known about the impact of training on the hypothalamic-releasing factors and adrenocorticotrophic hormone, a definite pattern occurs with the five hormones directly involved in carbohydrate, fat, and protein substrate regulation. That pattern is one of a blunted response in which the amount of hormone secreted during submaximal aerobic activity is reduced. This pattern occurs whether the load is absolute or relative and in both the fast-responding and slow-responding hormones. Thus, the rise in epinephrine and norepinephrine is less in the trained state. As a result, the rise in glucagon (stimulated by epinephrine) is lower and there is less suppression of insulin (caused by norepinephrine). Similarly, the rise in growth hormone and cortisol is less during submaximal exercise in trained individuals than in untrained individuals ([Galbo, 1983](#); [Talanian et al., 2007](#)). Because of these smaller disruptions at submaximal levels, more work can be done before maximum is reached.

Carbohydrate (1)

The rate-limiting step for glucose utilization in muscles is glucose transport, and glucose transport is primarily a function of GLUT-4 transporters. Exercise training increases the GLUT-4 number and concentration in skeletal muscle, especially slow-twitch oxidative fibers ([Daugaard et al., 2000](#); [Gibala and McGee, 2008](#); [Perry et al., 2008](#); [Sato et al., 1996](#); [Seki et al., 2006](#)). This results in a greater uptake of glucose under the influence of insulin. Thus, at

any resting insulin level, the whole-body glucose clearance is enhanced. This occurs in both young and older healthy individuals as well as in individuals with non-insulin-dependent diabetes (Dela, 1996). Despite this increase in the number of GLUT-4 transporters, endurance exercise training reduces glucose utilization during both absolute and relative, moderate-intensity submaximal exercise. This occurs because the translocation of GLUT-4 decreases during exercise. As a result, trained muscles take up and utilize less glucose than do untrained muscles during moderate exercise and have a greater reliance on fat metabolism during moderate exercise than untrained individuals (Mougios, 2006).

Both endurance and sprint training increase muscle and liver glycogen reserves. In addition, at the same absolute submaximal workload (the same rate of oxygen consumption), muscle and liver glycogen depletion occurs at a slower rate in trained individuals than in untrained individuals (Abernethy et al., 1990; Gollnick and Hermansen, 1973; Gibala and McGee, 2008; Green et al., 2013; Holloszy, 1973; Holloszy and Coyle, 1984; Karlsson et al., 1972). Thus, the trained individual uses less total carbohydrate in his or her fuel mixture. These changes result in lower respiratory exchange ratio (RER) values (Figure 5.4A). Because glycogen is the primary source of fuel for high-intensity work, a larger supply of glycogen used less quickly enables an individual to participate in fairly intense activities at submaximal levels longer before fatigue occurs. Furthermore, trained individuals are more able to directly oxidize lactate produced from high-intensity work, which allows glycolytic tissues to support oxidative tissues by redistributing carbohydrate fuel and sparing glycogen within exercising muscles (Emhoff et al., 2013). On the other hand, sprint training can also increase the rate of glycogenolysis at higher levels of work, giving the exerciser a fast supply of energy when needed for short bursts of maximal or supramaximal activity.

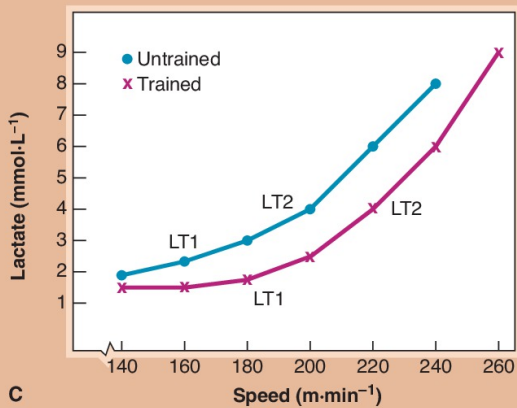
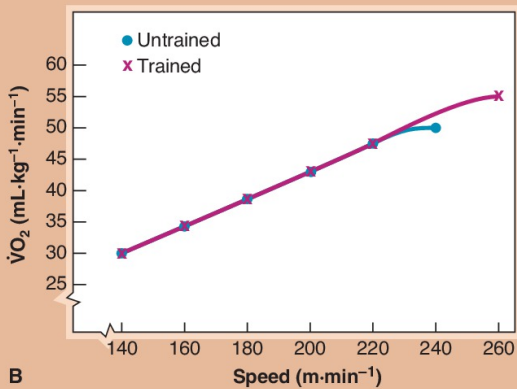
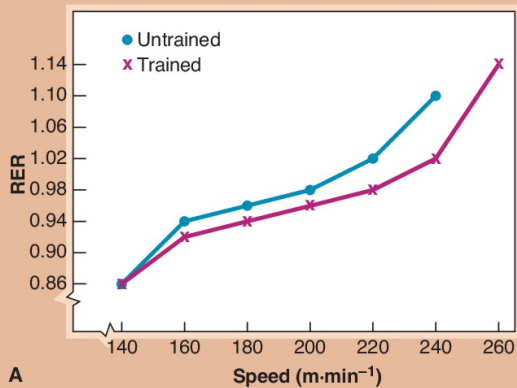




Figure 5.4 Metabolic Responses (Respiratory Exchange Ratio [RER] in panel A; Oxygen consumption VO_2 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, in panel B; lactate accumulation in panel C) of Endurance-Trained versus Untrained Individuals to Incremental Exercise to Maximum.

RER and lactate are lower in trained individuals; submaximal oxygen consumption is unchanged but maximal is increased in trained individuals. See animation, Oxygen Consumption, on Lippincott Connect.

Fat (2)

A trained individual can use his or her carbohydrate stores more slowly than can an untrained individual because of the changes that occur in fat metabolism. Both trained and untrained individuals have more than adequate stores of fat. However, the rate of free fatty acid oxidation is determined not by the storage amount but by the concentration of free fatty acids in the bloodstream and the capacity of the tissues to oxidize the fat. Training brings about several adaptations in fat metabolism, including the following:

1. Increased mobilization or release of free fatty acids from the adipose tissue
2. Increased level of plasma free fatty acids during submaximal exercise
3. Increased fat storage adjacent to the mitochondria within the muscles
4. Increased capacity to utilize fat at any given plasma concentration

The rise in the capacity of the muscle to oxidize lipids is larger than the rise in its capacity to oxidize glycogen. This in

conjunction with the lower plasma glucose uptake because of the decreased translocation of the GLUT-4 receptors, leads to the larger contribution of fat to energy production. The increased reliance on fat as a fuel is said to have a *glycogen-sparing effect* and is responsible for lowered RER values (**Figure 5.4A**) at the

same absolute and same relative (% $\dot{V}O_{2\max}$) work intensities. Because glycogen supplies last longer, fatigue is delayed allowing greater endurance at submaximal work levels. Both endurance and sprint training have glycogen-sparing effects (Abernethy et al., 1990; Gollnick and Hermansen, 1973; Holloszy, 1973; Holloszy and Coyle, 1984; Mougios, 2006; Perry et al., 2008; Scharhag-Rosenberger et al., 2010; Stisen et al., 2006; Talanian et al., 2007). Although the individuals who were tested in the accompanying Focus on Research box were children, this study illustrates the shift to a greater utilization of fat and lower utilization of carbohydrate at submaximal power outputs and a shift to a higher % $\dot{V}O_{2\max}$ before the crossover point where exercise intensity requires the use of more carbohydrate than fat.

FOCUS ON RESEARCH

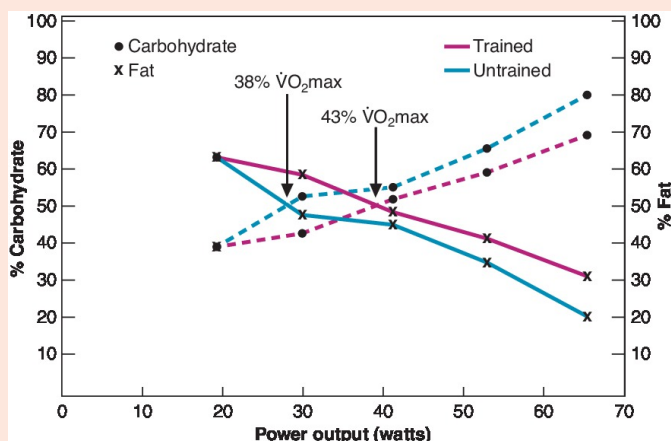
Substrate Training Adaptations in Children

As has been described, it is clear that in adults, the primary substrate utilized to fuel exercise depends on the modality, intensity, and duration of the activity as well as the individual's training status. The “*crossover*” concept states that at some point, as the intensity increases during incremental exercise, the predominant fuel source shifts from fat to carbohydrate. The crossover point is the power output at which this occurs.

This study by Duncan and Howley shows that the same processes occur in children. Twenty-three volunteer boys and girls (aged 7–12 years) were divided into a training group ($N = 10$) and a control group ($N = 13$). All were tested for $\dot{V}O_2$

peak on a cycle ergometer and then at five power outputs designed to elicit approximately 35%, 45%, 55%, 65%, and 75% of $\dot{V}O_2$ peak. RER values were determined by open-circuit spirometry for the five-stage submaximal test before and after 4 weeks of training. Training consisted of three 10-minute work bouts separated by 1–2 minutes of rest at roughly 50% $\dot{V}O_2$ peak, three times per week.

The results, presented in the accompanying graph, clearly show that as the intensity of the submaximal exercise increased, so did the percentage of carbohydrate utilized as fuel, both before and after the training. Furthermore, the crossover point was delayed or shifted to the right in the training group (data for the control group are not shown). This means that the trained children could work harder while using fat as the predominant fuel. Training apparently has the same carbohydrate-sparing benefit for children as for adults.



Source: Duncan, G. E., & E. I. Howley: Metabolic and perceptual responses to short-term cycle training in children. *Pediatric Exercises Science*. 10:110–122 (1998).

Protein (3)

Although proteins are the least important energy substrate,

changes do occur as a result of exercise training that enhance their role in metabolism. Adaptations in protein metabolism include an increased ability to utilize the branched chain amino acid leucine and an increased capacity to form alanine and release it from muscle cells. This increased production of alanine is accompanied by decreased levels in the plasma, probably indicating an accelerated removal for gluconeogenesis. In ultraendurance events, this increased gluconeogenesis effect is beneficial for maintaining blood glucose levels ([Abernethy et al., 1990](#); [Holloszy and Coyle, 1984](#); [Hood and Terjung, 1990](#)).

In addition to endurance training, resistance training influences protein metabolism as well. Physiological adaptations from resistance training result in decreased whole-body protein and leucine turnover and increase dietary nitrogen retention. These adaptations suggest that resistance training increases the efficiency of protein metabolism toward anabolic mechanisms. Therefore, as a result of resistance training, a larger percent of protein is shuttled toward muscle protein synthesis ([Hartman et al., 2006](#); [Moore et al., 2007](#)).

Enzyme Activity

The key to increasing the production of ATP is enzyme activity. Since every step in each metabolic pathway is catalyzed by a separate enzyme, the potential for this training adaptation to influence energy production is great. However, it appears that not all enzymes respond to the same training stimulus nor change to the same extent.

Glycolytic Enzymes (4)

The results of studies on the activity of the glycolytic enzymes have historically been contradictory, but a pattern is emerging. Glycolysis is involved in both the aerobic and anaerobic production of energy, and it may be that high-intensity training is required for some glycolytic enzymes to adapt, while others respond better to endurance training. Strength and sprint training appear to increase glycolytic enzyme activity ([Mougios, 2006](#)). These changes are generally less than the activity increases seen in aerobic enzymes, and the functional significance in terms of

actual performance remains questionable (Ross and Leveritt, 2001). Three key enzymes have shown significant training changes: glycogen phosphorylase, phosphofructokinase (PFK), and lactate dehydrogenase (LDH).

GLYCOGEN PHOSPHORYLASE Glycogen phosphorylase catalyzes the breakdown of glycogen stored in the muscle cells for use as fuel in glycolysis. An increase in this enzyme's activity has been found with high-intensity sprint training consisting of either short- (<10 seconds) or long- (>10 seconds) sprint intervals (Mougios, 2006; Ross and Leveritt, 2001). The ability to break down glycogen quickly is important in near-maximal, maximal, and supramaximal exercises.

PHOSPHOFRUCTOKINASE PFK is the rate-limiting enzyme of glycolysis. Results of endurance and sprint training studies are inconsistent but tend to suggest an increase in this enzyme activity with adequate levels of training especially consisting of long-duration sprint repetitions or a combination of long- and short-sprint efforts (Ross and Leveritt, 2001). Increased PFK activity leads to a faster and greater quantity of ATP being produced glycolytically.

LACTATE DEHYDROGENASE LDH catalyzes the conversion of pyruvate into lactate. It exists in several discrete forms, including a cardiac muscle form (LDH 1) that has a low affinity for pyruvate (thus making the formation of lactate less likely) and a skeletal muscle form (LDH 5) that has a high affinity for pyruvate (thus making the formation of lactate more likely). Endurance training tends to have two effects on LDH. It lowers the overall activity of LDH, and it causes a shift from the skeletal muscle to the cardiac muscle form. Thus, lactate is less likely to be produced in the skeletal muscle, and pyruvate is more likely to enter the mitochondria for use as an aerobic fuel. Both of these changes are beneficial to endurance performance (Abernethy et al., 1990; Holloszy and Coyle, 1984; MacRae et al., 1992; Sjodin et al., 1982). Strength and sprint training (consisting of both short- and long-sprint intervals) show opposite effects from endurance training, increasing the overall amount of LDH and favoring the LDH skeletal muscle form because of the changes in

fast-twitch muscle hypertrophy (Mougios, 2006; Ross and Leveritt, 2001).

Shuttles (5)

The hydrogen ions removed in glycolysis must be transported across the mitochondrial membrane by a shuttle because that membrane is impermeable to $\text{NADH} + \text{H}^+$. No training changes have been found in the glycerol-phosphate shuttle enzymes that predominate in the skeletal muscle. Conversely, training leads to large increases in the enzymes of the cardiac muscle's malate-aspartate shuttle in both the cytoplasm and the mitochondria, thus increasing shuttle activity. This increase enhances aerobic metabolism in the heart (Holloszy and Coyle, 1984).

Mitochondrial Enzymes (6)

Changes in the mitochondrial enzymes of beta-oxidation, the Krebs cycle, electron transport, and oxidative phosphorylation are coupled with changes in the mitochondria themselves. Both the size and the number of the mitochondria increase with endurance and high-intensity interval training. In adults, these mitochondrial adaptations of increased density and oxidative enzyme activity, known as *mitochondrial biogenesis*, can be on the magnitude of 30–100% within 4–6 weeks of starting training. The extent of the increase in mitochondrial content depends on the initial content. For instance, type IIX (fast twitch, glycolytic) fibers will increase mitochondrial content to a greater amount than type IIA (fast twitch, glycolytic-oxidative) fibers because they have lower mitochondrial content initially (Hood et al., 2012). As a result of this biogenesis, mitochondria occupy a proportionally larger share of the muscle fiber space. The sarcolemmal mitochondria are affected more than the interfibrillar mitochondria. The stimulus for these increases appears to be a contractile activity itself, rather than any external stimulus such as hormonal changes, since only those muscles directly involved in the exercise training show these changes. For example, runners have an increase in mitochondrial size and number only in the legs, whereas cross-country skiers have mitochondrial increases in both arms and legs.

Within limits, the extent of the augmentation in the mitochondria seems to be a function of the total amount of contractile activity. That is, the more contractions, the greater the change in the mitochondria. It does not seem to matter whether the increase in contractile activity is achieved by completing more contractions per unit of time (speed work) or by keeping the rate of contractions steady but increasing the duration (endurance training). Contractile activity simultaneously brings about an increase in mitochondrial protein levels, the production of reactive oxygen species (ROS; see the Focus on Application box), and modifies intracellular calcium levels. All three of these results appear to be important mechanisms in bringing about mitochondrial biogenesis. These responses start with the first acute bout of exercise, accumulate with more exercise bouts, and manifest themselves during recovery when the muscle is at rest. This emphasizes the importance of sufficient recovery time ([Hood et al., 2012](#)).

In general, as noted earlier, the physiological consequence of mitochondrial adaptations in trained distance runners favors a greater reliance on lipid, rather than carbohydrate metabolism. However, when individuals regularly train at high exercise intensity, mitochondrial adaptations seem to favor the CHO pathway over the fatty acid lipid pathway for the highest energy production through improvement in complex I (see [Figure 2.10](#)) rather than complexes II and IV ([Daussin et al., 2008](#)) of the electron transport system. Resistance training can also improve skeletal muscle mitochondria quality and quantity ([Porter et al., 2015](#)).

With larger mitochondria, more transport sites are available for the movement of pyruvate into the mitochondria. The enzymatic activity per unit of mitochondria appears to be the same in trained and untrained individuals; however, the greater mitochondrial protein content means an overall greater enzyme activity to utilize the pyruvate that has been transported there. Interestingly, although most mitochondrial enzymes increase in activity, not all do; nor is the rate of change the same for all. The myoglobin concentration in the muscles also increases with endurance training in the muscles directly involved in the activity. As a consequence, the rate of oxygen diffusion through the cytoplasm into the mitochondria increases, making more

oxygen available quickly. The overall effect of the increased enzyme activity, more oxygen, and the increased availability of pyruvate is an enhanced capacity to generate ATP by oxidative phosphorylation. This augmented capacity is more important for supplying energy for submaximal exercise than for maximal exercise (Abernethy et al., 1990; Gibala and McGee, 2008; Gollnick et al., 1986; Hawley and Spargo, 2007; Holloszy, 1973; Holloszy and Coyle, 1984; Mougios, 2006; Wibom et al., 1992).

Oxygen Utilization (7)

Maximal Oxygen Uptake

$\dot{V}O_{2\max}$ increases with training (Figure 5.4B) from both traditional endurance training and high-intensity interval training (Gibala and McGee, 2008; Gist et al., 2014; Kessler et al., 2012; Nalcakan, 2014; Weston et al., 2014). Even though this is a measure of the amount of oxygen utilized at the muscle level,

$\dot{V}O_{2\max}$ is determined more by the cardiovascular system's ability to deliver oxygen than by the muscle's ability to use it. Evidence for the subsidiary role of muscle in determining

$\dot{V}O_{2\max}$ includes the fact that individuals can have essentially the same mitochondrial content but very different $\dot{V}O_{2\max}$ values. Conversely, individuals with equivalent $\dot{V}O_{2\max}$ values can have quite different mitochondrial enzyme levels. Additionally, small training changes can occur in one factor (mitochondrial activity or $\dot{V}O_{2\max}$) without concomitant changes in the other—although, typically, both will increase. These differences probably explain why some runners are more economical (use less oxygen at a given pace than others do) and others possess a greater aerobic power (have a higher $\dot{V}O_{2\max}$) (Holloszy and Coyle, 1984).

Submaximal Oxygen Cost

The oxygen cost ($\dot{V}O_2$ in $\text{mL}\cdot\text{min}^{-1}$ or $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) of any given absolute submaximal workload is the same before and after

the training, assuming that no skill is involved where efficiency would change (Gollnick et al., 1986; Holloszy and Coyle, 1984) (Figure 5.4B). For example, if an individual has a smooth, coordinated front crawl stroke but has not participated in lap swimming, the oxygen cost of covering any given distance at a set pace will remain the same as this person trains. However, in an individual who is just learning the front crawl stroke, the oxygen cost could actually go down. It decreases not because of a change in the oxygen requirements but because extraneous inefficient movements that add to the oxygen cost are eliminated as skill is improved (Daniels et al., 1978; Ekblom et al., 1968; Gardner et al., 1989). The practical aspect of this is that applications such as the calculation of energy expenditure during walking, running, and cycling (Arenas et al., 2014), lists of MET levels (ACSM, 2022; Ainsworth et al., 2011), and caloric costs are deemed to be acceptable for use for adults of both sexes and all ages without regard to fitness level. This may or may not be an accurate assumption.

Running economy (RE) measured as the oxygen cost of running at any given submaximal velocity has been a highly controversial issue in terms of training response. Running economy depends both on the energy needed to move at a particular speed (external energy) and on the energy used to produce that energy (internal energy) and, as explained in Chapter 4, it represents a complex interplay of physiological and biomechanical factors. Although studies have shown mixed results (Beneke and Hütler, 2005), traditionally an improvement in running economy has been seen as a benefit of endurance training such that trained runners have better running economy than do both lesser trained and untrained runners (Barnes and Kilding, 2015). Endurance training-induced adaptations such as improved cardiovascular function, increased mitochondria, and oxidative enzymes, the ability of the muscles to store and release elastic energy, and optimization of motor recruitment patterns (enhanced neuromuscular characteristics) may all be related to improvements in running economy. Highly trained runners display more refined patterns of muscle recruitment than do lesser trained or untrained runners. In essence, they have become more physiologically capable and more skilled (Bonacci et al., 2009; Burgess and Lambert, 2010; Saunders et al., 2004).

However, there is no surefire way for improving running economy. A number of factors have been investigated to try and improve economy. High-intensity interval training has produced improvements in running economy, but the evidence is not strong (Barnes and Kilding, 2015; Gunnarsson et al., 2012; McCann and Higginson, 2008). Some studies suggest that economy may be improved by the addition of resistance training to the normal aerobic endurance regime of runners and cross-country skiers. Traditional weight training, movement-specific resistance training, and plyometric training have been shown to be effective (Barnes and Kilding, 2015). Results from stretching and flexibility studies are equivocal. An acute bout of stretching may improve running economy, but regular stretching over time appears to have no effect on economy (Barnes and Kilding, 2015). Again, it has been speculated that a combination of improved running mechanics and neuromuscular function results in a decreased oxygen consumption, but many questions remain unanswered (Bonacci et al., 2009; Millet et al., 2002; Saunders et al., 2006; Spurrs et al., 2003; Turner et al., 2003).

If an individual increases his or her $\dot{V}O_{2\max}$ and yet the oxygen cost of any given (absolute) workload remains the same, then the % $\dot{V}O_{2\max}$ at which that individual is doing the given workload will go down. The task will be relatively easier for the individual, and endurance performance will be greatly enhanced.

Decreased efficiency or economy can also occur. If so, it should be interpreted as a symptom of overtraining (Fry et al., 1991).

Oxygen Deficit and Drift

The oxygen deficit at the onset of activity is smaller, but is not eliminated, in trained individuals. The primary reason for this reduction is that oxidative phosphorylation is activated sooner because of the greater number of mitochondria that are sensitive to low levels of ADP and Pi. This result is advantageous to the exerciser because fewer H⁺ and less lactate are produced and less creatine phosphate depleted (Holloszy, 1973; Holloszy and Coyle, 1984; Krstrup et al., 2004).

The magnitude of oxygen drift is also less after training. This

change may be caused by concomitant reductions in epinephrine, norepinephrine, lactate, and body temperature rise during any given submaximal workload (Casaburi et al., 1987; Hagberg et al., 1978).

Excess Postexercise Oxygen Consumption

Excess postexercise oxygen consumption (EPOC) is a function of the intensity and duration of the previous exercise bout. Aerobic endurance training has been shown (Sedlock et al., 2010) to significantly decrease the magnitude of EPOC after exercise at the same absolute submaximal intensity but to result in no change after exercise of the same relative submaximal intensity. This is because the same (pretraining) absolute intensity is a lower relative intensity after training. The mechanisms appear to be the same as for the changes in oxygen drift. These factors include training-induced decreases in blood lactate concentration, body temperature, epinephrine-mediated glucose production, and insulin-mediated glucose uptake. The training-induced shift toward greater fat utilization during exercise continues into the postexercise period.

Lactate Accumulation (8)

Lactate is produced when the hydrogen atoms carried on $\text{NADH} + \text{H}^+$ are transferred to pyruvate in a reaction catalyzed by lactate dehydrogenase (LDH). Lactate accumulates when the rate of production exceeds the rate of removal. Endurance exercise training increases the capacities for both lactate production and lactate removal. It also increases lactate clearance rate for both absolute and relative workloads (Messonnier et al., 2013). Although the capacity for production is higher, this does not mean that capacity is reached at all workloads. Indeed, training results in lower lactate production at any given absolute workload and as such less lactate is accumulated in a trained individual than in an untrained individual.

Factors that lead to a decrease in lactate production following training include the following:

1. Fuel shifts

2. Enzyme activity changes
3. Blunted neurohormonal responses

Pyruvate is the end product of carbohydrate metabolism (glycolysis). Less carbohydrate is utilized at an absolute submaximal workload after training; therefore, less pyruvate is available for conversion into lactate. At the same time, pyruvate dehydrogenase activity increases following training, causing more pyruvate to be converted to acetyl CoA. LDH enzyme shifts from the skeletal muscle form, which favors lactate production, to the cardiac muscle form, which has a lower affinity for pyruvate. In addition, following training, glycolysis is inhibited by several factors, two of which relate to the increased utilization of fat during submaximal exercise. The first is a high concentration of free fatty acid in the cytoplasm; the second is a high level of citrate (the first product in the Krebs cycle). Both factors cause the rate-limiting enzyme PFK to slow down glycolysis and thus decrease the possible production of lactate. Finally, a smaller increase in the concentration of epinephrine and norepinephrine has been found at the same absolute and relative workloads in trained individuals. This decreased sympathetic stimulation may also decrease the activation of glycogenolysis and the potential production of lactate ([Gollnick et al., 1986](#); [Holloszy and Coyle, 1984](#); [Messonnier et al., 2006](#)).

FOCUS ON APPLICATION

Oxygen-Free Radicals, Exercise, Exercise Training Adaptations, and Antioxidant Supplements

Cancer, atherosclerosis, cataracts, Alzheimer's disease, diabetes, loss of memory, Parkinson's disease, and aging may all, in part, be caused by free radical damage ([Keith, 1999](#)). What are free radicals? How can they cause so many different problems? What is the link between oxygenfree radicals,

exercise, and exercise training adaptations? Should antioxidant supplements be taken by individuals involved in exercise training?

Under normal conditions, electrons orbiting in the shells of a molecule are in pairs. If a single electron is added or removed, instability occurs. The resulting structure is called a *free radical*. Free radicals have a drive to return to a balanced stable state and attempt to do so by taking an electron from, giving an electron to, or sharing an electron with another atom. *Reactive oxygen species (ROS)* contain free radicals and reactive forms of oxygen.

ROS can be produced from sources originating outside the body, such as x-rays, UV rays in sunlight, altitude, air pollutants (ozone and nitric oxide in car exhaust), cigarette smoke, toxic chemicals (some pesticides), and physical injury (from contact sports or concussions). They may also result from sources within the body, specifically as part of normal immune function or as a normal by-product of the production of energy (Finaud et al., 2006; Keith, 1999).

Acute exercise produces free radicals. Production can occur at various sites in muscle cells: the mitochondrion, sarcoplasmic reticulum, transverse tubules, and cytosol. Surprisingly, mitochondria from fast twitch type II muscle fibers promote higher levels of ROS production than do those from slow twitch (type I) fibers. Mitochondrial-derived ROS are produced when electrons traveling through the electron transport chain of Stage IV are leaked at complexes I and III. The higher the rate of metabolism (as in moving from rest to submaximal to maximal exercise), the more free radicals are produced. Approximately 0.2–0.8% of the oxygen consumed is converted to free radicals (Hood et al., 2012; Powers et al., 2011). Anaerobic energy production provides an abundance of hydrogen ions that can react with an oxygen-free radical to form an ROS, such as hydrogen peroxide, H_2O_2 . Hypoxia leads to freeing of metals (Fe, Cu, and Mg) that are needed to catalyze free radical production. Exercise-induced hyperthermia may trigger free radical proliferation. Any damage to muscle fibers leads to increased immune response and free radical production (Finaud et al., 2006). Exercises

that result in alterations of blood flow and oxygen supply such as weight lifting trigger free radical production.

ROS have numerous negative effects. If the production of free radicals is continuous and high and exceeds that of components called *antioxidants* that suppress them and their harmful effects, *oxidative stress* occurs. Often, a chain reaction occurs that results in damage to lipids (especially the lipid bilayer of cell membranes), proteins (in enzymes, immune cells, joints, and muscles), and DNA (breaking strands or shifting bases, thus influencing the genetic code) and, in excess, decreases muscular contractile force or even cellular death. These changes ultimately can lead minimally to muscular fatigue or more seriously to the diseases listed earlier ([Alessio and Blasi, 1997](#); [Finaud et al., 2006](#); [Gross et al., 2011](#); [Hood et al., 2012](#); [Jenkins, 1993](#); [Keith, 1999](#)).

Conversely, ROS have several positive roles in the body including involvement in the immune system, cellular signaling, enzyme activation, facilitation of glycogen replenishment, and muscle fiber contractile force. A single acute bout of exercise that produces a small increase in ROS plays an essential role in the regulation of cell signaling pathways that ultimately lead to muscular adaptation ([Powers et al., 2011](#)). Among the cellular signaling done by ROS after aerobic endurance training are the pathways that enhance mitochondrial biogenesis, capillarization, muscle and heart hypertrophy, and glucose transport ability. They can act as a vasodilator and optimize both blood flow and the velocity of blood flow ([Gross et al., 2011](#)).

Despite the increased production of free radicals resulting from exercise, it is unlikely that exercise results in substantial damages to a normal healthy individual. The body has a number of natural (endogenous) defenses, and antioxidants ingested in food provide additional (exogenous) defenses. Each cell contains a variety of antioxidant scavenger enzymes, predominantly superoxide dismutase, catalase, and glutathione peroxidase. Antioxidant vitamins, minerals, and phytochemicals include vitamin E, vitamin C, beta carotene (precursor of vitamin A), selenium, coenzyme Q (ubiquinone), and flavonoids.

The relationship between exercise and oxidative stress appears to be an inverted U, based on intensity and total volume. That is, there are no benefits with a sedentary lifestyle. Single acute bouts of light to moderate and strenuous physical activity both have been shown to selectively enhance the antioxidant enzymes along with ROS. However, while light to moderate activity reduces oxidative stress, strenuous exercise increases oxidative stress. If too much strenuous exercise results in overtraining, the benefits of appropriate training (decreased ROS, increased antioxidants, increased activity of antioxidant enzymes, decreased oxidative damage, increased oxidative damage repair, and increased resistance to oxidative stress) are reversed, and instead of the exercise protecting against oxidative stress and being beneficial to health, the resistance is decreased ([Pingitore et al., 2015](#)).

Most exercise training studies have shown increased antioxidant levels following both aerobic endurance exercise and dynamic resistance exercise. Part of the training adaptation may be due to increases in the cytochromes in electron transport, which reduces electron leakage ([Alessio and Blasi, 1997](#); American College of Sports Medicine (ACSM), American Dietetic Association, Dietitians of Canada, 2009; [Powers et al., 2014](#)).

Several consensus reports ([ACSM et al., 2009](#); [Kreider et al., 2004](#); [Pendergast et al., 2011](#); [Pingitore et al., 2015](#)) do not support the idea that ingested supplementation of vitamins A, C, or E improve performance, delay fatigue, or protect against muscle damage in adequately nourished individuals. Indeed, exercise studies have provided evidence that high supplementation of antioxidants may actually be counterproductive both acutely and in terms of blunted training adaptations ([Peternelj and Coombes, 2011](#); [Powers et al., 2011](#)). Acute exercise responses to antioxidant supplementation indicate that the elevated oxidative defenses via the normal upregulation of the antioxidant enzymes may be suppressed. For example, [Knez et al. \(2007\)](#) reported significantly greater oxidative damage in half and full ironman triathletes who took antioxidant supplements than in those who did not. Chronic training adaptations with

antioxidant supplementation include evidence of suppressed capillarization and mitochondrial biogenesis ([Gross et al., 2011](#)).

However, this is not to say that all antioxidant supplementation should be avoided. Individual athletes and circumstances must be taken into account. Athletes at greatest risk for poor antioxidant intakes include those doing high-intensity training, following a low-fat diet, restricting energy intake, or limiting dietary intakes of fruits, vegetables, and whole grains. In addition, athletes training at high altitude (where free radical production is intensified and internal defenses are weakened by hypoxia) could benefit from supplementation. All individuals should make sure that their diets contain large amounts of antioxidant-rich foods, primarily fruits and vegetables. Prunes, raisins, blueberries, strawberries, oranges, spinach, broccoli, beets, onions, corn, eggplant, nuts, and whole grains are particularly beneficial. If vitamin supplements are ingested, they should not exceed recommended upper limits such as 1,000 mg of vitamin C daily or 400 IU per day for vitamin E. More is not better, and in excessive amounts, antioxidants can actually act as pro-oxidants with potential negative effects. Supplementation with coenzyme Q is not recommended at all. The search for the optimal balance between the beneficial and harmful effects of ROS and antioxidant supplementation continues ([ACSM et al., 2009](#); [Finaud et al., 2006](#); [Gross et al., 2011](#); [Jenkins, 1993](#); [Keith, 1999](#); [McGinley et al., 2009](#); [Pingitore et al., 2015](#); [Stear et al., 2010](#)).



Sources: ACSM et al. (2009); Alessio and Blasi (1997); Finaud et al. (2006); Jenkins (1993); Keith (1999); Knez et al. (2007); Kreider et al. (2004); McGinley et al. (2009); Pendergast et al. (2011); Peternelj and Coombes (2011); Pingitore et al. (2015); Stear et al. (2010).

Several other changes lead to higher removal and rate of clearance in trained individuals. These can be classified into two main factors:

1. Enhanced lactate transport
2. Enhanced lactate oxidation

Lactate transport is enhanced by a combination of increased substrate affinity (the ability of the lactate to bind to the transporter), increased intrinsic activity of the enzymes involved, and increased density of the mitochondrial membrane and cell membrane lactate transporters (Juel et al., 2004; Thomas et al., 2005). Although some studies have reported increases in both MCT1 and MCT4 transporters, it appears to be MCT1 that primarily changes with training. These changes have been seen after endurance, strength, all-out sprint, and interval training (Brooks et al., 2008; Gunnarsson et al., 2012; Thomas et al., 2012). At the same time, mitochondrial size, number, and

enzyme concentrations are increased. Taken together, these changes enable muscle cells to increase both the extracellular and intracellular lactate shuttle mechanisms. There is an overall uptake of lactate by muscles, and as a result, more lactate can be oxidized more rapidly during exercise. Concomitantly, blood flow to the liver is enhanced, which aids in overall lactate removal (Bonen, 2000; Brooks, 2000; Brooks et al., 2000; Gladden, 2000; Pilegaard et al., 1994). Training at the velocity of maximal lactate steady-state (MLSSv) has been shown to increase both time to exhaustion at MLSSv and $\dot{V}O_{2\max}$ without modifying either [La⁻] or reliance on carbohydrate fuel indicating an increased lactate clearance (Billat et al., 2004). The result is a decreased concentration of lactate in the muscles and blood at the same relative workload ($\dot{V}O_{2\max}$) after training.

As a consequence of the decrease in production and increase in removal after training, a higher workload (both in absolute and relative terms) is required to reach lactate levels in the 2- to 4-mmol·L⁻¹ range (Figure 5.4C). This means that an individual can exercise at a higher relative intensity for a given period of time and yet delay the onset of fatigue because the lactate thresholds (LT1 and LT2) have been raised (Allen et al., 1985; Henritze et al., 1985; Holloszy and Coyle, 1984; Londeree, 1997; Skinner and Morgan, 1985; Williams et al., 1967; Yoshida et al., 1982). There appears to be a dose-response relationship between the frequency of interval training and the magnitude of lactate threshold improvement (expressed as % $\dot{V}O_{2\max}$) (Dalleck et al., 2010). At maximal aerobic/anaerobic endurance exercise, the level of lactate accumulation is higher as a result of training. The higher level probably results from the greater glycogen stores and increased activity of some of the glycolytic enzymes other than LDH (Abernethy et al., 1990; Gollnick et al., 1986). It may also be more a psychological than a physiological adaptation, in that the trained individual is more motivated and can better tolerate the pain caused by lactate and H⁺ (Galbo, 1983) when working at a higher absolute load.

Resistance training has been shown to affect lactate response to both weight-lifting exercise and dynamic aerobic exercise. That is, after resistance training, more work can be done before achieving the same accumulation of lactate that occurred before

training; less lactate is accumulated at the same absolute workload; and at the same relative workload, the accumulation of lactate is unchanged (Greco et al., 2011; Marcinik et al., 1991; Reynolds et al., 1997; Yamamoto et al., 2008).

ATP Production, Storage, and Turnover

ATP-PC (9)

Although exercise training increases the potential for the production of larger quantities of ATP by oxidative phosphorylation, it does not change the efficiency of converting fuel to ATP or ATP to work. Thirty-two actual ATP molecules are still produced from glucose in skeletal muscle, and the potential energy per mole of ATP is still between 7 and 12 kcal (Abernethy et al., 1990; Gollnick and Hermansen, 1973; Gollnick et al., 1986; Holloszy, 1973; Karlsson et al., 1972; Skinner and Morgan, 1985).

However, the amount of ATP and PC stored in the resting muscle is higher in trained than in untrained individuals, especially if muscle mass increases. Whether this amount is large enough to markedly increase anaerobic capacity is questionable. The resting PC/ATP ratio does not differ among sprint-trained runners, endurance-trained runners, and untrained individuals (Johansen and Quistorff, 2003). At the same absolute workload, there is less depletion of the PC and degradation of ATP levels after training. At the same relative workload, PC depletion and ATP degradation do not change with training. However, the activity of the enzymes responsible for the breakdown of ATP to ADP and the regeneration of ADP and ATP increase. Therefore, the rate of turnover of ATP and PC increases and may be as much as double that of untrained individuals in both sprint- and endurance-trained runners (Johansen and Quistorff, 2003). Taken together, the ATP-PC-LA changes indicate an increased anaerobic power and capacity with sprint-type training (Medbø and Burgers, 1990). Values for the ATP-PC, LA, and O₂ systems are presented in **Table 5.3**. The values for the untrained were previously presented in **Table 3.1** but are now contrasted with trained males. The LA system changes much more with training than does the ATP-PC system, but the greatest change is in the O₂

system (Bouchard et al., 1982, 1991).

TABLE 5.3 Estimated Maximal Power and Capacity for Untrained (UT) and Trained (TR) Males

| System | Power | | | | Capacity | | | |
|--|------------------------|-------|----------------------|---------|-----------|---------------|-------------|---------------|
| | kcal·min ⁻¹ | | kJ·min ⁻¹ | | Kcal | | kJ | |
| | UT | TR | UT | TR | UT | TR | UT | TR |
| Phosphagens (ATP-PC) | 72 | 96 | 300 | 400 | 11 | 13 | 45 | 55 |
| Anaerobic glycolysis (LA) | 36 | 60 | 150 | 250 | 48 | 72 | 200 | 300 |
| Aerobic glycolysis plus Krebs cycle plus ETS/OP (O ₂) | 7–19 | 32–37 | 30–80 | 135–155 | 360–1,270 | 10,770–19,140 | 1,500–5,300 | 45,000–80,000 |

Source: Modified from Bouchard et al. (1982, 1991).

Work (Power) Output

Work (power) output—measured as watts or kilocalories per kilogram of body weight on a bicycle test such as the 10- or 30-second Wingate Anaerobic Test and/or a 90-second test—improves with training. This is evidenced by higher scores of athletes than nonathletes and by higher posttraining than pretraining scores in all populations. Furthermore, sprint- or power-type athletes typically have higher anaerobic values and greater adaptations than do endurance-type athletes. Elite sprinters and power athletes score higher than do less successful competitors (Bar-Or, 1987; Beld et al., 1989; Horswill et al., 1989; Patton and Duggan, 1987).

Aerobically, a trained individual can continue any given submaximal workload longer than can an untrained individual. The trained individual can also accomplish more total work and a higher absolute maximum than can an untrained individual. Overall, the trained individual has a metabolic system capable of supporting enhanced performance, both at submaximal and at maximal levels. These changes, summarized in Table 5.4, depend on the type of training used.

TABLE 5.4 Metabolic Training Adaptations

1. Fuel Supply
 - a. Carbohydrate
 1. ↑ GLUT-4 transporter number and concentration; ↓ exercise-induced translocation
 2. ↓ Glucose utilization
 3. ↑ Muscle and liver glycogen reserves
 4. ↓ Rate of muscle and liver glycogen depletion at absolute submaximal loads, that is, glycogen sparing
 5. ↑ Velocity of glycogenolysis at maximal work
 - b. Fat
 1. ↑ Mobilization, transportation, and beta-oxidation of free fatty acids
 2. ↑ Fat storage adjacent to mitochondria
 3. ↑ Utilization of fat as fuel at the same absolute and the same relative workloads
 - c. Protein
 1. ↑ Ability to utilize the BCAA leucine as fuel
 2. ↑ Gluconeogenesis from alanine
2. Enzyme Activity
 - a. ↑ Selected glycolytic enzyme activity: glycogen phosphorylase and probably PFK
 - b. ↓ LDH activity with some conversion from the skeletal muscle to cardiac muscle form with endurance training but ↑ with strength/sprint training
 - c. ↑ Activity of the malate-aspartate shuttle enzymes but not the glycerol-phosphate shuttle enzymes
 - d. ↑ Number and size of mitochondria (mitochondrial biogenesis)
 - e. ↑ Activity of most, but not all, of the enzymes of beta-oxidation, the Krebs cycle, electron transport, and oxidative phosphorylation due to greater mitochondrial protein amount
3. O₂ Utilization
 - a. ↑ $\dot{V}O_2$ max with aerobic endurance and high-intensity interval training but not with dynamic resistance training
 - b. = $\dot{V}O_2$ cost at absolute submaximal workload unless neuromuscular skill aspects improve; → ↑ running economy
 - c. ↑ Myoglobin concentration
 - d. ↓ Oxygen deficit
 - e. ↓ Oxygen drift
 - f. ↓ EPOC at same absolute submaximal workload
4. Lactate Accumulation
 - a. ↑ MCT1 lactate transporters
 - b. ↑ Intracellular and extracellular lactate shuttle activity
 - c. ↓ La^- accumulation at the same absolute workload and % $\dot{V}O_2$ max relative intensity for endurance activity
 - d. ↓ La^- accumulation at the same absolute workload but = La^- accumulation at the same relative intensity for resistance exercise
 - e. ↑ Workload to achieve lactate thresholds
 - f. ↑ Velocity at maximal lactate steady state (MLSSv) and time to exhaustion at MLSSv
 - g. ↑ $[La^-]$ at maximum
5. ATP Productions, Storage, and Turnover
 - a. = ATP from gram of precursor fuel substrate
 - b. ↑ ATP-PC storage
 - c. ↓ Depletion of PC and degradation of ATP at the same absolute workload
 - d. = Depletion of PC and degradation of ATP at the same relative workload
 - e. ↑ ATP-PC turnover

Note: ↑, increase; ↓, decrease; =, no change.

Complete the [Check Your Comprehension 3—Case Study 1](#) box to test your knowledge of metabolic training adaptations.

CHECK YOUR COMPREHENSION 3—CASE STUDY

Listed in the accompanying table are values obtained from a 25-year-old male, obtained at rest and during an incremental treadmill test to maximum, before (PRE) and after (POST) a 6-month endurance training program that included both continuous and interval sessions. Your task is to make two columns labeled PRE training and POST training and put the value for each variable in the appropriate column thus demonstrating your understanding of the selected training

adaptations. In your report to this individual, would you tell him he is more fit now or not?

| | |
|---|-----------------------|
| $\dot{V}O_2\text{max}$ mL·kg ⁻¹ ·min ⁻¹ | 48.3; 42.6 |
| RERmax | 1.11; 1.06 |
| RER at 6 mph (160 m·min ⁻¹) | 0.84; 0.93 |
| [La]max mmol·L ⁻¹ | 12; 13.5 |
| [La] mmol·L ⁻¹ at 6 mph (160 m·min ⁻¹) | 2.3; 2.8 |
| $\dot{V}O_2$ mL·kg ⁻¹ ·min ⁻¹ at 6 mph (160 m·min ⁻¹) | 34; 35 |
| Resting ATP mM·g wet muscle ⁻¹ | 5.5; 3.5 |
| Resting PC mM·g wet muscle ⁻¹ | 10; 15 |
| Resting glycogen mM·g wet muscle ⁻¹ | 80; 111 |
| Number of mitochondria mmol ³ | 1.08; 0.45 |
| Glycogen phosphorylase mM·g wet muscle ⁻¹ | 5; 7 |
| Running velocity at LT2 mph (m·min ⁻¹) | 7 (190); 8.5 (230) |

The Influence of Age and Sex on Metabolic Training Adaptations

With the exception of $\dot{V}O_2\text{max}$, there are few research data on most of the metabolic variables across the age spectrum. Some data in children suggest that children lose exercise adaptations more slowly than adults due to potential neural adaptations (Vassilis et al., 2019), while others observed that training and detraining changes in children, adolescents, and older adults are similar in direction and magnitude to changes in adults in the 20- to 50-year range. This is especially true when changes are considered relative to baseline values (i.e., as a percentage of change) rather than as absolutes (Adeniran and Toriola, 1988; Bar-Or, 1983; Clarke, 1977; Eriksson, 1972; Gaisl and Wiesspeiner, 1986; Massicotte and MacNab, 1974; McNarry and

Jones, 2014; Rotstein et al., 1986; Rowland, 1990).

Adaptations in Children and Adolescents

The one remarkable exception to the above generalization is the uncoupling of endurance performance changes and $\dot{V}O_{2\max}$ changes in youth such that children exhibit a limited capacity to improve $\dot{V}O_{2\max}$, despite being able to improve endurance exercise performance. That is, the same exercise training program that would generate a 15–20% improvement in adults in $\dot{V}O_{2\max}$ typically results in only a 5% increase in prepubertal children of both sexes. The question, of course, is why. There does not appear to be a “maturational threshold” or “trigger point” that coincides with puberty and its associated hormonal changes that is responsible for the improvements in $\dot{V}O_{2\max}$ (Armstrong, 2015; McNarry and Jones, 2014).

Rowland (2009) has proposed the *crowded cell hypothesis*. In the skeletal muscle cell, there is only so much space and that space is shared by the contractile apparatus, the sarcoplasmic reticulum that regulates each contraction-relaxation cycle, and the mitochondria that provides the energy. Adaptations of cellular elements to training are limited by cell space. The space allocated to the contractile apparatus and sarcoplasmic reticulum cannot be compromised. Children have less space for expansion than do adults because before training, their muscle fibers have a higher mitochondrial density ($\sim +30\text{--}50\%$) and oxidative enzyme activity than do adults. The limited change in mitochondria results in $\dot{V}O_{2\max}$ increases with endurance training that are typically a lower percentage in children than adults. Further research is needed to confirm or modify this hypothesis.

Male-Female Differences in Adaptations

There are minimal data on metabolic adaptations in females of all

ages, again with the exception of $\dot{V}O_{2\max}$ (Shepard, 1978; [Thlusty, 1969](#); [Wells, 1991](#)). From the available studies, it appears that males and females respond to the same training with the same adaptations ([Johnson et al., 2010](#); [Slade et al., 2002](#); [Wells, 1991](#); [Weltman et al., 1978](#)). However, some research does suggest there is an adipose tissue-sparing mechanism in females with aerobic training, potentially relating to the increased need for adipose tissue in women than in men ([Andersson et al., 1991](#)). Sex differences in the metabolic variables are not obliterated in equally trained males and females, but both sexes are trainable and probably to the same extent.

Adaptations in Older Adults

In general, data show that older adults experience similar metabolic adaptations compared to younger adults as a result of training. However, there are some discrepancies, and because the older individuals generally have impaired baseline values, the magnitude of their adaptation is often less than younger individuals ([Gurd et al., 2008](#)).

Changes in substrate utilization per se with aerobic training do not appear to be the age dependent. GLUT 4 protein content increases thereby improving glucose transport ([Kim et al., 2004](#); [Short et al., 2003](#)) in both older and younger individuals. Fat metabolism has been shown to increase in older females after strength training ([Treuth et al., 1995](#)) and in older males and females with endurance training ([Sial et al., 1998](#)), along with a corresponding decrease in reliance on carbohydrate, although there may be a relationship to fat intake in pretraining. In one study, fat utilization increased in older individuals with a high pretraining RER (indicating high carbohydrate utilization) but decreased in individuals with a low pretraining RER (indicating an already relatively high reliance on fat utilization ([Meijer et al., 2000](#)).

There continues to be a great deal of interest on aging, training, and oxidative/mitochondrial enzymes. However, research results are equivocal. Many studies have observed no meaningful impairments, while numerous others have found age-

related deficiencies including decreases in complexes I, II, III, and IV of the electron transport chain, a decrease in oxidative phosphorylation capacity, low mitochondrial enzyme activity, and an excessive production of reactive oxygen species (ROS). These changes can lead to mitochondrial dysfunction and, hence, the production of ATP. Conversely, there seems to be almost universal agreement on the positive effects of exercise training on many of these same mitochondrial markers and functions with no difference between older and younger males or females. Endurance training has been shown to increase protein content and volume, as well as oxidative, Krebs cycle and Electron Transport chain enzyme activity in older individuals. Similarly, resistance exercise training has resulted in an increase in mitochondrial volume, as well as mitochondrial ATP production, but apparently not mitochondrial complex (I-V) protein abundance or citrate synthase activity. Therefore, both aerobic endurance and resistance exercise training can improve some aspects of mitochondrial function in the healthy older individual (Berg et al., 2020; Cobley et al., 2015; Coggan et al., 1993; Fritzen et al., 2020; Konopka et al., 2014; Lanza et al., 2008; Short et al., 2003, 2005; Ziaaladini et al., 2017).

FOCUS ON RESEARCH

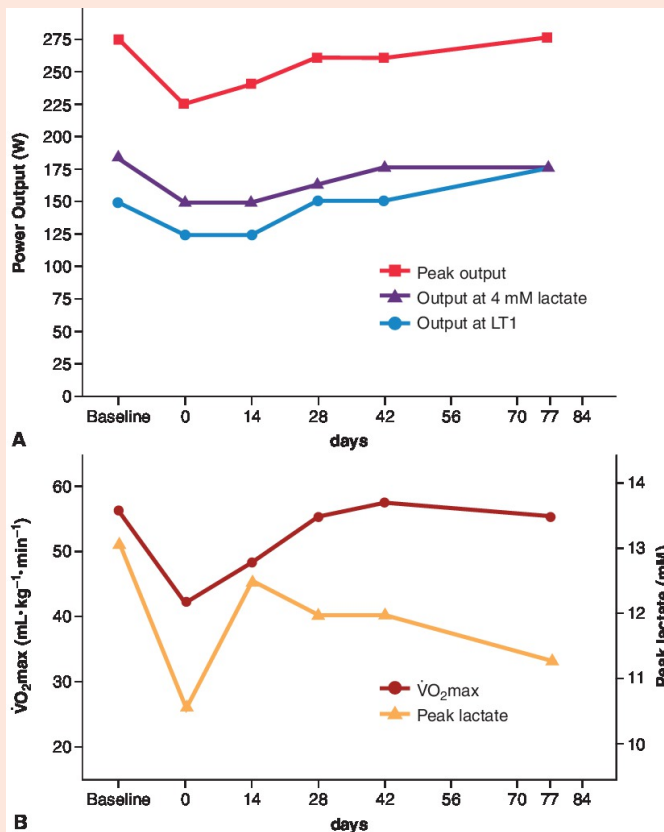
Reversibility and Retraining

When reversibility occurs as in the case of an injury, retraining does not occur as rapidly as the detraining and is not easier nor more rapid than the initial training (Wilmore and Costill, 1988). This case study of detraining reversibility and retraining was conducted on a 49.5-year-old female elite competitive cyclist. Two days after undergoing testing for a research project, the cyclist sustained a right clavicular fracture during a criterion race. She continued a modified training regime until 22 days after the injury, when she developed loss of motion in the shoulder and pain and numbness in the hand. Surgery was performed 26 days after the injury for brachial plexus impingement. Thus, the

detraining period began approximately 4 weeks after the injury. Retraining began the 32nd day after surgery (0 on the X-axis). Results for her metabolic variables are presented in the accompanying graphs. The baseline represents the values obtained 2 days before the injury. Retesting was done approximately every 2 weeks (0, 14, 28, 42, 56, and 70 days) for 6 weeks and then again at week 11 of retraining (77 days).

VO_2max decreased approximately 25% during detraining, but by week 11, it was within 2 $\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$ of preinjury values. The improvement was steady over the first 6 weeks of retraining.

Power output decreased 18.2% at peak, 16.7% at LT1, and 18.9% at 4 mM lactate. Peak lactate decreased by a comparable 19.1%. Although peak power output increased during the first 2 weeks, power output at LT1 and 4 mM lactate did not. This probably occurred because high-intensity work was not included in the initial retraining weeks as is appropriate given the progression principle. By week 11, peak power output and power output at LT1 had returned to baseline values. However, at that point, neither peak power output at 4 mM nor peak lactate had regained preinjury levels.



Source: Nichols, J. F., D. Robinson, D. Douglass, & J. Anthony: Retraining of a competitive masterathlete following traumatic injury: A case study. *Medicine & Science in Sports & Exercise*. 32(6):1037–1042 (2000).

Most studies on oxygen utilization have concentrated on maximal oxygen consumption. Maximal oxygen consumption declines with age. The ability to improve $\dot{V}O_{2\max}$ with aerobic training, however, remains strong in both male and female elderly although the changes are typically lower in older than younger individuals (Fritzen et al., 2020; Konopka et al., 2014; Montano et al., 2018; Sant'Ana et al., 2020; Short et al., 2003, 2004). Submaximal economy and endurance performance

also improve as a result of training in the elderly (Fritzen et al., 2020; Gomeñuka et al., 2020).

Almost no information is available regarding the impact of aerobic, anaerobic sprint, or resistance training on lactate accumulation or ATP production, storage, and turnover in older males or females. Despite their training regimes, Masters athletes still demonstrate age-related declines in anaerobic performances, some of which may be attributed to changes in enzyme activity and decreased lactate production. How much, if any, of these changes are minimized by specific high-intensity resistance or sprint training in these older athletes is unknown (Reaburn and Dascombe, 2009). One study (Berg et al., 2018) that investigated the impact of maximal strength training in older individuals reported a shift toward a more glycolytic muscle type. This shift, in turn, resulted in a greater muscle force production during sustained maximal contraction and was supported by an increased rate of ATP synthesis from anaerobic glycolysis. There was no significant alteration in the ATP cost of contraction.

Summary

1. The most important considerations for applying each training principle to achieve metabolic adaptations are as follows:
 - a. For specificity, match the energy system of the activity.
 - b. For overload, manipulate time and distance or lactate level.
 - c. For rest/recovery/adaptation, alternate hard and easy days.
 - d. For progression, re-overload if additional improvement is desired.
 - e. or individualization, evaluate the individual according to the demands of the activity and develop a periodization training sequence, system, and load on the basis of this evaluation.
 - f. For maintenance, emphasize intensity.

- g. For retrogression, plateau, and reversibility, evaluate the training adaptations and modify as indicated.
 - h. For warm-up and cooldown, include activities that will actually elevate and reduce body temperature, respectively.
2. Properly prescribed training programs bring about positive adaptations in fuel supply, enzyme activity, oxygen utilization, lactate accumulation, and ATP production, storage, and turnover.
 3. Available evidence indicated that training changes in children, adolescents, and older individuals are similar in direction and magnitude to those in young-middle-aged adults with the possible exception of $\dot{V}O_{2\max}$.
 4. Both males and females respond to training with the same adaptations, but these do not obliterate the relative differences between the sexes.
 5. A reversal of training adaptations can occur in days to weeks for the metabolic variables but appears to be faster for endurance performance than strength performance. Retraining does not occur as rapidly as does detraining and is not easier or more rapid than initial training.

Review Questions

1. Name and briefly describe the eight training principles. Select a sport or fitness activity and show how each of the training principles can be specifically applied to that activity.
2. Describe and explain the metabolic adaptations to exercise training for each of the following factors:
 - a. Substrate or fuel supply
 - b. Enzyme activity
 - c. Oxygen utilization
 - d. Lactate accumulation
 - e. ATP production, storage, and turnover

3. Describe and explain the effect of detraining on each of the following factors:
- a. Substrate or fuel supply
 - b. Enzyme activity
 - c. Oxygen utilization
 - d. Lactate accumulation
 - e. ATP production, storage, and turnover

Literature Search

1. We discussed metabolic adaptations in this chapter. Metabolic adaptation is important to understand physiological changes that occur as a result of exercise training. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.
 - a. Search metabolic adaptation. This will yield a huge selection of articles.
 - b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Metabolic adaptation and resistance training
 - ii. Metabolic adaptation and aerobic training
 - iii. Metabolic adaptation and age
 - iv. Metabolic adaptation and sex
 - v. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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6 Nutrition for Fitness and Athletics



CHAPTER OUTLINE

Introduction

Nutrition for Training

Kilocalories

Carbohydrates (CHO)

Protein

Fat

Vitamins

Minerals

Nutrition for Competition

Carbohydrate Loading (Glycogen Supercompensation)

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Nutrient Timing

Eating Disorders

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Risk Factors

The Consequences of Eating Disorders

Prevention and Treatment

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- List the goals for nutrition during training and for nutrition during competition, and explain why they are different.
- Compare a balanced diet for sedentary individuals with a balanced diet for active individuals in terms of caloric intake; carbohydrate, fat, and protein intake; and vitamin, mineral, and fluid requirements.
- Discuss the positive and negative aspects of a high-carbohydrate

diet.

- Explain the glycemic index; identify common high-, moderate-, and low-glycemic foods; and explain the best use of each classification. Describe a training situation when fat intake can be too low.
- Develop a pre-event meal plan for athletic competition.
- Develop a plan for feeding and drinking during an endurance event versus a power event, and defend each.
- Judge the value of commercially available sports drinks.
- Compare the recommended daily allowance (RDA) for protein to current higher protein recommendations for active individuals.
- Explain the term anabolic resistance and how to mitigate this during aging.
- Understand nutrient timing and why it may be important for exercise benefits.
- Differentiate among the eating disorders anorexia nervosa, bulimia nervosa, and anorexia athletica in terms of definitions and characteristics.
- Identify the risk factors for developing an eating disorder.
- Construct guidelines to help prevent or manage eating disorders in exercise settings.

Introduction

Proper nutrition and exercise are natural partners for health, fitness, and athletic performance. Consequently, many fitness enthusiasts pursue healthy diets, and athletes try to optimize their performance by implementing appropriate diets. Although these are very positive trends, they also have the potential to be taken to an extreme, which may simply involve spending money needlessly on “nutritional supplements” or may actually be harmful, as with eating disorders. It is the responsibility of all physical educators, athletic personnel, rehabilitation clinicians, and fitness professionals to understand what constitutes optimal nutrition for fitness and athletics.

Nutrition education should be a part of physical fitness classes, community adult fitness and rehabilitation programs, and

athletic training. Most individuals who train regularly want to eat right, but they may confuse advertisements and media hype with factual information.

Optimal nutrition for fitness and athletics must be considered for two different situations. The first is training, and the second is competition, whether on the “fun run” fitness or elite competitive level. With the exception of some youth sports, individuals typically spend more total time training than competing, making daily nutritional practices critical. No amount of dietary manipulation the day of or the day before a competition can make up for otherwise poor nutritional habits (Burke and Read, 1989).

Nutrition for Training

Individuals in exercise training need to match their training regimen with an appropriate diet. This often involves consultation with a fitness professional, a complete diet analysis by a nutritionist, and, many times, a trial-and-error approach to find what works best for a given individual.

The goals of an optimal training diet are to the following:

1. Provide caloric and nutrient requirements.
2. Incorporate nutritional practices that promote good health.
3. Achieve and maintain optimal body composition and competition weight.
4. Promote recovery from training sessions and physiological adaptations.
5. Try variations of precompetition and competition fuel and fluid intake to determine the body's responses.

There is almost universal agreement that poor nutritional status impairs work performance. There is also considerable, although not universal, agreement that good general nutrition (the balanced diet recommended for just about everyone, as shown in **Table 6.1**) is adequate and probably even optimal for most active individuals as well as sedentary individuals.




TABLE 6.1 Balanced Diets

| For Sedentary Individuals | For Active Individuals |
|--|--|
| Calories Calorie balance of intake and expenditure to maintain acceptable body composition and weight | Adequate caloric intake to balance caloric expenditure of training and competition in excess of normal living while maintaining optimal body composition and playing weight |
| Protein 5–20% protein (0.95 g·kg ⁻¹ ·d ⁻¹), 1–3 y; 10–30%, (0.85 g·kg ⁻¹ ·d ⁻¹), 4–18 y; 10–35% (0.8 g·kg ⁻¹ ·d ⁻¹), adults 19+ y | 10–35% protein (1.2–2 g·kg ⁻¹ ·d ⁻¹) adults 19+ y |
| Fat 30–40% fat, 1–3 y; 25–35% 4–18 y; 20–35%, adults 19+ y [†] (<10% saturated; trans fats as low as possible) 65 g·2,000 kcal ⁻¹ ; 80 g·2,500 kcal ⁻¹ or 0.5–1.5 g·kg ⁻¹ ·d ⁻¹ | 20–35% fat (0.8–1.0 g·kg ⁻¹ ·d ⁻¹) |
| Carbohydrates (CHO) 45–65% CHO all ages (130 [‡] –300 g·d ⁻¹) (4.5 g·kg ⁻¹ ·d ⁻¹) | <ul style="list-style-type: none"> • For low-intensity, short-duration exercise: 4.5–5 g·kg⁻¹·d⁻¹ • For moderate exercise, 1 hr·d⁻¹: 5–7 g·kg⁻¹·d⁻¹ • For endurance training, moderate to high intensity 1–3 hr·d⁻¹: 6–10 g·kg⁻¹·d⁻¹ • For extreme training, moderate to high intensity >4–5 hr·d⁻¹: 8–12 g·kg⁻¹·d⁻¹ • 45–70% CHO |
| Vitamins and Minerals DRI/RDA for vitamins and minerals | DRI/RDA for vitamins and minerals |
| Fluids Total water [§] 2,700–3,700 mL·d ⁻¹ (80–110 fluid oz·d ⁻¹) or 2,200–3,000 mL·d ⁻¹ (74–100 fluid oz·d ⁻¹) drinking water + beverages | Fluids adequate to prevent dehydration: baseline values plus (if needed) 5–7 mL·kg ⁻¹ (0.2–0.25 fluid oz·kg ⁻¹) 4 hr and 3–5 mL·kg ⁻¹ (0.1–0.2 fluid oz·kg ⁻¹) 2 hr prior to exercise; as during exercise, 1.5 L (50 fluid oz) postexercise for each kg of body weight lost |

***Note:** the 2015 Dietary Guidelines no longer include a recommended limit on cholesterol intake or the total fat level for adults. However, the natural limit for total fat, if individuals are following a healthy dietary pattern, should be in the 32–34% range.

†The RDA value of 130 g·d⁻¹ is based on the amount of CHO needed for brain function; the % is based on the role of CHO as an energy source to maintain body weight.

‡Total water includes drinking water, water in beverages, and water that is a part of food.

Sources: Based on information from [ACSM \(2007\)](#); [ACSM et al. \(2009, 2016\)](#); [Brotherhood \(1984\)](#); [Burke et al. \(2011\)](#); [Dietary Guidelines for Americans \(2020\)](#); [Haymes \(1983\)](#); [Kreider et al. \(2010\)](#); [Millen et al. \(2016\)](#); and [Venkatraman and Pendergast \(2002\)](#).

Unfortunately, a typical American does not eat the recommended healthy balanced diet. If the recommended percentages from a healthy variety of foods are consumed, the vast majority of youth sport, middle and secondary school, and college athletes, as well as fitness participants of all ages, will not

need any modification in their diet. For those athletes or fitness participants training very long, hard, and often competing at an elite level, a few modifications may be beneficial (Allen et al., 1979; American College of Sports Medicine [ACSM] et al., 2009; American Dietetic Association [ADA], 1987; Belko, 1987; Brotherhood, 1984; Burke and Read, 1989; Lemon and Nagle, 1981; Nieman, 1990). **Table 6.1** summarizes these recommendations, which are discussed in the following sections.

The United States Department of Agriculture's (USDA) food icon, called MyPlate (**Figure 6.1**), symbolizes healthy nutrition. The Canadian version, the Eat Well Plate, is very similar but places both a glass of water and a container for oils and fat outside of the actual plate (Health Canada, 2013). There are four primary messages to the ChooseMyPlate campaign (ChooseMyPlate.gov):

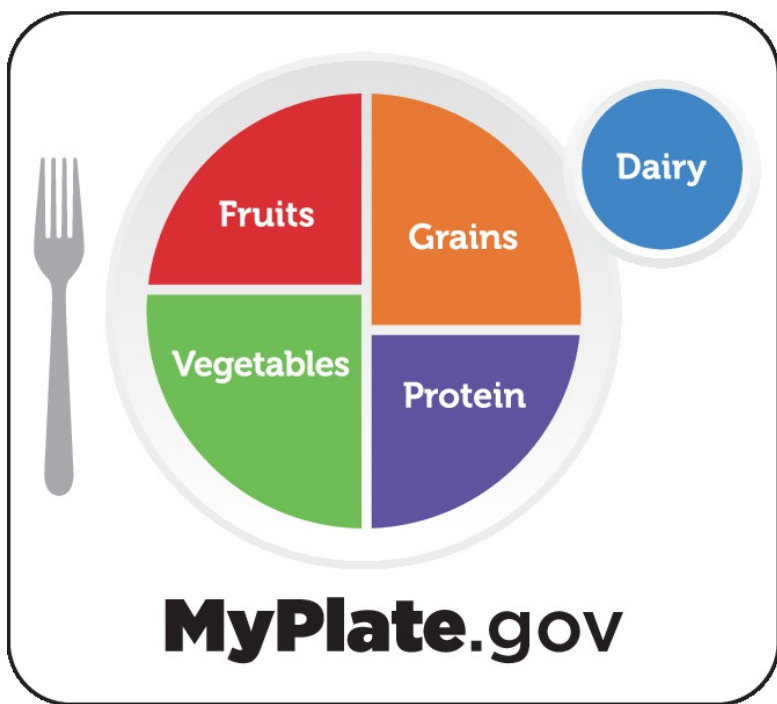


Figure 6.1 Food Guide for Sedentary and Active Individuals.

This plate icon from the U.S. Department of Agriculture

(USDA) emphasizes that one half of your plate should be filled with fruits and vegetables, with whole grains and lean protein on the other half. Low-fat dairy on the side is also suggested. The Web site MyPlate.gov offers resources for both individuals and professionals teaching others about the concepts based on the *2020 Dietary Guidelines for Americans* to help meet nutrient and caloric needs and make positive eating choices. **Source:** From the USDA Web site: www.choosemyplate.gov.

1. Build a healthy plate that is filled one half with fruits and vegetables and the other half with whole grains and lean protein; add a side of low-fat dairy.
2. Cut back on foods high in solid fats, added sugars, and salt (SoFAS).
3. Eat the right amount of calories for you.
4. Be physically active your way.

The intent is that individuals will be consuming a diet that emphasizes vegetables, fruits, whole grains, legumes, and nuts; includes low- and nonfat dairy products, poultry, seafood, and nontropical vegetable oils; and limits sodium, saturated fat, refined grains, added sugar, sugarsweetened foods and beverages, and red and processed meats (USDA, Department of Health & Human Services [DHHS], 2015). However, a recent meta-analysis suggests that the consumption of fruits and vegetables alongside red and processed meat decreases the risk of specific cancers and all-cause mortality (Maximova et al., 2020). Individuals are encouraged to go to www.choosemyplate.gov, create a profile, and obtain a daily physical activity target and nutrition plan based on age, sex, height, current weight, and current physical activity. For example, for a 21-year-old female who is 5'8" tall and weighs 145 lb, the food recommendations would be 8 oz. of grains, 3 cups of vegetables, 2 cups of fruit, 3 cups of dairy, 6.5 oz. of protein, and 7 tsp of oil. This would leave approximately 330 kcal of empty calories that could be ingested from the limit list of solid fats and added sugars. For a male, the only changes are that he is allotted 9 oz. of grains, 3.5 cups of vegetables, 8 tsp

of oil, and approximately 362 empty calories. Fruit, dairy, and protein amounts remain the same. Each plan gives examples of what foods count as that category and tips to help make healthy choices. If you have not yet visited this Web site, it might be an interesting exercise to do. Information for professionals is also included on the Web site.

Figure 6.2 shows the usual intake of select foods in a typical American diet as a percent above or below the dietary guideline recommendations from eating the recommended healthy balanced diet. Approximately 90 and 80% of Americans consume below the recommended quantity of vegetables and fruits, respectively ([Dietary Guidelines for Americans, 2020](#)). How does your diet compare? The Focus on Application box provides information on nutrition label information that can be useful in developing a balanced diet.

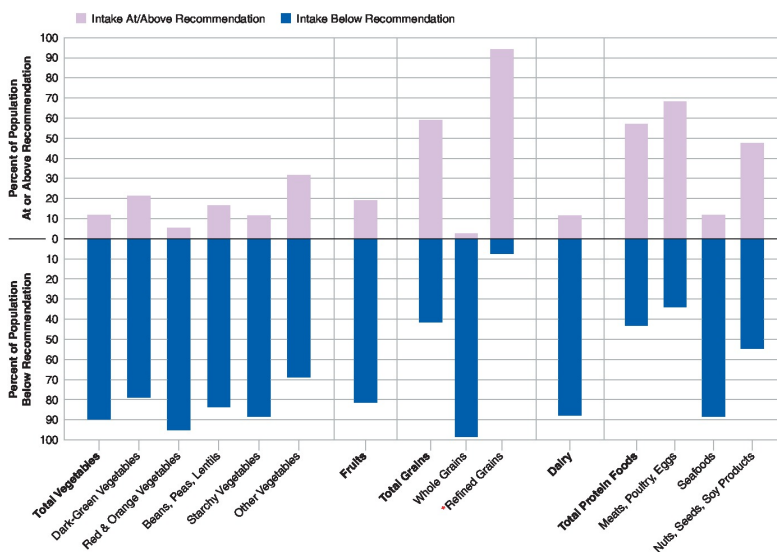


Figure 6.2 The Usual Intake of Select Foods in a Typical American Diet as a Percent Above or Below the Dietary Guideline Recommendations.

Approximately 90 and 80% of Americans consume below the recommended quantity of vegetables and fruits, respectively whereas approximately 50-60% consume total grains and proteins in excess of the recommendations.

Source: From www.dietaryguidelines.gov (2020).

Kilocalories

The most obvious dietary distinction between active and inactive individuals is the number of calories required per day. Everyone needs sufficient calories to support their daily needs, and children need adequate calories for growth. In addition, an active individual can expend several hundred to several thousand kilocalories more per day than a sedentary individual. The actual amount depends on the individual's size and the intensity, duration, and frequency of the workouts. For example, large football players doing two-a-day workouts and an endurance athlete of any size expend a large amount of calories, but golfers or softball and baseball players of any size expend much smaller amounts (ACSM et al., 2009; ADA, 1987; Brotherhood, 1984; Burke and Read, 1989; Kreider et al., 2010; Leaf and Frisa, 1989). Costill (1988) cites figures of 900–2,400 kcal·d⁻¹ expended in activity for elite distance runners, 6,000 kcal·d⁻¹ for cyclists, and 1,250–3,750 kcal·d⁻¹ for swimmers during training. These calories must be replaced.

Published reports indicate that male basketball and football players may consume as many as 9,000–11,000 kcal·d⁻¹, triathletes 3,500–6,400 kcal·d⁻¹ (female and male), cross-country skiers 4,000–5,500 kcal·d⁻¹ (female and male), and track and field athletes (male) 3,500–4,700 kcal·d⁻¹, depending on the event. It is highly likely that these athletes are adequately resupplying their energy needs (Burke and Read, 1989).

However, competitors in other sports (long-distance running, gymnastics, wrestling, and dance) often attempt to maintain extremely low and, in some cases, “unnatural” body fat and body weight. Intake values as low as 600 kcal·d⁻¹ for male gymnasts and 900 kcal·d⁻¹ for female ballet dancers have been reported. It is likely that these athletes are not adequately resupplying their energy needs (Burke and Read, 1989).

Table 6.2 presents caloric needs based on estimated energy requirements (EER) and activity levels for males and females from 2 to 76+ years. The EER is the average dietary energy intake that is predicted to maintain energy balance consistent

with good health, that is, with a BMI of 18.5–25 kg·m⁻² and a reduced risk of cardiovascular disease. Note that there are separate columns for sedentary, moderately active, and active individuals. The caloric values in **Table 6.2** should be sufficient for most individuals engaging in physical activity for health and fitness.

TABLE 6.2 Estimated Calorie Needs per Day by Age, Sex, and Physical Activity Level

| Males | Activity Level | | | Females | Activity Level | | |
|-----------|----------------|--------------|---------|-----------|----------------|--------------|---------|
| Age | Sedentary* | Mod. Active* | Active* | Age | Sedentary* | Mod. Active* | Active* |
| 2 | 1,000 | 1,000 | 1,000 | 2 | 1,000 | 1,000 | 1,000 |
| 3 | 1,200 | 1,400 | 1,400 | 3 | 1,000 | 1,200 | 1,400 |
| 4 | 1,200 | 1,400 | 1,600 | 4 | 1,200 | 1,400 | 1,400 |
| 5 | 1,200 | 1,400 | 1,600 | 5 | 1,200 | 1,400 | 1,600 |
| 6 | 1,400 | 1,600 | 1,800 | 6 | 1,200 | 1,400 | 1,600 |
| 7 | 1,400 | 1,600 | 1,800 | 7 | 1,200 | 1,600 | 1,800 |
| 8 | 1,400 | 1,600 | 2,000 | 8 | 1,400 | 1,600 | 1,800 |
| 9 | 1,600 | 1,800 | 2,000 | 9 | 1,400 | 1,600 | 1,800 |
| 10 | 1,600 | 1,800 | 2,200 | 10 | 1,400 | 1,800 | 2,000 |
| 11 | 1,800 | 2,000 | 2,200 | 11 | 1,600 | 1,800 | 2,000 |
| 12 | 1,800 | 2,200 | 2,400 | 12 | 1,600 | 2,000 | 2,200 |
| 13 | 2,000 | 2,200 | 2,600 | 13 | 1,600 | 2,000 | 2,200 |
| 14 | 2,000 | 2,400 | 2,800 | 14 | 1,800 | 2,000 | 2,400 |
| 15 | 2,200 | 2,600 | 3,000 | 15 | 1,800 | 2,000 | 2,400 |
| 16 | 2,400 | 2,800 | 3,200 | 16 | 1,800 | 2,000 | 2,400 |
| 17 | 2,400 | 2,800 | 3,200 | 17 | 1,800 | 2,000 | 2,400 |
| 18 | 2,400 | 2,800 | 3,200 | 18 | 1,800 | 2,000 | 2,400 |
| 19–20 | 2,600 | 2,800 | 3,000 | 19–20 | 2,000 | 2,200 | 2,400 |
| 21–25 | 2,400 | 2,800 | 3,000 | 21–25 | 2,000 | 2,200 | 2,400 |
| 26–30 | 2,400 | 2,600 | 3,000 | 26–30 | 1,800 | 2,000 | 2,400 |
| 31–35 | 2,400 | 2,600 | 3,000 | 31–35 | 1,800 | 2,000 | 2,200 |
| 36–40 | 2,400 | 2,600 | 2,800 | 36–40 | 1,800 | 2,000 | 2,200 |
| 41–45 | 2,200 | 2,600 | 2,800 | 41–45 | 1,800 | 2,000 | 2,200 |
| 46–50 | 2,200 | 2,400 | 2,800 | 46–50 | 1,800 | 2,000 | 2,200 |
| 51–55 | 2,200 | 2,400 | 2,800 | 51–55 | 1,600 | 1,800 | 2,200 |
| 56–60 | 2,200 | 2,400 | 2,600 | 56–60 | 1,600 | 1,800 | 2,200 |
| 61–65 | 2,000 | 2,400 | 2,600 | 61–65 | 1,600 | 1,800 | 2,000 |
| 66–70 | 2,000 | 2,200 | 2,600 | 66–70 | 1,600 | 1,800 | 2,000 |
| 71–75 | 2,000 | 2,200 | 2,600 | 71–75 | 1,600 | 1,800 | 2,000 |
| 76 and up | 2,000 | 2,200 | 2,400 | 76 and up | 1,600 | 1,800 | 2,000 |

*Calorie levels are based on the Estimated Energy Requirements using reference weights for age and sex and activity levels from the Institute of Medicine Dietary Reference Intakes Macronutrients Report, 2002.

Sedentary, only the light physical activity associated with typical day-to-day life.

Moderately active, equivalent to walking 1.5–3 mi·d⁻¹ at 3–4 mi·hr⁻¹ in addition to daily activities.

Active, equivalent to walking more than 3 mi·d⁻¹ at 3–4 mi·hr

– 1 in addition to daily activities.

Source: USDA [Dietary Guidelines for Americans \(2020\)](#).

FOCUS ON APPLICATION

Food Label Interpretation

The dietary recommendations presented in **Table 6.1** are expressed as percentages and $\text{g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$. Information about both of these measures can be obtained from reading the “Nutrition Facts” label found on most foods. Content of these labels is regulated by the U.S. Food and Drug Administration (FDA). In the spring of 2016, the FDA approved changes to this label. The accompanying figure shows the newly approved food label.

Food labels provide information on the caloric and nutrient composition of different foods. For example, the food label in the accompanying image states that the serving size is 2/3 cup, which equals 55 g and 230 (kilo) calories. Note that both the serving size and the total calories are in larger bold letters and given prominence in the newly approved new label. Individual nutrients are also given in grams. The unnamed food in this label contains 8 g of fat, 37 g of CHO, but no added sugar, and 3 g of protein. Fat is not usually calculated in $\text{g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$, but to obtain this value for CHO and protein, all that is needed is to divide by body weight in kilograms. For example, for a 154 lb (70 kg) individual, this represents $0.53 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ of CHO and $0.04 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ of protein. Obviously, this does not satisfy the daily recommendations, but it does illustrate how these values can be obtained. All that is needed would be to total the CHO and protein intake for all the food ingested during the day and divide it by body weight. Thus, if this individual ate 310 g of CHO during the day, it would result in $4.43 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$, very close to the recommended $4.5 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$. Note that the new label would include sugar that is not natural to the food, that

is, “added sugar” to encourage food choices with less added sugar.

The % daily value (%DV) column based on a 2,000-kcal diet and shows what percentage of the daily recommendation is achieved by eating the contents to which the label is attached. The fat content represents 10% DV and CHO 13% DV. A %DV for protein is not required by the FDA unless a claim is made such as “high in protein” or the product is intended for children under the age of 4 years. This is because protein intake is not a public health concern. Note that there is also no %DV for *trans*-fat because this type of fat should be kept as low as possible. Foods that are “low” in a nutrient generally contain $\leq 5\%$ DV; foods that are a “good” source of a nutrient generally contain 10–19% DV; foods that are a “high/rich/excellent” source of a nutrient generally contain $\geq 20\%$ DV per serving.

The information in a food label is based in large part on the Dietary Reference Intakes (DRIs) established by the Food and Nutrition Board of the National Academy of Sciences. The primary goals of the basic recommendations are to prevent nutrient deficiencies and to reduce the risk of chronic diseases such as cardiovascular disease, cancer, and osteoporosis. The two DRI reference values used in this text are as follows:

1. **Recommended daily allowance (RDA)**—the average daily intake level that is sufficient to meet the nutrient requirement of 97–98% of healthy individuals by age and sex.

Recommended Daily Allowance (RDA) The average daily intake level that is sufficient to meet the nutrient requirement of 97–98% of healthy individuals by age and sex.

2. **Adequate intake (AI)**—used when an RDA cannot be determined. The AI is based on observation or is

experimentally determined and is an estimate of intake by healthy individuals.

Adequate Intake (AI) Used when an RDA cannot be determined. The AI is an estimate of intake by healthy individuals.

Most vitamin and mineral percentages presented in food labels are based on DRI/RDA values. Values are presented for only one vitamin (D), but also for calcium, iron and potassium.

It is also proposed that the nutrition labels be moved to the front of the food container. Food companies will, of course, be given a couple years to comply with the new labeling regulations.

Food Label

Nutrition Facts

8 servings per container

Serving size 2/3 cup (55g)

Amount per serving

Calories 230

% Daily Value*

Total Fat 8g 10%

Saturated Fat 1g 5%

Trans Fat 0g

Cholesterol 0mg 0%

Sodium 160mg 7%

Total Carbohydrate 37g 13%

Dietary Fiber 4g 14%

Total Sugars 12g

Includes 10g Added Sugars 20%

Protein 3g

Vitamin D 2mcg 10%

Calcium 260mg 20%

Iron 8mg 45%

Potassium 235mg 6%

* The % Daily Value (DV) tells you how much a nutrient in a serving of food contributes to a daily diet. 2,000 calories a day is used for general nutrition advice.

Sources: Dietary Guidelines for Americans (2020); International Food Information Council (2002); U.S. Food and Drug Administration (2015).

If the relative proportion of nutrients obtained from a healthy diet remains the same as the calories are increased to support training, the active individual would have an acceptable diet. However, some subtle shifting of percentages and/or amounts can be of benefit in certain situations. Chief among them is an increased percentage of carbohydrate (CHO) ingestion for endurance athletes.

Carbohydrates (CHO)

The discussion of CHO metabolism in [Chapter 2](#) pointed out several important facts about CHOs as a fuel for exercise ([ACSM et al., 2009](#); [ADA, 1987](#); [Burke and Read, 1989](#); [Costill, 1988](#); [Nieman, 1990](#)):

1. The higher the intensity of exercise (whether continuous or intermittent; aerobic, anaerobic, or aerobicanaerobic), the more important glycogen is as a fuel.
2. The body can only store limited amounts of CHOs. Training increases the ability to store CHO and to spare CHO. However, 60–90 minutes of heavy endurance work seriously depletes glycogen stores, and depletion can be complete in 120 minutes. Muscle glycogen can also be depleted by 15–30 minutes of near-maximal intensity or supramaximal-intensity interval work.
3. Fat metabolism is linked to CHO metabolism. Fatigue, “hitting the wall,” and exhaustion are related to glycogen depletion during high-intensity, long-duration activity. Thus, having an adequate supply of muscle glycogen is necessary to avoid fatigue. Whatever glycogen is utilized, in training or competition, must be replenished before more heavy work can be done.

How much CHO should be included in the diet? The RDA of CHO ([Table 6.1](#)) for the average sedentary individual is 4.5 g·kg⁻¹·d⁻¹. For an individual in training, a graduated increase in CHO ingestion is recommended ([Burke et al., 2011](#)) as follows:

- For low-intensity, short duration exercise: 4.5–5 g·kg⁻¹·d⁻¹

- For moderate exercise, 1 hr·d⁻¹: 5–7 g·kg⁻¹·d⁻¹
- For endurance training, moderate to high intensity 1–3 hr·d⁻¹: 6–10 g·kg⁻¹·d⁻¹
- For extreme training, moderate to high intensity greater than 4–5 hr·d⁻¹: 8–12 g·kg⁻¹·d⁻¹

Which CHO should be eaten? All CHO are not the same, and the traditional classification of CHO into “simple” or “complex” CHO according to chemical structure is not useful in terms of the “healthfulness” and impact on glucose and insulin levels. To solve this dilemma, the concept of the glycemic index is used.

Glycemic Index

The **glycemic index (GI)** categorizes foods based on the glucose response they produce, using a numerical value for comparing foods. The GI compares the elevation in blood glucose caused by the ingestion of 50 g of any CHO food with the elevation caused by 50 g of white bread or glucose (dextrose) 2-hour postingestion (Foster-Powell et al., 2002; Wolever, 1990; Wong and Chung, 2003). Thus, the GI value of any given food depends on the speed at which it is digested and absorbed compared with a reference food. When glucose is used as the reference CHO, it has a GI of 100.

Glycemic Index (GI) A measure that compares the elevation in blood glucose caused by the ingestion of 50 g of any CHO food with the elevation caused by the ingestion of 50 g of white bread or glucose.

- High-glycemic foods have a rating of 70 or greater.
- Moderate-glycemic foods rate from 56 to 69.
- Low-glycemic foods have a rating less than 55.

Values above 100 are possible; for example, glucose has a value of 143 compared to white bread. When glucose is used as the reference food, the values of all other foods are lower than when white bread is the reference. When referring to tables of GI,

it is important to note whether white bread or glucose is the reference value. All the values in **Table 6.3** use white bread as the reference food.

TABLE 6.3 Glycemic Index of Selected Foods

| High-Glycemic Food (HGI) (85 or Greater) | Moderate-Glycemic Food (MGI) (60–85) | Low-Glycemic Food (LGI) (Under 60) |
|--|--|------------------------------------|
| Sugars, Syrups, and Jellies | | |
| White table sugar | | Fructose |
| Maple syrup | | M&M peanuts |
| Honey | | Nutella |
| Sports drinks (6–20% CHO concentration) | | |
| Cereal Products | | |
| Bagel | Rice | Barley |
| Bread (white and wheat) | Pasta (spaghetti and macaroni) | All-Brn cereal |
| Cornflakes/Cheerios | Bread (whole grain and rye) | Wheat tortilla |
| Shredded wheat | Corn tortilla | 9-grain bread |
| Crackers | Oatmeal (cooked) | Brown rice |
| Puffed wheat and rice | Pita bread | |
| Fruits | | |
| Raisins | Grapes | Apples/apple juice |
| Watermelons | Orange juice | Cherries |
| Cranberry juice | Bananas | Dried apricots |
| | Kiwi | Peaches |
| | Mangos | Pears |
| | | Plums/prunes |
| | | Grapefruits |
| | | Strawberries |
| Vegetables | | |
| Potatoes (baked, microwaved, mashed, and French fried) | Yams/sweet potatoes | Tomato soup/juice |
| Carrots | Sweet corn (frozen) | |
| Parsnips | Potato chips | |
| Sweet corn (fresh) | Peas | |
| | Popcorn | |
| Legumes | | |
| | Baked beans | Beans (butter, green), chickpeas, |
| | Beans (kidney and pinto, canned) | kidney, navy, and pinto (dried) |
| | Peas (chick and green, canned or frozen) | Lentils (green and red, dried) |
| | Lentils (red and green, canned) | Peanuts/cashews |
| | | Hummus |
| Dairy Products | | |
| Ice cream | Ice cream (low fat) | Milk (skim, whole) and yogurt |
| | | Custard |
| Convenience Foods | | |
| Kraft macaroni and cheese | Snickers bar | Fish fingers |
| Angel food cake | Chicken nuggets | Pizza Hut Supreme |
| | Peanut butter sandwich | Ironman bar (chocolate) |
| | Ensure | |
| | Instant noodles | |
| | PowerBar (chocolate) | |

Sources: Compiled from Coyle and Coyle (1993); Foster-Powell et al. (2002); Jenkins et al. (1981); Wolever (1990); and Walton and Rhodes (1997).

Foods with a high GI (HGI) cause a fast elevation in glucose and subsequently in insulin; foods with lower indices cause a slower rise in both glucose and insulin. **Table 6.3** gives examples of high-, moderate-, and low-glycemic foods. In general, sugars

and sports drinks, syrups and jellies, and grain, pasta, and cereal products have high or moderate glycemic indices (MGIs). Most fruits, legumes, and dairy products have low glycemic indices (LGIs). Meat, poultry, fish, avocados, salad vegetables, cheese, and eggs do not have GI values because these foods contain little or no CHO and when eaten alone do not usually cause a meaningful increase in blood glucose. A food's GI value also depends on where it is grown, its variety (with foods such as rice), and how it is processed (Foster-Powell et al., 2002).

In addition to the GI of specific foods, how an individual's body responds to CHO ingestion depends also, in part, on the person's fitness level (Manore, 2002). **Figure 6.3** shows the typical response pattern of a sedentary individual, a moderately active individual, and a trained endurance runner. In general, trained individuals have a lower glycemic (and hence lower insulin) response to food than do untrained individuals.

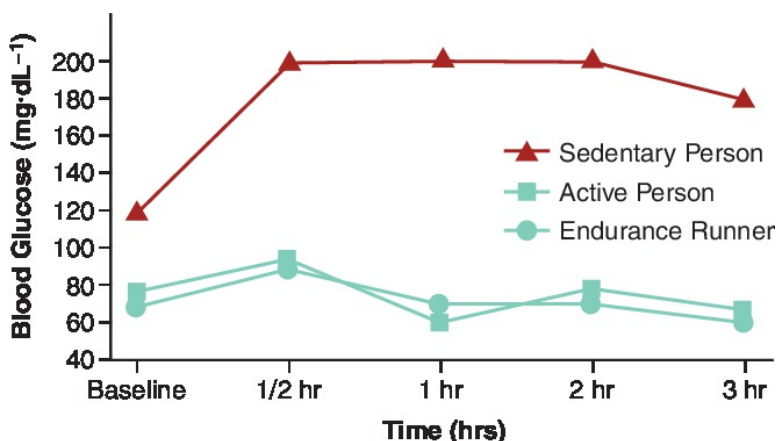


Figure 6.3 Blood Glucose Changes after Ingestion of 100 g of Glucose in Sedentary, Active, and Endurance-Trained Individuals.

Source: Reprinted with permission from Manore, M. M.: Carbohydrate: Friend or foe? Part II: Dietary carbohydrate and changes in blood glucose. *ACSM's Health and Fitness Journal*. 6(3):26–29 (2002). Copyright ©2002 American College of Sports Medicine.

The GI compares food containing the same amount of CHO. It does not take into account the serving size. To do this, the glycemic load (GL) must be calculated. The GL is the product of the amount of available CHO (g) and the GI divided by 100 (Manore et al., 2004; Wong and Chung, 2003). Thus, eating a large portion of any food produces a higher GL than does a small portion. The higher the GL is, the greater the elevation in blood glucose and insulin. The premise of GL is that the effect, if any, on exercise performance is determined by the overall glycemic effect of a diet and not by the amount of CHO alone (O'Reilly et al., 2010). The following section will concentrate on glycogen replenishment, called resynthesis, because of the importance of adequate glycogen levels to training.

Complete the [Check Your Comprehension 1](#) to determine your understanding of the Glycemic Index and the Glycemic Load.

CHECK YOUR COMPREHENSION 1

Rank these CHO foods (normal serving of each) from low to high on the basis of glycemic load (GL)

| Food | Glycemic Index (GI) (compared to glucose) | CHO (g) |
|--------------------------------------|--|---------|
| Bagel (70 g) | 69 | 35 |
| Snickers bar (60 g) | 68 | 34 |
| Banana (120 g) | 47 | 24 |
| Gatorade orange flavor (16 oz) | 89 | 29 |
| Pop tart double chocolate (50 g) | 70 | 36 |
| Clif bar chocolate brownie (65 g) | 57 | 38 |

How does the calculation of the GL affect the impact of the GI of foods? Support your answer.

Check your answer in [Appendix C](#).

Glycogen Resynthesis

The storage of CHO in liver and muscles depends primarily on the severity of glycogen depletion resulting from activity, the extent of muscle trauma, and the amount of dietary CHO. Muscle glycogen resynthesis is fastest when the muscle has been depleted, but not necessarily exhausted, and the diet is high in CHO. Muscle fiber damage associated with exhaustive eccentric exercise (in which a contracting muscle is forcibly lengthened), such as running marathon distances especially if downhill running is involved, may delay resynthesis for as long as 7–10 days despite elevated dietary CHO (Burke and Read, 1989; Costill, 1988).

Given optimal amounts of dietary CHO, muscle glycogen resynthesis is faster (per muscle mass per hour) after short-term, high-intensity exercise than after long-term, submaximal endurance exercise. Rates of muscle glycogen resynthesis following dynamic resistance exercise are slower than after short-term, high-intensity exercise but may be less than, equal to, or slightly faster than resynthesis following prolonged endurance exercise. The rate at which glycogen resynthesis occurs largely depends on the blood lactate concentration (higher levels result in faster rates of synthesis) and eccentric loading (higher levels result in slower rates of resynthesis) (Pascoe and Gladden, 1996).

Glycogen resynthesis occurs at about 5–6% per hour under optimal dietary conditions, thus requiring approximately 17–20 hours for complete recovery (Coyle, 1991; Coyle and Coyle, 1993). Optimal conditions involve both the timing and the type of CHO ingestion. Glycogen resynthesis postexercise occurs in two physiological phases: early (<4 hours), which appears to be insulin independent, and late (>4 hours), which shows a period of enhanced insulin sensitivity and is thus, insulin dependent. Consuming CHO immediately after exercise results in higher glycogen levels 4 hours after exercise than if CHO ingestion is delayed 2 hours. Thus, when the interval between exercise sessions is short (<8 hours), CHO ingestion (50–100 g at the rate of 1.2 g·kg BW⁻¹·hr⁻¹) should

- Begin as soon after the workout or competition as is practical (15–30 minutes).

- Continue at that rate every 15–20 minutes until a larger meal of solid food (150–250 g of CHO) is desired and possible.
- Be maintained for at least 4–6 hours (Betts and Williams, 2010; Cermak and van Loon, 2013; Coyle and Coyle, 1993; Jentjens and Jeukendrup, 2003).

However, when a longer recovery time is available

- The consumption of CHO immediately is not as important but is recommended (Ivy and Ferguson-Stegall, 2014).
- As long as 7–10 g·kg BW⁻¹ of CHO is consumed over 24 hours at a rate of at least 50 g·hr⁻¹, muscle and liver glycogen will be replaced over this time (Donaldson et al., 2010).

There is some controversy as to whether or not a ceiling effect occurs with CHO ingestion for glycogen storage. It has been shown that 12 g·kg BW⁻¹ of CHO increased stored glycogen more than 6 g·kg BW⁻¹ of CHO (Tomcik et al., 2018). For a 176-lb (80 kg) individual, this is equivalent to 960 g of CHO consumed, which is higher than the approximately 600 g that was considered the ceiling for CHO intake to help with glycogen resynthesis (Millard-Stafford et al., 2008).

Selection of the amount and type of postexercise CHO should be based on the time available for replenishment, the goal of the person exercising, and GI of the food (Coyle, 1991; Coyle and Coyle, 1993; Escobar et al., 2016; Kerksick et al., 2017).

In general, this means

- Individuals should ingest higher-glycemic foods immediately after exercise (during recovery) in combination with protein and the addition of caffeine, especially when exercising again within close proximity to the first workout and rapid glycogen resynthesis is required (Manore et al., 2004; Kerksick et al., 2017).
- Moderate-glycemic foods may also be useful, however, because they appear to promote glycogen resynthesis just about as effectively (Coyle and Coyle, 1993; Hausswirth and Le Meur, 2011).

- For optimal athletic performance, CHOs consumed postexercise should be influenced by personal preference and quantity consumed rather than GI (Baur and Saunderson, 2021).

The chemical makeup of the CHO also matters. Recent studies have shown that the consumption of multiple transportable CHOs, which are CHOs that depend on different transporters for intestinal absorption, may be an effective strategy for improved exercise performance and recovery. For example, intestinal absorption of glucose is dependent of the SGLT1 transporter, whereas the absorption of fructose is dependent on the GLUT5 transporter. Since different transporters are used for the absorption of various CHO sources, the availability of one transporter does not limit the absorption of the other CHO sources that use a different transporter for intestinal absorption. Exogenous CHO oxidation rates have been shown to be as high as $1.75 \text{ g}\cdot\text{min}^{-1}$ using multiple transportable CHOs compared to $1 \text{ g}\cdot\text{min}^{-1}$ when using single-source CHO supplementation (Jeukendrup, 2013).

The addition of amino acids and/or protein to a CHO supplement can potentially increase muscle glycogen synthesis because of an enhanced insulin response. Insulin stimulates both glucose uptake and activation of glycogen synthase—the rate-limiting enzyme for glycogen synthesis. When CHO is ingested at the rate of $\geq 1.2 \text{ g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ at regular intervals, however, higher insulin concentrations do not further increase the rate of muscle glycogen resynthesis. Conversely, when CHO intake is insufficient ($< 1.2 \text{ g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$), the addition of amino acids and/or protein can be beneficial. It may not always be possible for an athlete to consume this much CHO, or the athlete may simply prefer a moderate CHO intake ($0.8 \text{ g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$) in combination with amino acids or protein at the rate of $\geq 0.3 \text{ g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$. This should result in the same amount of glycogen resynthesis as the high CHO alone. The combination may have an additional advantage of stimulating postexercise protein anabolism, thus stimulating tissue growth and repair. Thus, the combination of CHO and protein may be particularly beneficial after heavy resistance training (Betts and Williams, 2010; Cermak and van Loon, 2013; Donaldson et al., 2010; Ivy and Ferguson-

Stegall, 2014; Jentjens and Jeukendrup, 2003; Manninen, 2006; McLellan et al., 2014; O'Reilly et al., 2010; Poole et al., 2010).

Interestingly, chocolate milk has been shown to be beneficial as a recovery beverage (**Table 6.4**). Cow's milk provides all the essential amino acids, and the CHO content of fatfree chocolate milk exceeds that of white milk. One study compared endurance capacity after a glycogen depletion trial and a 4-hour recovery period during which the participants ingested either chocolate milk (CM), a fluid replacement drink (Gatorade), or a CHO replacement drink (Endurox R4). All three beverages had similar amounts of CHO. The CM and CHO replacement drink had similar amounts of PRO and total kcal. Participants cycled 51% longer after ingesting CM than the CHO replacement alone and 43% longer after ingesting CM than fluid replacement alone (Thomas et al., 2009). Another study compared the effects of fat-free chocolate milk with an equal caloric CHO drink. The milk contained 16 g of protein, and the CHO drink had none. The milk was as effective as the CHO drink at maintaining muscle glycogen during recovery and for performance in a subsequent exercise bout. In addition, milk ingested during recovery significantly increased skeletal muscle protein synthesis, decreased whole-body protein breakdown, and suppressed or maintained molecular activity of protein breakdown during the recovery period. Similarly, chocolate milk has been shown to improve overall strength composite scores after 5 weeks of resistance training in adolescents compared to a CHO recovery drink (Born et al., 2019). These results suggest that chocolate milk supports both skeletal muscle and whole-body protein recovery as well as glycogen resynthesis (Lunn et al., 2012).

TABLE 6.4 Composition of Selected Sports Drinks
(per 8 oz. or 240 mL)

| Name | Type of CHO* | Energy (kcal) | CHO (g) | CHO [†] Concentration (%) | Na (mg) | K (mg) | Other |
|---|--------------|---------------|---------|------------------------------------|---------|--------|--|
| Fluid/Electrolyte Replacement Drinks | | | | | | | |
| Water | | 0 | 0 | 0 | Low | Low | Depends on source |
| All Sport Zero | | <40 | <1 | <1 | 56 | 60 | Vitamins C and E; niacin; |
| Propel Zero | | | 0 | 0 | 80 | 0 | B ₆ ; pantothenic acid |
| Fluid/Fuel Drinks | | | | | | | |
| Accelerade | S, F, GP | 80 | 14 | 6 | 140 | 57 | Vitamin E and C; Ca, |
| All Sport | F | 60 | 16 | 7 | 56 | 60 | protein; and fat |
| Cytomax | GP, F | 60 | 14 | 6 | 90 | 40 | Vitamins C, B ₃ , B ₆ , B ₁₂ |
| Gatorade Thirst | G | 54 | 14 | 6 | 108 | 30 | Vitamins A and C; Ca, Mg, Cr |
| Quencher | GP, F | 26 | 7 | 3 | 95 | 11 | Vitamin C, Mg, Cl, P |
| GU Brew | F, GP | 70 | 17 | 7 | 25 | 50 | Vitamin C, B ₃ , B ₆ , B ₁₂ , B ₁₃ , |
| Powerade Ion 4 | F, G | 60 | 14 | 7 | 20 | 235 | A, E, Mg, P, Zn |
| Body Armor | | | | | | | |
| Recovery/Replenishment Drinks | | | | | | | |
| Chocolate milk* | G | 150–210 | 25–26 | 9–10 | 160–190 | 420 | Fat, protein (8 g; 3%), vitamins |
| Gatorade protein | G | 194 | 32 | 14 | 227 | 482 | A and C; Ca, Fe |
| Shake | G | 90 | 8 | 6 | 125 | 100 | Protein (14 g; 6%), Ca |
| GU Recovery | GP | 165 | | 4 | 262 | 640 | Protein (3 g; 1%), Ca; Fe, Mg |
| Muscle Milk | F | | | | | | Fat, protein (16 g, 32%), |
| Protein Nutrition | | | | | | | vitamins A, C, D, E, B ₆ , B ₁₂ , |
| Shake | | | | | | | Ca, Fe, Cr, Cu, I, Mg, Zn |

*F, fructose; G, glucose; GP, glucose polymer; S, sucrose.

[†]% concentration = [CHO (g)] ÷ [volume (mL)] × 100, rounded to nearest whole percentage.

‡Content varies between low-fat and whole milk.

Note: Some values may vary slightly based on flavors; all values are rounded to the nearest whole number.

Source: Manufacturer's Web sites and product labels.

Fox et al. (2004) studied the impact of fat calories added to meals after exercise on glycogen resynthesis and glucose tolerance and found that as long as the meals ingested in the hours after exercise contain the same amount of CHO, the addition of fat neither helped nor hindered muscle glycogen resynthesis. However, there is evidence that co-consumption of dietary supplements, including caffeine and creatine, alongside CHOs may improve the rate of glycogen resynthesis (Pedersen et al., 2008; Roberts et al., 2016).

If CHOs are not replenished between training bouts, local muscle fatigue will occur, and work output during succeeding training sessions will decline (Costill, 1988; Coyle, 1991). Severe depletion followed by a nonoptimal diet will require more than 1 day of rest, which is one reason to alternate body parts and hard or easy exercise days. CHO intake can also have a significant positive effect by counteracting the suppressed immune function and inflammatory processes that occur during and after intense,

prolonged exercise (Ivy and Ferguson-Stegall, 2014).

Due to known physiological responses resulting from the manipulation of CHO availability, the concept of nutritional periodization has received much attention. **Nutritional periodization** is the intentional manipulation of nutritional intake to optimally influence training adaptations (Jeukendrup, 2017). There are a variety of methods to periodize nutrition, one of which is “train low-compete high” (Bartlett et al., 2015). In the “train lowcompete high” (meaning low CHO availability during training but high CHO during competition) variation, selected training sessions are deliberately started with reduced CHO availability, but competition starts with high CHO availability. A growing body of scientific evidence has demonstrated that reduced CHO availability can promote training-induced adaptations in human skeletal muscle. These adaptations include increased maximal mitochondrial enzyme activity and/or mitochondrial content, increased rates of lipid oxidation, and in some instances, improved exercise capacity. Marquet et al., demonstrated that following a sleep “low” strategy for 1 week in trained cyclists resulted in improved performance compared to cyclists who evenly distributed CHOs throughout the week (Marquet et al., 2016a). Assuming that the individual does not have a chronically low CHO diet, such sessions could still occur, for example, after an overnight fast, during 2 per day training when CHO is not ingested between sessions, or if CHO intake is withheld during the first few hours of recovery. The periodization aspect of this technique must be emphasized owing to the potential negative responses to a consistent lack of CHO. Performing repeated training sessions with reduced CHO may lead to decreased training intensity, immunosuppression, muscle protein breakdown, and an impairment in the ability to subsequently oxidize ingested CHO. To minimize these possible outcomes, it is suggested that individuals should consume caffeine prior to these workouts and/or practice CHO mouth rinse during exercise to help maintain exercise intensity. During mouth rinsing, a CHO solution is taken into the mouth, swished around, and spit out. Although the research has not been unanimous, enough studies have shown improved performance over placebo trials to recommend this procedure. It is thought that there may be receptors in the mouth that sense the availability of CHO and

communicate this to the brain immediately, thereby facilitating corticomotor output to skeletal muscle (Cermak and van Loon, 2013). Protein should be ingested before, during, and/or immediately after exercise to enhance muscle protein synthesis. The practice of training low should be alternated with sessions of training high where the intended competition-nutrition schedule (CHO loading, preexercise meal that includes CHO, and CHO during exercise) is followed to maintain CHO oxidation ability. The “train low-compete high” training technique is currently a widely accepted topic among coaches, athletes, and sport scientists. However, both the optimal approach to follow and the extent of performance enhancement have yet to be elucidated (Bartlett et al., 2015; Marquet et al., 2016b).

Nutritional Periodization The intentional manipulation of nutritional intake to optimally influence training adaptations.

Although most individuals would benefit from increasing their percentage of CHO ingested, the levels listed above are recommended only for athletes or fitness participants who are actually using high amounts of CHO in their training regimens. Ingesting 70% CHOs has little advantage and possibly some risk, for nonendurance athletes such as golfers, softball or baseball players, fitness walkers, or sedentary individuals, especially if much of this food has a high-glycemic index and the diet has a high-glycemic load (high GL). As mentioned earlier, a high GL can result in temporary hyperglycemia and an increased insulin response. Because glycogen storage is limited in sedentary and non-endurance-trained individuals, the glycolytic pathway is overloaded. The result is a greater-than-normal reliance on a side pathway that converts the glucose to free fatty acids (and then triglycerides) and cholesterol (Costill, 1988; Newsholme and Leech, 1983). Absorption of the same amount of low-glycemic CHOs results in a smaller blood glucose and insulin rise and thus less lipid formation. High blood levels of triglycerides and LDL cholesterol are frequently associated with an increased risk of cardiovascular disease. The long-term consumption of a diet with a relatively high GL (adjusted for total energy) has been shown to

be associated with increased risk of type 2 diabetes, cardiovascular disease, breast and colon cancer, and obesity (Foster-Powell et al., 2002). However, the relationship between the consumption of high-glycemic index foods and certain diseases is not universally agreed upon in the literature. For example, a recently published meta-analysis concluded that there is no compelling evidence suggesting that low GI diets have protective effects against cardiovascular diseases or most cancers (Clar et al., 2017; Turati et al., 2019).

Endurance-trained individuals demonstrate less hyperglycemia and a lower insulin response to a given glucose load than do untrained individuals. Endurance-trained individuals thus appear to convert high dietary levels of CHO, especially high-glycemic foods, into glycogen storage without an elevation of blood lipids.

Three categories of CHO sources have been developed that are specifically marketed for active individuals and athletes: sports drinks, bars, and gels.

Sports Drinks

Liquid CHO sources are as effective as solid CHO sources for glycogen replenishment. In fact, liquids may be even more useful, because many individuals are not hungry after an intense bout of exercise but are willing to drink liquid, and fluid replacement is also important (Betts and Williams, 2010; Costill, 1988; Coyle, 1991; Jentjens and Jeukendrup, 2003). Along with chocolate milk, **Table 6.4** compares several currently available commercial sports drinks. Note that the drinks have been categorized as fluid/electrolyte replacement drinks, fluid/fuel replacement drinks, and recovery/replenishment drinks. Water is included for comparison. Many more with different formulas are available. Note also that sports drinks are not the same as “energy drinks” or “energy shots,” which are marketed for their mental stimulatory effects and sudden “bursts” of energy. Energy drinks typically contain caffeine, carbohydrate, and herbal ingredients such as (but not limited to) ginseng extract, green tea extract, yerba mate, S-HTP, and St. John’s wort extract. These ingredients in proprietary blends may or may not be safe or useful for active individuals (Campbell et al., 2013; Duchan et al., 2010; Gurley et al., 2015). Although some sports drinks contain low amounts of the

stimulant caffeine, they are designed to maximize fluid absorption and support performance or recovery by delivering water, electrolytes, CHO, and, in some cases, protein. Sports drinks with appropriate amounts of CHO and electrolytes (especially sodium and potassium) aid in the maintenance of homeostasis, prevent injuries, delay fatigue, and optimize performance (Coombes and Hamilton, 2000; von Duvillard et al., 2008). Those manufactured by reputable companies have generally been tested and found to be both safe and effective (Seebohar, 2007). CHO may be in the form of glucose or glucose polymer (maltodextrin), fructose, or sucrose (glucose + fructose), either alone or in combination. The longer the endurance event, the more important the inclusion of a variety of CHO forms is. Physiologically, this is related to the way in which glucose is transported from the small intestine. Glucose is transported across the membrane of the intestinal epithelium using the sodium-dependent glucose transporter SGLT1. This transporter becomes saturated when glucose intake is around $60 \text{ g}\cdot\text{hr}^{-1}$. Fructose is transported by GLUT5 and is independent of the saturation of SGLT1. Thus, when glucose and fructose are ingested in combination, total delivery of CHO can be increased from 1.0 to $1.2 \text{ g}\cdot\text{min}^{-1}$ to as high as $1.75 \text{ g}\cdot\text{min}^{-1}$. This occurs only when glucose/glucose polymers are already being consumed at the near maximal rates of approximately $1.2 \text{ g}\cdot\text{min}^{-1}$. All monosaccharides are transported into the circulation by GLUT2 (Baker and Jeukendrup, 2014; Cermak and van Loon, 2013; Jeukendrup, 2013; Marieb and Hoehn, 2018; Hall and Hall 2021). The concentration (%) of CHO in these selected sports drinks varies from less than 1–17% in the different categories of drink. In general:

- A CHO concentration of less than 4% is the most useful for workouts lasting less than an hour and in situations where hydration, not substrate availability, is the primary concern. A mouth rinse may be sufficient. Single or multiple transportable CHOs may be used.
- CHO concentrations of 6–8% are optimal for use during workouts longer than 60 minutes when both fluid and fuel need to be supplied. The optimal amount of CHO is approximately $30 \text{ g}\cdot\text{hr}^{-1}$ for 1–2 hours of activity, $60 \text{ g}\cdot\text{hr}$

-1 for 2–3 hours of exercise, and 90 g·hr⁻¹ for events longer than 2.5 hours for well-trained endurance or intermittent sport athletes working at moderate to high intensity. Single or multiple transportable CHOs may be used for sessions under 2.5 hours. Over 2.5 hours, only multiple transportable CHOs are recommended (Jeukendrup, 2014). Individuals exercising at lower intensity do not need such high CHO ingestion (Cermak and van Loon, 2013). CHO concentrations greater than 8% are best for recovery situations when glycogen replenishment is the primary goal. The inclusion of sodium, CHO, and protein in postexercise fluid ingestion also stimulates rehydration and retention of ingested fluids (Baker and Jeukendrup, 2014).

The processing of exogenous CHO is independent of body or muscle mass so fluid amounts do not need to be recommended on a per kg mass basis. A key to selecting a sports drink is to find one that tastes good to the individual and is well tolerated. These guidelines will be the same for competition as for training, but it is vitally important that they be practiced in training sessions before being used in competition.

Sports Bars

Table 6.5 provides the macronutrient breakdown for selected sports bars and a Snickers candy bar for comparison purposes. Sports bars (**Figure 6.4**) provide readily available CHO and fall into two generic categories: (1) high CHO (>60% of total calories) with minimal fat and protein and (2) minimal to moderate CHO (20–55% of total calories) with balanced fat and protein (~22–40% of each) (Applegate, 1998; Manore, 2000). High-CHO bars are suitable for ingestion before, during, and after exercise. CHO in the form of glucose plus fructose in a sports bar plus water is effectively utilized during exercise (Pfeiffer et al., 2010b). Fat consumed during exercise is not readily available for energy; moreover, fat slows digestion and can lead to gastrointestinal distress. Bars with 4 g or less of fat (per 230 kcal serving size) are fine for workouts, but bars with higher fat content are best used as dietary supplements or snacks after

exercise.

TABLE 6.5 Composition of Selected Sports Bars

| Name | Energy (kcal) | CHO | | Fat | | Protein | | Fiber (g) | Vitamins/Minerals |
|-------------------------------------|------------------|-----|------|-----|------|---------|------|-----------|--------------------------|
| | | (g) | (~%) | (g) | (~%) | (g) | (~%) | | |
| Balance Bar™ | 200 | 22 | 44 | 7 | 32 | 14 | 28 | 2 | 23 |
| Clif Bar™ | 250 | 42 | 67 | 6 | 22 | 10 | 16 | 5 | 24 |
| Clif Luna™ | 180 | 29 | 64 | 5 | 25 | 8 | 18 | 4 | 24 |
| EAS Myoplex 30® | 360 | 33 | 37 | 12 | 30 | 30 | 33 | 1 | 19 |
| Met-Rx Prime Bar® | 210 | 29 | 57 | 5 | 20 | 20 | 38 | 12 | 6 |
| PowerBar Performance Energy Bar® | 240 | 45 | 75 | 3 | 11 | 8 | 13 | 3 | 6 |
| PowerBar ProteinPlus® | 330 | 40 | 49 | 9 | 25 | 30 | 36 | 5 | 2 |
| PR-Bar® | 200 | 22 | 44 | 7 | 32 | 15 | 30 | 1 | 16 |
| Snickers Bar® | 250 | 33 | 53 | 12 | 43 | 4 | 6 | 1 | Ca, Fe, vitamins A and C |
| Snickers Marathon Energy Bar® | 210 | 22 | 42 | 5 | 21 | 11 | 21 | 7 | 22 |

*Values may vary slightly by flavor; based on nutrients, some caloric values appear to be underestimates.

Sources: Based on manufacturers Web sites and product labels.



Figure 6.4 Ingestion of Carbohydrate or CHO and Protein before, during, and after Exercise Training Sessions Can Be Accomplished by Ingesting Specially Formulated Sports Drinks and/or Eating Sports Bars with Water.

Before or during exercise, it is best to select sports bars with no more than 8–10 g of protein because higher protein amounts also slow digestion. Likewise, bars with more than 5 g of fiber should not be ingested before or during exercise because fiber slows digestion. However, high-fiber bars can be good choices for snacks because fiber delays hunger pangs. If a sports bar is to be used as a meal replacement, one with high protein should be selected. Similarly, high-protein bars can be used in recovery.

The quantity of vitamins and minerals included in many sports bars ranges from 5 to 100% of suggested daily values. Whenever a bar is eaten, it is important to drink at least 350–475 mL (12–16 oz.) of water to aid digestion. Sports bars can serve as a convenient, effective fuel source, but they are engineered food and should not completely replace a healthy natural food diet.

Sports Gels

Another type of packaged food for active individuals is the sports gel. Sports gels are products with a consistency of syrup or pudding and come in small plastic or foil packets. They contain roughly 20–25 g (~100 kcal) of easy-to-digest CHOs per serving. Some gels contain electrolytes (especially potassium) and/or caffeine, but none contains fat (**Table 6.6**). Gels are best consumed every 30–45 minutes during longer training sessions and races lasting longer than 60 minutes. An interesting variation on the gel is the sports jelly bean. These are similar in nutrient content to the other semiliquid gels but may be less messy to consume. Any number of “chews” are available as well. As with sports bars, it is important to ingest sufficient water (8 oz. per packet of gel) and to try out a variety of gels during training to determine individual reactions before ingesting any during competition. A glucose plus fructose mixture of CHO ingested as a gel (plus water) leads to similar oxidation rates and efficiencies as a liquid CHO mixture (Pfeiffer et al., 2010a).

TABLE 6.6 Composition of Selected Sports Gels

| Name | Type of CHO* | Energy (kcal) | CHO | | Fat | | Protein | | Fortification |
|---------------------------|--------------|---------------|-----|-----|-----|-----|---------|-----|---|
| | | | (g) | (%) | (g) | (%) | (g) | (%) | |
| Accel Gel | GP | 90 | 20 | 88 | 0 | 0 | 5 | 22 | Vitamins C and E; Na, K |
| Gu | GP, F | 100 | 23 | 92 | 0 | 0 | 0 | 0 | Caffeine (some flavors), Na, K |
| PowerBar Gel | GP, F | 110 | 27 | 100 | 0 | 0 | 0 | 0 | Caffeine, Na, K |
| Jelly Belly™ Sports Beans | S, G | 100 | 25 | 100 | 0 | 0 | 0 | 0 | Vitamins B ₁ , B ₆ , B ₁₂ , C, Na, K |

*F, fructose; G, glucose; GP, glucose polymer; S, sucrose.

Note: Some contents may vary based on flavors.

Sources: Manufacturers' Web sites and product labels.

Protein

The optimal amount of dietary protein for individuals engaged in exercise training and competition has long been debated but remains controversial. The RDA of protein varies among countries for adults (United States and Canada: $0.8 \text{ g}\cdot\text{kg}^{-1}$ for male and female; Australia: $1.0 \text{ g}\cdot\text{kg}^{-1}$ for male and female; and the Netherlands: $1.0 \text{ g}\cdot\text{kg}^{-1}$ for female and $1.2 \text{ g}\cdot\text{kg}^{-1}$ for male) and within countries for various age groups. For example, the U.S. and Canadian RDA decreases from $0.95 \text{ g}\cdot\text{kg}^{-1}$ for 4 to 13 year olds to $0.85 \text{ g}\cdot\text{kg}^{-1}$ for 14 to 18 year olds. Pregnant or lactating females are encouraged to ingest higher levels of protein (Kominiarek and Rajan, 2016).

The reluctance of nutritional experts to recommend increased protein intake relative to body weight for physically active individuals is because it is assumed that the increase in calories to match those expended will also include an increase in protein to sufficiently cover any additional exercise-induced needs. However, in several situations, this assumption is questionable (Campbell et al., 2007; Kreider et al., 2010; Phillips and Van Loon, 2011), as discussed in the following sections. In all situations, in order to maximize adaptations from training sessions, the exerciser needs to rehydrate, replenish (muscle glycogen), and repair (damaged protein) in the postexercise period.

Resistance Training

The first situation in which a higher protein intake might be beneficial is training with the primary goal of a large increase in

muscle mass (Kreider et al., 1993; Lemon, 1989a, 1989b; Lemon and Nagle, 1981; Phillips and Van Loon, 2011). Individuals involved in resistance training—such as weightlifters, powerlifters, body builders, football players, sprinters, and wrestlers—strive for increased muscular strength and/or hypertrophy. Training of this type typically increases protein in the muscle fibers (see Chapter 19).

After an acute bout of dynamic resistance training, both muscle protein synthesis and breakdown rates are increased. Synthesis increases by approximately 50–100%, while breakdown increases less than 50%, resulting in a positive turnover. However, net protein balance remains negative unless protein/amino acids are provided soon after the exercise ends. Thus, both the nutritional content and the timing of postexercise caloric intake are important (Bird et al., 2006; Fielding and Parkington, 2002; Ivy and Ferguson-Stegall, 2014). The availability of protein/amino acids immediately after exercise (within 1 hour) probably maximizes muscle protein synthesis (Lemon, 2000; Phillips, 2013; van Loon, 2014; Wolfe, 2006).

Figure 6.5A–C shows the results of a study comparing supplement ingestion either just before or just after finishing a workout (PRE/POST) with ingestion in the morning before breakfast and late evening before bed (MOR/EVE). The supplement consisted (per 100 g) of 40 g of whey isolate protein, 43 g of glucose, less than 0.5 g of fat, and 7 g of creatine monohydrate. Although all subjects were familiar with resistance training, they underwent an 8- to 12-week structured program before being divided into two groups and adding the supplement component. The 10-week resistance program progressed from 70 to 75% 1-RM to 80–85% 1-RM and then to 90–95% 1-RM. As can be seen in **Figure 6.5**, the PRE/POST group improved their body composition, strength, and fast-twitch muscle fiber (IIa and IIx) hypertrophy more than did the MOR/EVE group (Cribb and Hayes, 2006).

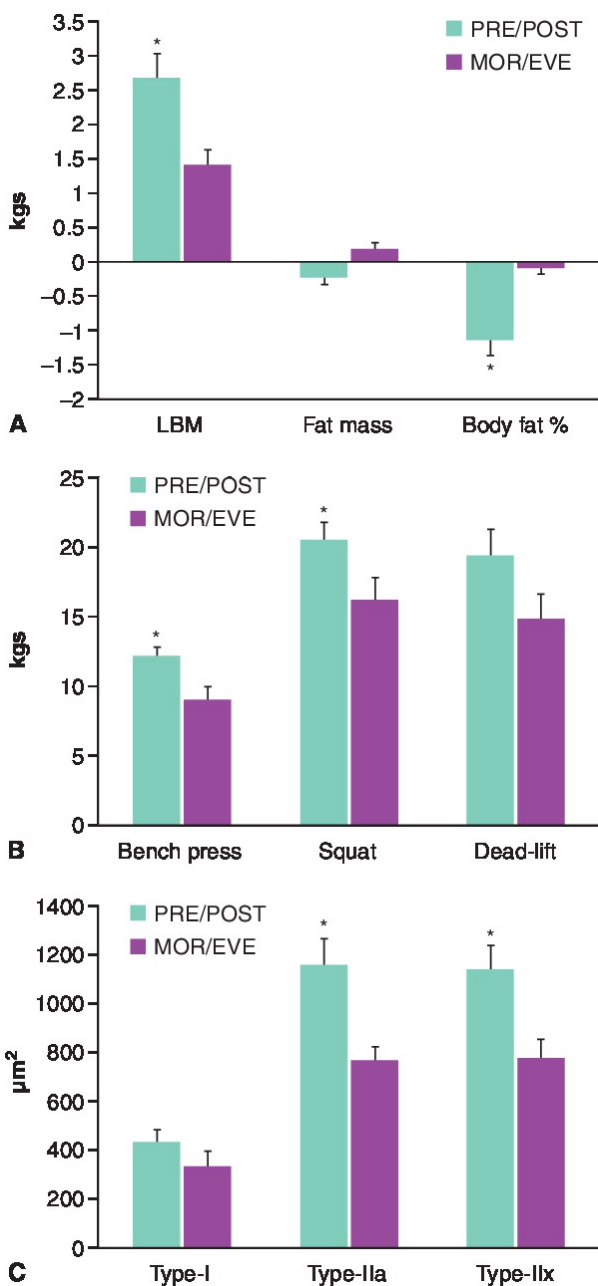


Figure 6.5 The Influence of Supplement Timing.

Changes in body composition (A), strength (B), and muscle fiber size (C) when CHO, protein, and creatine monohydrate

are ingested immediately before and after (PRE/POST) a resistance training session compared with early morning and late evening (MOR/EVE). $*P < 0.05$. **Source:** Reprinted with permission from Cribb, P. J., & A. Hayes: Effects of supplement timing and resistance exercise on skeletal muscle hypertrophy. *Medicine & Science in Sports & Exercise*. 38(11):1918–1925 (2006). Copyright ©2006 The American College of Sports Medicine.

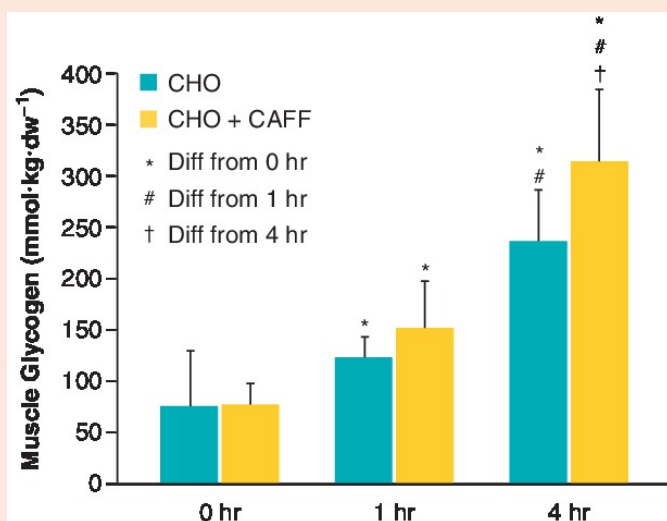
On the other hand, ingestion of 20–40 g of protein prior to sleep has been shown to increase plasma amino acid availability, stimulate postexercise protein synthesis, and improve whole-body protein balance during the night (Ivy and Ferguson-Stegall, 2014; van Loon, 2013).

FOCUS ON RESEARCH

Caffeine and Glycogen Replenishment

Caffeine is mostly known for its ergogenic properties. However, caffeine alongside CHOs has also been shown to improve rates of glycogen resynthesis following exercise compared to just CHOs. This study included seven trained endurance cyclists/triathletes who were cycling over 250 km·wk⁻¹. The exercise intervention was as follows. To control for exercise and diet, subjects performed a cycling workout until volitional exhaustion followed by a low CHO meal (1.2 g·kg BW⁻¹ CHO, 0.8 g·kg BW⁻¹ protein, and 1.4 g·kg BW⁻¹ fat) on the evening prior to exercise testing. On the day of testing, participants cycled until volitional exhaustion at 70% $\text{VO}_2 \text{ max}$ followed by supplementation of either CHO (4 g·kg BW⁻¹) or CHO + caffeine (same amount of CHO + 8 mg·kg BW⁻¹ of caffeine). Muscle biopsies were obtained from the vastus lateralis to measure concentrations of glycogen immediately after exercise (0 hour) and 1 and 4 hours postexercise.

Immediately following exercise to volitional exhaustion, glycogen levels were depleted to approximately $80 \text{ mmol}\cdot\text{kg}^{-1}$, with no differences between groups. One hour following, glycogen concentrations were increased approximately 80% in both groups, with no differences between groups ([CHO] $133 \pm 38 \text{ mmol}\cdot\text{kg}^{-1}$ vs. [CHO+caffeine] $149 \pm 48 \text{ mmol}\cdot\text{kg}^{-1}$). However, at 4 hours postexercise, glycogen concentrations were significantly higher in CHO+caffeine ($313 \pm 69 \text{ mmol}\cdot\text{kg}^{-1}$) versus CHO ($234 \pm 50 \text{ mmol}\cdot\text{kg}^{-1}$). Consumption of caffeine postexercise alongside CHOs may be an effective strategy to increase rates of glycogen resynthesis.



Source: Reprinted with permission from Pedersen D. J., S. J. Lessard, V. G. Coffey, E. G. Churchley, A. M. Wootton, T. Ng, M. J. Watt, & J. A. Hawley: High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coingested with caffeine. *Journal of Applied Physiology*. 105(1):7–13 (2008). Copyright © 2008 The American Physiological Society. All rights reserved.

Current recommendations for protein intake and resistance training include the following:

- Ingest 0.4 g·kg BW⁻¹ of easily digestible PRO immediately postexercise to maximize muscle protein synthesis.
- Ingest 0.25 g·kg BW⁻¹ PRO every 4–5 hours (20–25 g with each main meal) to maintain high rates of muscle protein synthesis.
- Ingest 0.25 g·kg BW⁻¹ aiming for 40 g PRO prior to sleep.
- Ingest a total daily protein intake of 1.6 g·kg BW⁻¹ and up to 2.2 g·kg BW⁻¹ to maximize muscle protein accrual.
- For those following a hypocaloric diet, higher quantities of daily dietary protein (2.3–3.1 g·kg BW⁻¹) may be necessary to maximize lean mass retention.

The amino acid composition is another consideration. Although complete protein with all amino acids is important in the overall diet, the essential amino acids have been shown to be the primary regulators of muscle protein synthesis. The branched chain amino acids (BCAAs) and particularly leucine appear to be the most important stimulators of skeletal muscle protein synthesis ([Campbell et al., 2007](#); [Phillips and Van Loon, 2011](#); [Volek, 2004](#); [Wolfe, 2006](#)). Because leucine seems to be the most important amino acid for stimulating muscle protein synthesis, it is suggested that individuals consume 2–3 g of leucine per meal to maximally stimulate muscle protein synthesis ([Jäger et al., 2017](#)). As an example, this would equate to 20–30 g of whey protein, which has roughly 10% leucine content. There is no evidence to support a recommendation of necessity for supplements of protein. Food protein, especially highquality dairy protein that contains the essential amino acids including leucine, may be more effective than other protein sources ([Phillips, 2013](#)). Whey (20%) and casein (80%) are the primary proteins in milk. Whey is the fluid portion of milk; casein is the curd. Both whey and casein components are highly digestible, have a high amino acid content, and have the ability to increase muscle protein. Whey protein elicits a sharp, rapid increase of plasma amino acids, especially leucine, which activates muscle protein synthesis. Due to its coagulation in the stomach, casein protein elicits a moderate, prolonged increase in plasma amino acids that is sustained for hours and hence enables sustained stimulation of protein synthesis ([Campbell et al., 2007](#); [Wolfe, 2015](#)). The recommended increases in protein ingestion for resistance

athletes vary somewhat, but generally fall between the values of 1.2–2.0 g·kg⁻¹·d⁻¹ of protein. This amount should be possible within the recommended 10–35% of total caloric intake (ACSM et al., 2009; Brotherhood, 1984; Haymes, 1983; Lemon, 1989a, 1989b, 1991; Tarnopolsky et al., 1988). For bodybuilders, during the energy-restriction phase of their training, 30% protein intake is recommended (Lambert et al., 2004) to reduce the loss of lean body mass (LBM). There is minimal scientific evidence that amino acid supplementation in excess of the additional amount needed to maintain a positive nitrogen balance enhances muscle hypertrophy or strength attainment (Lemon, 1989a, 1991). Highly trained resistance athletes may require less protein than novice lifters and only slightly more than the RDA (Fielding and Parkington, 2002; Lemon, 2000).

Endurance Training

The second situation in which increased protein might be beneficial is endurance training. When an individual begins a training program, a situation called **sports anemia** (sometimes called *sports pseudoanemia*) may develop. This is a transient decrease in red blood cells and hemoglobin level (grams per deciliter, g·dL⁻¹). During the initial 2–3 weeks of a training program, blood proteins, including erythrocytes (red blood cells, RBC), may be used to increase the myoglobin concentration, mitochondrial mass, and enzymes that are part of the training adaptation. In runners, RBCs may be destroyed by a mechanical trauma, called *foot strike hemolysis* (Hinton, 2014; Schumacher et al., 2002). Other possible causes of intravascular hemolysis (especially in swimmers and cyclists) include muscle contraction, dehydration, free radical actions, and the inflammatory response (Robinson et al., 2006). A higher intake of dietary protein may minimize the destruction of red blood cells, promote their regeneration, and provide the protein needed for the other training adaptations (Robinson et al., 2006).

Sports Anemia A transient decrease in red blood cells and hemoglobin level (grams per deciliter of blood).

Alternatively, sports anemia may be explained in terms of plasma volume expansion rather than protein (RBC) degradation. If plasma volume increases but hemoglobin and red blood cells do not (or they do not increase proportionally), a dilution effect will occur, resulting in decreased hemoglobin and red blood cell concentrations. Research supports that blood volume changes do occur. However, the two phenomena, increased dilution and protein degradation, are not mutually exclusive. Furthermore, research evidence suggests that experienced and not just novice endurance athletes may also suffer sports anemia ([ADA, 1987](#); [Haymes, 1983](#); [Hinton, 2014](#); [Lemon, 1989b](#); [Tarnopolsky et al., 1988](#)).

In addition, high-intensity, long-duration training and competition result in increased amino acid oxidation as fuel, ranging from 5 to 15% of the total calories used, especially if the individual is depleted of CHO ([Brotherhood, 1984](#); [Gleeson, 2005](#)). Cool-temperature training (5–20°C or 40–68°F) also utilizes more protein than does warm-weather (30°C or 86°F) training. Females utilize more protein during exercise in the midluteal phase of the menstrual cycle (~days 14–21) than during the follicular phase (~days 1–7) ([Phillips, 1999](#)).

Finally, skeletal muscle protein turnover increases at rest in healthy adults undergoing an aerobic exercise training program. In contrast to resistance training, a more negative net muscle protein balance (synthesis breakdown) has been shown with endurance training. In addition, plasma amino acid concentration decreases suggesting that 0.80 g·kg⁻¹·d⁻¹ is insufficient to optimize muscle protein despite adequate caloric energy intake ([Pikosky et al., 2006](#)). Protein availability is critical for maximizing muscle adaptations in response to aerobic endurance training, including increasing synthesis in relation to breakdown, so that the net muscle protein balance is positive, favoring synthesis ([Hawley et al., 2006](#)).

There is some evidence that the combined ingestion of CHO and PRO prior to and during prolonged ultraendurance exercise (>3–5 hours) may be beneficial. Such ingestion may prevent excessive muscle protein breakdown and allow muscle protein synthesis to be elevated during exercise. This could facilitate muscle reconditioning and improve training efficiency. On the

other hand, the combination could slow the delivery of CHO and fluid during the exercise session and cause gastrointestinal distress. Obviously, this is something the individual athlete needs to practice (Ivy and Ferguson-Stegall, 2014; van Loon, 2014). The impact of alcohol ingestion on the benefits of protein intake on postexercise muscle synthesis in college-aged individuals is presented in the Focus on Application box. Immediate postexercise ingestion recommendations are the same for endurance training individuals as for resistance training individuals: 20–25 g or 0.25 g·kg BW⁻¹ easily digestible PRO (Kerksick et al., 2017). Overall a small increase in the intake of dietary protein to 1.2–1.4 g·kg⁻¹·d⁻¹ is recommended for high-intensity, long-duration aerobic endurance training (ACSM et al., 2009; Kato et al., 2016). This increased amount of protein per kilogram of body weight, for high-intensity, long-duration aerobic endurance training, can be achieved without increasing the percentage above the recommended 10–35% of the total calories ingested (Lemon, 1989a; Paul, 1989). See the accompanying example for how this might be done.

Example

Calculate the protein intake for a 56-kg (123-lb) female in triathlete before she began training (pre) with her current training intake.

Pretraining caloric intake:

$$\begin{aligned} 1,500 \text{ kcal} \cdot \text{d}^{-1} \times 0.12 \text{ (12\% protein)} &= 180 \text{ kcal PRO} \\ 180 \text{ kcal PRO} \div 4 \text{ kcal} \cdot \text{g}^{-1} &= 45 \text{ g PRO} \\ 45 \text{ g PRO} \div 56 \text{ kg BW} &= 0.8 \text{ g} \cdot \text{kg}^{-1} \text{ of protein} \end{aligned}$$

In-training caloric intake: 2,250 kcal·d⁻¹

If she maintains 12% protein ingestion, the calculation becomes

$$\begin{aligned} 2,250 \text{ kcal} \cdot \text{d}^{-1} \times 0.12 &= 270 \text{ kcal PRO} \\ 270 \text{ kcal PRO} \div 4 \text{ kcal} \cdot \text{g}^{-1} &= 67.5 \text{ g PRO} \\ 67.5 \text{ g PRO} \div 56 \text{ kg BW} &= 1.2 \text{ g} \cdot \text{kg}^{-1} \text{ of protein} \end{aligned}$$

If she increases her PRO content to 20% of her increased calories

$$2,250 \text{ kcal} \cdot \text{d}^{-1} \times 0.20 = 450 \text{ kcal PRO}$$

$$450 \text{ kcal PRO} \div 4 \text{ kcal} \cdot \text{g}^{-1} = 112.5 \text{ g PRO}$$

$$112.5 \text{ g PRO} \div 56 \text{ kg BW} = 2.0 \text{ g} \cdot \text{kg}^{-1}$$

FOCUS ON APPLICATION | *Clinically Relevant*

Postexercise Alcohol Ingestion and Muscle Protein Synthesis

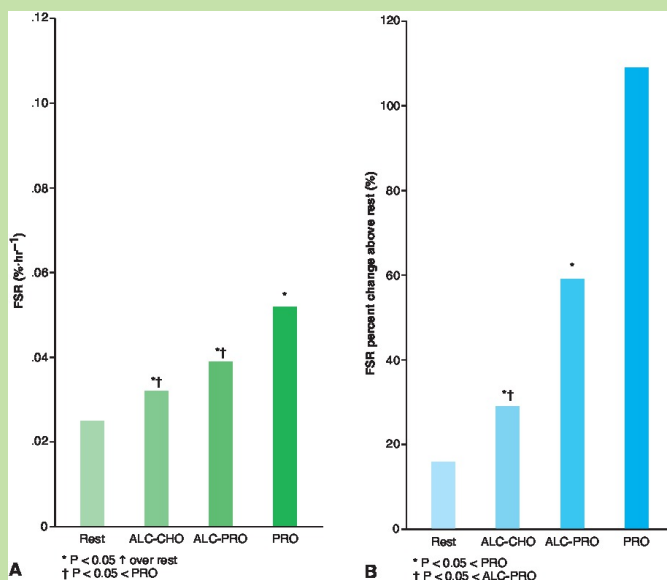
Alcohol ingestion contributes up to 5% of total caloric intake in the population and 9% of total caloric intake specifically in adults who drink ([Dietary Guidelines for Americans, 2020](#)). The cultural environment surrounding some sports often involves the intake of large amounts of alcohol after training and/or competition. A study was conducted to compare the effect of only protein (PRO), alcohol plus protein (ALC-PRO), and alcohol plus CHO (ALCCHO) ingestion on postexercise muscle (myofibrillar) protein synthesis.

Eight physically active males (\bar{X} age = 21.4 ± 4.8 years) completed three exercise trials in a randomized cross-over design. Each exercise trial consisted of resistance exercise (8×5 reps leg extension at 80% 1-RM), continuous cycling (30 minutes at 63% of individually determined peak power), and high-intensity interval cycling (10×30 seconds at 110% individually determined peak power). Each trial was separated by 14 days and preceded by a standardized meal. Immediately and 4 hours postexercise, subjects ingested either 500 mL (25 g) of whey protein (PRO & ALC-PRO conditions) or 25 g maltodextrin CHO (ALC-CHO trial). Alcohol ingestion (ethanol, $1.5 \text{ g} \cdot \text{kg BW}^{-1}$) began 1 hour postexercise and was consumed in six equal volumes of 60 mL (2 oz.) vodka plus 240 mL (8 oz.) orange juice once every

30 minutes for the next 3 hours. At 2 hours postexercise, all subjects received a CHO-rich meal ($1.5 \text{ g} \cdot \text{kg BW}^{-1} \text{ CHO}$). Blood samples indicated that blood alcohol peaked at $0.06 \text{ g} \cdot 100 \text{ mL}^{-1}$ in both the ALC-CHO and ALC-PRO trials at 4 hours postexercise and remained elevated ($0.05 \text{ g} \cdot 100 \text{ mL}^{-1}$ ALC-CHO; $0.03 \text{ g} \cdot 100 \text{ mL}^{-1}$ ALC-PRO) at 8 hours postexercise. Muscle biopsies were obtained preexercise, 2 hours postexercise, and 8 hours postexercise.

The results are shown in the accompanying graphs. Panel A presents the (myofibrillar) muscle protein synthesis rates measured as FSR (functional synthesis rate). Muscle protein synthesis increased significantly above rest for all conditions. However, compared to PRO alone, there was a significant reduction in FSR for the ALC-PRO and even more so for the ALC-CHO trial. Panel B presents these differences in percentage. The percent increase in FSR for PRO alone was 109%, 57% for the ALC-PRO trials, but only 29% for the ALC-CHO trial.

These results provide clear evidence of impaired muscle recovery when a large amount of alcohol is consumed postexercise in the presence of optimal nutritional conditions otherwise. The negative effects of alcohol on muscle protein synthesis have also been shown by others with lower doses ($1.09 \text{ g} \cdot \text{kg BW}^{-1}$) of alcohol intake. For a 77 kg (170-lb) male, this would equate to 6 drinks. This may impair not only recovery but also adaptation to training and/or subsequent performance.



Source: [Duplanty et al. \(2017\)](#); [Parr et al. \(2014\)](#).

The key point is that the total caloric intake must be increased. If the total energy intake is insufficient, then the percentage allotted to protein may be inadequate. For example, if the athlete increased her caloric intake to only 1,875 kcal·d⁻¹ at 12% protein intake, this increase would amount to only 1 g·kg⁻¹·d⁻¹. Also, if the athlete increased her CHO intake to 70%, she would need to carefully select the remaining 30% of her foodstuffs to get an adequate protein intake.

The problem of ingesting too few calories and thus inadequate protein is especially evident when athletes are concerned with percentage of body fat or making weight in a sport with weight categories. The problem is compounded in children and adolescents. As previously mentioned, individuals at this age need more protein than do adults. Inadequate protein intake at this age might adversely affect not only exercise performance but also growth ([Lemon, 1991](#); [Steen, 1994](#)). Of course, if the extra protein ingested is also excess calories, body weight and fat will increase, and that also is not usually desirable.

Older Individuals

For older adults, inadequate nutrition and inadequate protein intake can also be a problem. Older women (ages 60 and older), on average, consume below the recommended amount of protein rich foods (Brown et al., 2019; Dietary Guidelines for Americans, 2020). In addition, the current RDA of $0.8 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$ seems insufficient to meet the protein needs of most elderly. Older individuals require more dietary protein than do younger adults to support good health, promote recovery from illness, and maintain functionality. They need to make up for age-related changes in protein metabolism such as declining anabolic responses to ingested protein and resistance exercise (Nowson and O'Connell, 2015; Phillips, 2014; Wall et al., 2014). The decline in anabolic response provided by a particular anabolic stimulus as a result of aging is known as **anabolic resistance**. This can be overcome by ingesting foods that are rich in protein and have adequate leucine content, especially in close temporal proximity to physical activity (Wall et al., 2014). Given adequate protein, rates of muscle protein synthesis (MPS) is the same in older versus younger adults. However, the protein requirements to maximize the protein synthetic response is greater in older adults. Moore et al., demonstrated that older adults required a single protein ingestion of approximately $0.40 \text{ g} \cdot \text{kg} \text{ BW}^{-1}$ of protein to maximize MPS while younger adults achieved the same rate of MPS at $0.24 \text{ g} \cdot \text{kg} \text{ BW}^{-1}$ of protein (Moore et al., 2015). Therefore, protein recommendations for older adults should be higher than younger individuals. If considering protein synthetic rates, those recommendations can be nearly twice those of younger adults. A 2017 study demonstrated that consuming twice the RDA for protein ($1.6 \text{ g} \cdot \text{kg} \text{ BW}^{-1} \cdot \text{day}^{-1}$ of protein) significantly increases arm and leg (appendicular) lean mass and lower body power in older adults compared to eating the RDA ($0.8 \text{ g} \cdot \text{kg} \text{ BW}^{-1}$) (Mitchell et al., 2017).

Anabolic Resistance The decline in anabolic response provided by a particular anabolic stimulus as a result of aging.

Randomized controlled studies clearly show a benefit of increased dietary protein on lean mass gain and leg strength when combined with resistance exercise in those greater than 65 years. If insufficient protein is ingested, the body adapts by breaking down lean muscle mass to maintain nitrogen balance, which ultimately results in a cascade from the loss of muscle mass to frailty, falls, and a lower quality of life (Nowson and O'Connell, 2015). The European Union Geriatric Society, in cooperation with other scientific organizations, appointed an international study group to review dietary protein needs for individuals greater than 65 years. This group recommended:

- An average daily protein intake of at least $1.0\text{--}1.2\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ with $25\text{--}30\text{ g}$ being ingested per meal
- An intake of $1.2\text{--}1.3\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for individuals engaged in either or both endurance and resistance training
- An intake of $1.2\text{--}1.5\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for individuals at risk of malnutrition because of acute and chronic diseases (except those with kidney and other specific diseases) (Bauer et al., 2013; Deutz et al., 2014)
- Adequate total calories intake to be ingested along with the increased protein

Before dietary protein is increased, the individual should undergo a complete dietary nutritional analysis under the guidance of a trained nutritionist. It is important to note that the US RDA for adults of any age remains at $0.8\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$. However, no adverse events have been reported for the recommended reasonable increases in protein intake for those older than 65 years given above (Wolfe, 2012).

Can too much protein in the diet be harmful to a fitness participant or competitive athlete? Although concern has been expressed that excessive protein intake ($>3\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$) may have negative effects including increased blood lipid levels, kidney damage, and dehydration (Gleeson, 2005), there is no substantive evidence that protein intakes in the suggested ranges for active individuals will have adverse effects in healthy, exercising individuals (Campbell et al., 2007; Jäger et al., 2017). In individuals with preexisting liver or kidney abnormalities, a high-protein diet can lead to further deterioration of the function

(ADA, 1987; Campbell et al., 2007; Lemon, 1989b). However, a recently published meta-analysis concluded that kidney function, in regard to glomerular filtration rate, is no different in health adults when comparing high- and low-protein diets (Devries et al., 2018).

The metabolism of protein requires more water than does the metabolism of CHO or fat. Increased excretion of nitrogen in urine increases urinary volume and the risk for dehydration. Therefore, water intake should be increased to avoid dehydration if protein intake is increased.

There is also some concern regarding the impact of high-protein intake on calcium loss. Early studies reported evidence in sedentary individuals that increasing the dietary protein leads to increased calcium excretion, suggesting a loss in bone calcium (Allen et al., 1979; Campbell et al., 2007; Eisenstein et al., 2002; Lemon and Nagle, 1981). It is now known that the phosphate content of protein food and supplements fortified with calcium and phosphorous negates this effect. Data from isotope studies suggest that the main source of the increase in urinary calcium from a high-protein diet is intestinal (dietary) and not from bone resorption. So, as with the concern for kidney function, there appear to be no adverse outcomes to increased protein intake within the recommended levels (up to $2 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$) in healthy, exercising individuals (Campbell et al., 2007; Devries et al., 2018).

Fat

As discussed in Chapters 2 and 4, fat is a major fuel for exercise of low or moderate intensity, and in most individuals, it is readily available in more-than-adequate stored amounts. In addition, studies have documented (Chapter 5) that with endurance training, more fat can be utilized as a fuel, thereby sparing CHO stores, and can be used at higher absolute levels of work intensity.

This increased ability to utilize fat and spare CHO helps postpone fatigue in endurance and ultra-endurance events. There has been a great deal of interest in whether a diet high in fat could enhance the glycogen sparing effect that occurs with

training.

To be useful during training, a high-fat diet (defined as 60–70% FAT) must allow continuation of the intended training levels and progression in training adaptation through periodic tests or time trials. There is evidence that a relatively high training intensity has been maintained on a high-fat diet for at least 7 weeks in previously untrained and moderately trained individuals and for at least a short time in elite athletes. However, at all levels of training, higher ratings of perceived exertion (RPE) were reported at constant exercise loads ([Fleming et al., 2003](#); [Helge, 2000, 2002](#)). **Figure 6.6** provides an example of the effects of a high-fat diet on oxygen consumption compared to a high-CHO diet in elite race walkers. At the start of the study, there were no differences in oxygen consumption between the participants. (A) After 3 weeks of a high-fat ketogenic diet (< 50 g CHO/day), oxygen consumption increased at similar exercise intensities compared to baseline oxygen consumption indicating impaired exercise efficiency. (B) After 3 weeks of a high CHO diet (8.6 g CHO·kg⁻¹·day⁻¹), there were no differences in oxygen consumption at similar exercise intensities compared to baseline oxygen consumption.

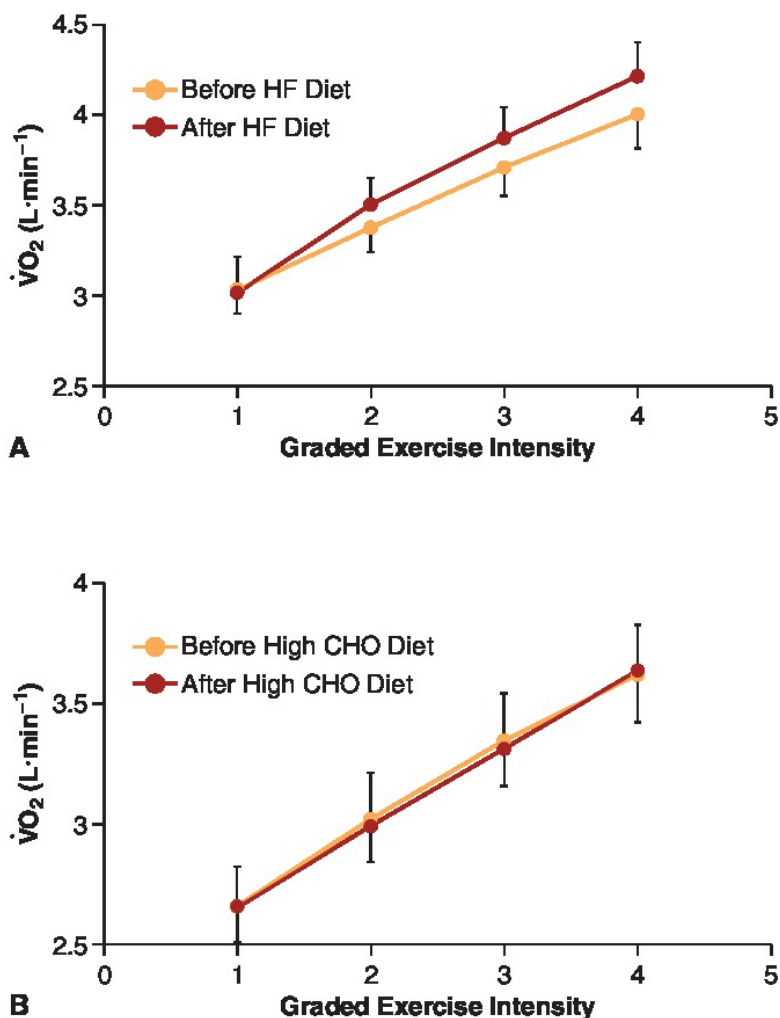


Figure 6.6 The Impact of a High-Fat Ketogenic Diet versus a High-CHO Diet on Oxygen Consumption during a Graded Exercise Test before and after 3 Weeks of Training and Dietary Manipulation.

A. After 3 weeks of a high-fat ketogenic diet, oxygen consumption increased at similar exercise intensities compared to baseline oxygen consumption indicating impaired exercise efficiency. **B.** After 3 weeks of a high-CHO diet, there were no differences in oxygen consumption at similar exercise intensities compared to baseline oxygen

consumption).

Adaptation to a fat-rich diet does lead to an increased ability to store, mobilize, transport, and utilize fat. There is also evidence that the storage of muscle glycogen and liver glycogen is lower with high-fat diets. As a result, more fat is oxidized during submaximal exercise, presumably sparing CHO compared with an equal-calorie high-CHO diet, even if CHO is ingested immediately before or during the exercise (Burke and Hawley, 2002; Helge, 2000, 2002). However, there generally appears to be no particular performance advantage to these metabolic shifts. The exercise performance of those consuming a high-fat diet is at best equal to, or in some cases lower than, that of those consuming a more traditional high-CHO training diet (ACSM et al., 2009, 2016; Burke and Hawley, 2002; Fleming et al., 2003; Helge, 2000, 2002; Jacobs et al., 2004; Vargas-Molina et al., 2020; Vogt et al., 2003). **Figure 6.7** shows the results of 7 weeks of training on either a high-fat or a high-CHO diet on a time-to-exhaustion test. Performance improved significantly in both groups. However, the high-CHO diet group improved significantly more than did the high-fat diet group (Helge, 2002). Additionally, there is evidence that higher fat diets may negatively impact exercise economy, increasing oxygen requirements at similar exercise intensities compared to high CHO availability in elite athletes (Burke et al., 2020).

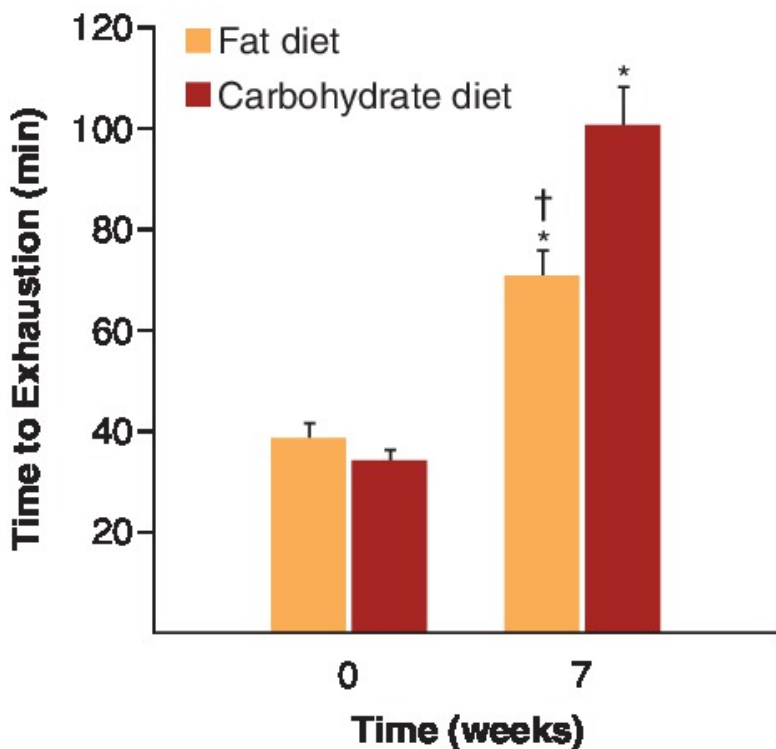


Figure 6.7 The Impact of a High-Fat Diet versus a High-CHO Diet during Training on Submaximal Exercise Performance.

Before training and dietary manipulation, there was no significant difference in the time to exhaustion between the two groups. After 7 weeks, the high-CHO group exercised for significantly longer than did the high-fat group ($*P < 0.05$, significant difference pre vs. post 7 weeks of training; $\dagger P < 0.05$ fat vs. CHO diet). **Source:** Reprinted by permission from Springer Helge, J. W.: Adaptation to a fat-rich diet: Effects on endurance performance in humans. *Sports Medicine*. 30(5): 347–357 (2000). Copyright © 2012 Springer Nature.

Obviously, these high-fat diets are far above the recommended daily intake for health. The primary concern is that high-fat diets

lead to unhealthy lipid profiles. Surprisingly, based on the few available studies, welltrained individuals who maintain that training seem to have no adverse effects in their lipid/lipoprotein profiles when consuming a high-fat diet over periods of up to 3 months. However, when training is not maintained or when a high-fat diet is consumed for longer, there probably are detrimental effects in general health (Helge, 2000). Thus, given the lack of any beneficial training effects and the real possibility of eventual negative health effects, a high-fat diet is not recommended for fitness or athletic training.

On the other hand, no attempt should be made to totally remove fat from the diet (ACSM et al., 2009, 2016; Kreider et al., 2010; Sherman and Leenders, 1995). Diets in which less than 20% of the energy is provided from fat increase the risk of inadequate intakes of vitamin E, α -linolenic acid, and linoleic acid and for adverse changes in HDL cholesterol and triglycerides. What then should fat intake be for active individuals?

- There is no established RDA in $\text{g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for fat but 0.5–1.5 $\text{g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ has been recommended (Kreider et al., 2010).
- As indicated on **Table 6.1**, 65–80 $\text{g}\cdot\text{d}^{-1}$ is a reasonable level for a total caloric input of 2,000–2,500 $\text{kcal}\cdot\text{d}^{-1}$ or proportionally more ($\sim 3 \text{ g}\cdot 100 \text{ kcal}^{-1}$) for higher caloric intakes in athletes.
- Although diets that include $\geq 20\%$ fat are adequate for sedentary individuals, it may be that well-trained athletes doing endurance training should not drop below 30% fat in their diet.

It is important to optimize the type of fat ingested. Most fats should come from polyunsaturated and monounsaturated fatty acid sources such as fish, nuts, and vegetable oils. Two servings of fish per week are recommended—with the caution that women of childbearing age, pregnant women, nursing mothers, and young children should avoid fish and shellfish that are likely to contain high levels of mercury. No more than 10% of dietary fat should be saturated, regardless of the activity level (Burke and Read, 1989; Leaf and Frisa, 1989). Restrictions on cholesterol intake level have been lifted because available evidence shows no

appreciable relationship between dietary cholesterol intake and serum cholesterol level. Trans fatty acid consumption should be as low as possible. As of June 2018, partially hydrogenated oils (the largest source of artificial trans fats) are no longer Generally Recognized As Safe (GRAS) and are no longer added to foods ([Dietary Guidelines for Americans, 2020](#)). Some trans fats do occur naturally in dairy foods and meat but are not considered a health concern ([U.S. Food and Drug Administration, 2015](#)).

Complete the [Check Your Comprehension 2—Case Studies A](#) and B to apply your knowledge of nutrition for athletes.

CHECK YOUR COMPREHENSION 2—CASE STUDIES

Complete the recommended nutrient intake for each of the following athletes in the open boxes. Check your answer in [Appendix C](#).

- A. Female tennis player, 21 years, 117 lb. Total caloric estimated daily need from **Table 6.2** = 2,400 kcal

| | | | |
|--|-------|--------------|------------|
| Situation: Training 10:00–12:00 AM; 6:00–7:30 PM | Grams | Kilocalories | Percentage |
| CHO | | | |
| Protein | | | |
| Fat | | | |
| Situation: Immediate postexercise recovery | Grams | Kilocalories | Percentage |
| Carbohydrate | | | |
| Protein | | | |
| Fat | | | |
| Suggest specific drinks, bars, and/or gels from Tables 6.4 to 6.6. | | | |

B. Male marathon runner, 35 y, 145 lb. Projected finish time 3:15

| | | |
|--|-------|--------------|
| Situation: 24 hr prior to event | Grams | Kilocalories |
| CHO | | |
| Situation: 1–4 hr prior to event | Grams | Kilocalories |
| CHO | | |
| Protein | | |
| Fat | | |
| Situation: 5 min prior to event | Grams | Kilocalories |
| CHO | | |
| Protein | | |
| Fat | | |
| Situation: During the event | Grams | Kilocalories |
| CHO | | |
| Suggest specific drinks, bars, and/or gels from Tables 6.4 to 6.6. | | |

Vitamins

Thirteen compounds are now considered to be vitamins. **Vitamins** are organic substances of plant or animal origin that are essential for normal growth, development, metabolic processes, and energy transformations. The B complex vitamins have two major roles directly related to exercise: (1) involvement in energy production (see [Chapter 2](#) Focus on Application: Metabolic pathways: The vitamin connection) and (2) tissue synthesis and repair. For example, vitamin B6 is important in amino acid metabolism and the breakdown of glycogen to glucose. Thiamin (B1) is important in CHO metabolism. Riboflavin (B2) and niacin are important as the hydrogen carriers FAD and NAD, respectively. B12 is needed for the production of red blood cells and protein synthesis. Vitamin D enhances calcium absorption in the intestines and is essential for normal calcium metabolism. Roles for vitamin D have been established in the function of the cardiovascular, immune, and musculoskeletal

system. Ascorbic acid (vitamin C) is necessary for the formation of connective tissues and the catecholamine hormones (important in the stress response) and for the maintenance and function of blood vessels. Vitamin E influences the flow of electrons within the mitochondrial respiratory chain. Both C and E are important in protecting cell membranes from oxidative damage (see [Chapter 5 Focus on Application: Oxygen-Free Radicals, Exercise, Exercise Training Adaptations, and Antioxidant Supplements](#)) ([ACSM et al., 2009](#); [Belko, 1987](#); [vander Beek, 1985, 1991](#)). See animation, *Biological Function of Vitamins*, on Lippincott Connect

Vitamins Organic substances of plant or animal origin that are essential for normal growth, development, metabolic processes, and energy transformations.

Vitamin D is obtained from only a few foods naturally or from fortified foods such as milk, juices, and cereals. It is synthesized in the skin through sunlight exposure. Athletes living far from the equator or those without sun exposure may need supplemental vitamin D during the winter ([Moran et al., 2013](#)). It appears that low vitamin D status in adolescent athletes can impair performance and increase the risk of injury. In this situation, monitoring of vitamin D status should be undertaken and correction of any deficiency be undertaken through increased sun exposure or supplementation ([Desbrow et al., 2014](#)). Some evidence suggests that exercise training may increase the need for vitamin C (especially in hot climates), B complex vitamins (particularly B6 and B2, also especially in hot climates), and vitamin E at high altitudes and as antioxidant defense against free radicals ([Belko, 1987](#); [Blom et al., 1987](#); [Kreider et al., 2010](#); [vander Beek, 1985, 1991](#)). These increased needs, as with other nutrients, should be adequately covered if the exerciser increases his or her total caloric intake with a balanced diet. The increased needs also mean that athletes who are concerned with restricting body weight or body fat or making weight (gymnasts, dancers, figure skaters, divers, wrestlers, boxers, and jockeys) and who restrict caloric intake could be at risk for an inadequate intake of vitamins. For these individuals, a generic, one-a-day vitamin and

mineral tablet might be appropriate to ensure adequate intake (ACSM et al., 2009; Belko, 1987; Kreider et al., 2010). Athletes who participate in prolonged, strenuous exercise training should consume 100–1,000 mg of vitamin C daily (ACSM et al., 2009).

After decades of research, there is no evidence that vitamin supplementation improves an adequately nourished, healthy individual's exercise or athletic performance, speeds up recovery, or decreases injuries (ACSM et al., 2009; Belko, 1987; Haymes, 1983; Kreider et al., 2010; Lukaski, 2004). If a deficiency is present, supplementation to the normal physiological level can improve performance. This is another reason for a complete nutritional analysis for anyone training or competing. However, megadoses of vitamins are neither substitutes for vigorous training nor necessary for training adaptations (vander Beek, 1985, 1991). Furthermore, extremely large doses of either water- or fat-soluble vitamins can be toxic, can impair performance, and, more importantly, can cause health problems. Once again, if some is good, more is not necessarily better. For example, very high doses of vitamin C ($>1 \text{ g}\cdot\text{d}^{-1}$) have been linked with the formation of kidney stones, the breakdown of red blood cells (which causes loss of hemoglobin), and an impairment of sport performance (Braahkuis, 2012). Megadoses of niacin inhibit fatty acid mobilization and utilization during exercise, thereby increasing the rate of glycogen use (Nieman, 1990). Doses of antioxidants such as vitamin E that are too high could be pro-oxidative instead of antioxidative (ACSM et al., 2009, 2016). Furthermore, recent data suggest that high doses of antioxidants may negatively influence resistance training adaptations. Dutra et al., demonstrated that after a 10-week resistance training protocol those consuming vitamin C ($1 \text{ g}\cdot\text{day}^{-1}$) and vitamin E ($400 \text{ IU}\cdot\text{day}^{-1}$) did not experience positive body composition changes whereas those on placebo significantly increased muscle mass and decreased fat mass (Dutra et al., 2019).

Minerals

Minerals are elements, not of animal or plant origin, that are essential constituents of all cells and for many body functions. Minerals are classified as microminerals (trace elements) or macrominerals based on the amount in the body. Minerals are

important for bone density, energy metabolism, enzyme function, muscle contraction, oxygen transport, insulin regulation, and ATP composition, to name just a few functions.

Minerals Elements, not of animal or plant origin, that are essential constituents of all cells and of many functions in the body.

Microminerals

Of the 15 microminerals with DRI/RDAs ([Dietary Guidelines for Americans, 2020](#)), only 5 (zinc, chromium, copper, selenium, and iron) have been shown to be affected by or potentially beneficial for exercise training or performance.

Zinc plays a role in repair of muscle tissue and energy production among other things. Low dietary zinc has been shown to impair metabolic responses during exercise ([Lukaski, 2005](#)). Because exercise can cause a sizable loss of zinc in sweat and urine, training may lead to a zinc deficiency. However, excess zinc intake above the RDA ($11 \text{ mg}\cdot\text{d}^{-1}$ for adult males and $8 \text{ mg}\cdot\text{d}^{-1}$ for adult females) can result in an impaired immune response and decreased iron and copper absorption. Two studies with wrestlers have shown that zinc supplementation ($3 \text{ mg}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$) had a positive effect on hematological variables and moderated the inhibition of thyroid hormones and testosterone after exhaustive exercise ([Kilic et al., 2004, 2006](#)). Studies also indicate that zinc supplementation during training minimizes exercise-induced changes in immune function ([Kreider et al., 2010](#)). Evidence is insufficient, however, to conclude that zinc status or zinc supplementation affects exercise performance ([ACSM et al., 2009](#); [Campbell and Anderson, 1987](#); [Clarkson, 1991b](#); [Lemon, 1989b, 1991](#); [McDonald and Keen, 1988](#)).

Chromium has a role in maintaining proper CHO and lipid metabolism. There is speculation but little evidence that exercise and training may increase the requirements for chromium in part because exercise increases urinary chromium loss. Chromium has no RDA because data are insufficient for establishing these. Instead, daily AI values are $35 \text{ }\mu\text{g}\cdot\text{d}^{-1}$ for adult males and $25 \text{ }\mu\text{g}\cdot\text{d}^{-1}$ for adult females.

-1 for adult females. Speculation that chromium can increase muscle mass and decrease the percentage of body fat in conjunction with resistance training remains just that—speculation (Lefavi et al., 1992). Chromium supplementation (as chromium picolinate) has not been shown to have any positive effect on body composition in healthy individuals when taken alone or used in conjunction with an exercise program (Kreider et al., 2010; Volpe, 2008). Conversely, recent studies suggest potentially deleterious effects (Lukaski et al., 2007; Vincent, 2003).

There is no evidence that either selenium or copper has an impact on acute or chronic exercise responses, although copper may be lost in sweat. The RDA for copper is 890 $\mu\text{g}\cdot\text{d}^{-1}$ for adolescents and 900 $\mu\text{g}\cdot\text{d}^{-1}$ for adults. Selenium functions as an antioxidant, so the need for selenium is increased during exercise training without being linearly related to energy expenditure (Margaritis et al., 2005). Suboptimal selenium status has been implicated in a worsening of muscle function following eccentric muscle contractions (Miliadis et al., 2006), but it does not appear that selenium supplementation would be beneficial for exercise performance (Kreider et al., 2010; Volpe, 2008). The RDA for selenium is 55 $\mu\text{g}\cdot\text{d}^{-1}$ for male and female adolescents and adults. Intakes of selenium above 1,000 $\mu\text{g}\cdot\text{d}^{-1}$ can be toxic (Campbell and Anderson, 1987; Clarkson, 1991b).

Iron is required for the formation of hemoglobin and myoglobin as well as enzymes involved in energy production. Iron deficiency can be a problem for exercising or training individuals, especially for menstruating females. Iron deficiency occurs in three stages: (1) iron depletion or low-storage levels of iron; (2) iron deficiency erythropoiesis, which is an impairment of the ability to produce red blood cells; and (3) iron deficiency anemia or low hemoglobin levels ($<12\text{ g}\cdot\text{dL}^{-1}$ for females and $<13\text{ g}\cdot\text{dL}^{-1}$ for males). Iron deficiency anemia is also characterized by small, pale red blood cells; decreased iron levels in the blood; decreased iron stores; and increased total iron binding capacity as the body attempts to mobilize as much iron as possible. Iron depletion is not associated with reduced performance, and iron deficiency erythropoiesis has marginal impact. However, iron deficiency anemia definitely impairs performance. Lower hemoglobin levels mean lower oxygen

transport. Moderate levels of exercise do not appear to affect iron status.

Iron supplementation given to individuals with iron deficiency anemia consistently improves iron status and exercise performance (Kreider et al., 2010). Iron supplementation given to individuals with iron depletion or iron deficiency erythropoiesis shows variable but primarily nonsignificant changes in performance. Iron requirements for endurance athletes are increased by approximately 70% (ACSM et al., 2009); however, excessive iron intake can inhibit zinc and copper absorption and cause free radical production. The iron status of an individual should be ascertained before supplementation is given that exceeds the RDA (11 mg·d⁻¹ for adolescent males, 8 mg·d⁻¹ for adult males, 15 mg·d⁻¹ for adolescent females, and 18 mg·d⁻¹ for adult females). Supplemental iron should be taken with ascorbic acid (vitamin C) to enhance absorption. Iron status should be monitored every 4–6 weeks during supplementation (ACSM et al., 2009, 2016; ADA, 1987; Clarkson, 1990; Haymes, 1983; Hinton, 2014; McDonald and Keen, 1988; Zoller and Vogel, 2004).

Macrominerals

Macrominerals include calcium, chlorine, magnesium, phosphorus, potassium, sodium, and sulfur. As macromineral electrolytes, chlorine, potassium, and sodium are discussed in the section “Fluid Ingestion During and After Exercise” in Chapter 14. Sulfur has no direct importance for exercise.

The function of calcium in bone health is directly related to exercise training and is fully described in Chapter 16. Beyond its role in bone health, the relationship between exercise, training, and calcium level and supplementation is largely unknown (Clarkson, 1991a).

Magnesium is involved in oxygen uptake and energy production. Because magnesium is lost in sweat and urine, strenuous exercise may increase the need for magnesium by 10–20%. Some evidence suggests that a marginal magnesium deficit impairs exercise performance and increases oxidative stress (ACSM et al., 2009). Increased magnesium intake has beneficial effects on exercise performance in magnesium-deficient

individuals. However, supplemental magnesium has not been shown to increase performance in active individuals with adequate magnesium status (Kreider et al., 2010; Nielsen and Lukaski, 2006; Volpe, 2008). Levels above the RDA (400–420 mg·d⁻¹ for adolescent and adult males and 360–310 mg·d⁻¹ for adolescent and adult females) do not appear to be harmful (McDonald and Keen, 1988).

Several studies have shown that phosphate loading (in the form of sodium phosphate)—increasing phosphate ingestion for several days before an event—may improve performance by delaying the onset of anaerobic metabolism; other studies using a variety of phosphate formula have shown no beneficial effects (Kreider et al., 2010). Because phosphorus supplementation over an extended period of time can result in lowered blood calcium levels, it is not recommended. The RDA for phosphorus is 1,250 mg·d⁻¹ for adolescents and 700 mg·d⁻¹ for adult males and females (Dietary Guidelines for Americans, 2020).

In summary, the pattern here is the same for minerals as the one for vitamins. That is, neither microminerals nor macrominerals can be considered ergogenic aids. Exercisers should ingest the RDA/AI amounts and, unless diagnosed with a deficiency, only those amounts. For those who are strictly limiting their caloric intake, a generic vitamin-and-mineral one-a-day pill can be recommended.

Nutrition for Competition

The five goals for an optimal competitive diet are to

1. Ensure adequate fuel supplies in the pre-event time span
2. Ensure adequate fuel supplies during the event, regardless of its duration
3. Facilitate temperature regulation by preventing dehydration
4. Achieve the desired weight classification while maintaining fuel and water supplies
5. Avoid gastrointestinal discomfort during competition

Depending on the event, an athlete's diet may need to be adjusted during the hours or days before competition. Often, manipulation focuses on CHO consumption (ACSM et al., 2016; Burke and Read, 1989).

Carbohydrate Loading (Glycogen Supercompensation)

Individuals competing in continuous endurance events lasting at least 90 minutes at 65–85% $\dot{V}O_2 \text{ max}$ may use CHO loading, sometimes called glycogen supercompensation (ACSM et al., 2016; ADA, 1987; Brotherhood, 1984; Hawley et al., 1997). **Carbohydrate loading** is a nutritional modification that results in an additional storage of glycogen in muscle fibers that can be approximately two to three times the normal level. As mentioned previously, the time to exhaustion in long-duration, relatively high-intensity activities is related to initial levels of muscle glycogen.

Carbohydrate Loading (Glycogen Supercompensation) A process of nutritional modification that results in an additional storage of glycogen in muscle fiber up to two to three times the normal levels.

CHO loading techniques have been implemented as a strategy to help improve performance in endurance athletes. In this section, several CHO loading techniques that have been popularized over the years to help improve physical performance will be briefly highlighted.

One-Week CHO-Loading Technique

Popularized in the 60s, this technique involved hard exercise to deplete muscle glycogen stores 1 week before the competitive event, followed by 3 days of hard training and 3 days of rest. During the first 3 days after the depletion exercise, the individual ate almost no CHOs, and then for the next 3 days, almost

exclusively CHOs. This technique was effective but difficult and uncomfortable to follow (Bergstrom et al., 1967).

Short CHO-Loading Technique

For an athlete who might not want the strong taper or rest component, a short technique is available. In this version, the athlete either completes a normal training session the day before CHO loading (Bussau et al., 2002) or a supramaximal short depletion routine on the day of CHO loading (Fairchild et al., 2002). **Figure 6.8** shows a scan of muscle fibers packed with glycogen following a short CHO-loading protocol study (Fairchild et al., 2002). The Focus on Research: Clinically Relevant box describes the impact of increased glycogen storage on endurance exercise performance.

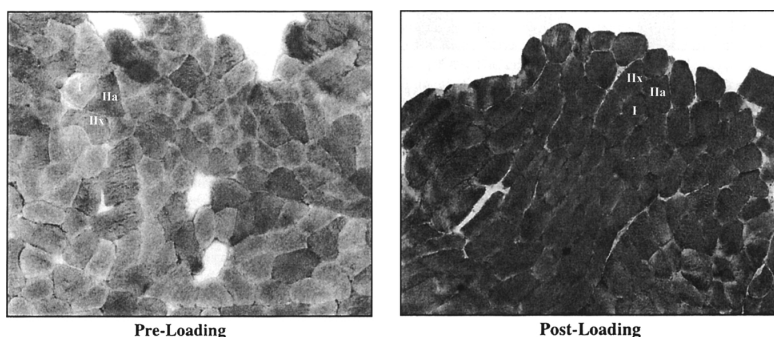


Figure 6.8 Glycogen Supercompensation.

Muscle fiber scan before and after CHO loading. The darker stain in the after scan indicates the increased glycogen storage. Note that glycogen supercompensation occurred in SO (Type I), FOG (Type IIa), and FG (Type IIX) fibers.

Source: Reprinted with permission from Fairchild, T. J., S. Fletcher, P. Steele, C. Goodman, B. Dawson, & P. A.

Fournier: Rapid carbohydrate loading after a short bout of near maximal-intensity exercise. *Medicine & Science in Sports & Exercise*. 34(6):980–986 (2002). Copyright ©2002 The American College of Sports Medicine.

Fat Plus CHO-Loading Technique

In a simple fat-loading technique, individuals ingest a high-fat diet from 1 to 6 days before an endurance event, which has been shown to actually decrease performance ([Burke and Hawley, 2002](#); [Helge, 2000](#)). Three days is too short a time to increase the capacity for fat utilization, and the resting levels of muscle glycogen at the start of an event are lowered. To counteract these shortcomings, a strategy has emerged, which involves 5–6 days of fat loading (60–70% fat in the diet) followed by 1 day of CHO loading (~90% CHO in the diet), which increases fat utilization at rest and during exercise, increases muscle glycogen stores, and reduces muscle glycogen utilization during exercise. However, as with the studies using high-fat diets in training, there has been no clear evidence of performance improvement. While carbohydrate loading techniques have primarily been used to improve endurance performance, they may also be implemented in other areas, such as bodybuilding.

Results from a study investigating the effects of CHO loading on male bodybuilders are presented in **Figure 6.9**. Girth measurements of the chest, waist, hips, thighs, arms, calves, and forearms were taken from 24 bodybuilders before and after weigh-ins. Nine of the 24 bodybuilders performed CHO-loading techniques, although specific methodology of the CHO-loading techniques were not provided in the article. The bodybuilders who performed CHO-loading techniques significantly increased the girth of several of the areas measured compared to those who did not perform CHO loading techniques ([de Moraes et al., 2019](#)). It may be necessary to increase total caloric intake as well as the increase in percentage of CHO to achieve an impact on physique appearance. It may also be beneficial to increase the CHO content of the diet in the last several weeks prior to a competition. If CHO loading is to be used, a trial run before competition (once the competitor has nearly reached competition leanness) should be attempted to develop an individualized strategy for competition ([Helms et al., 2014](#)).

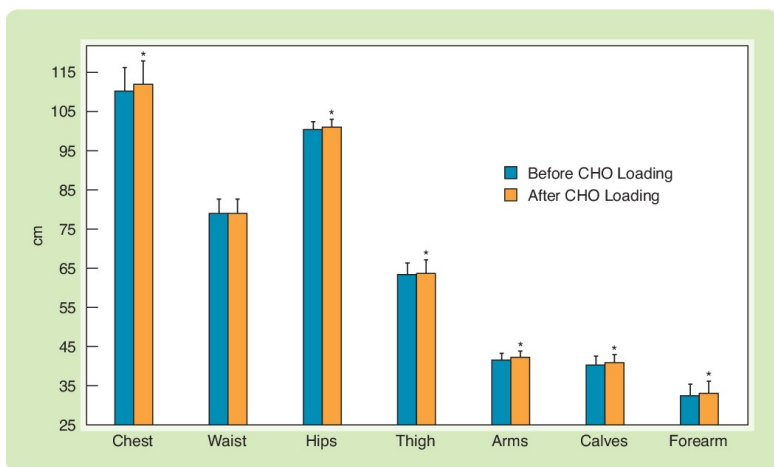


Figure 6.9 Girth Measurements in Body Builders before and after CHO Loading.

Following a 3-day CHO depletion period. Significant increases were found in chest, hips, thigh, arm, calves, and forearm girth after a 24 hours CHO loading protocol compared to before CHO loading. **Source:** Based on data from [de Moraes et al. \(2019\)](#).

Pre-Event Meal

Considerations for the pre-event meal involve the meal's timing and nutrient content. Most road races and other endurance events, unless scheduled for the convenience of a television broadcast such as during the Olympics, take place in the morning. Competing after an overnight fast is probably not effective for an athlete in any sport, but it is most detrimental for endurance athletes. Liver glycogen is the primary endogenous source of blood glucose, and an overnight fast reduces the liver's supply of glycogen. Although it has been demonstrated that fat oxidation can be maximized when an individual exercises in a fasted state, a high-CHO pre-event meal is generally accepted to benefit performance more than fasting ([O'Reilly et al., 2010](#)). This is despite the fact that getting up early and eating can be inconvenient. Athletes who compete in the afternoon or evening can more easily time their meals.

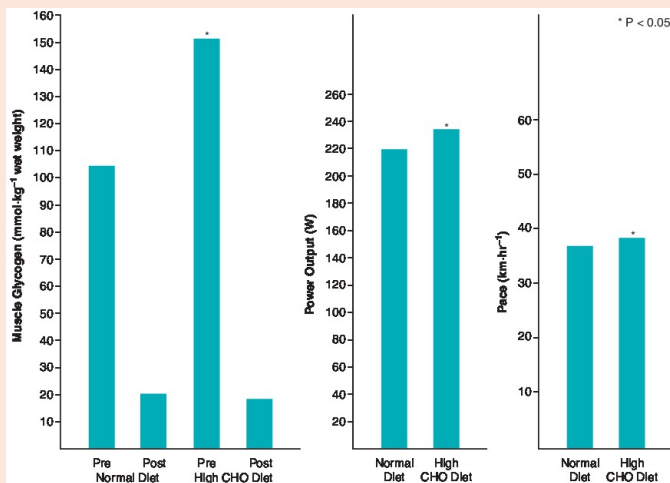
The Impact of Increased Glycogen Storage on Exercise Performance

In this study, well-trained male endurance cyclists completed two exercise trials separated by at least 4 days. Each trial consisted first of 2 hours of cycling at approximately 73%

VO₂ max. Every 20 minutes, an all-out 60-second sprint followed by 1 minute of unloaded cycling was interspersed. Immediately after the 2-hour ergometer ride, the athlete transferred to his own bike and completed a 1-hour time trial, blind to all data except elapsed time.

Dietary manipulation consisted of either 3 days of each athlete's normal diet or 3 days of a high-CHO diet. A standardized breakfast similar to what they would have eaten before a competition was ingested 3 hours before testing. During the 2-hour ride, each athlete drank 600 mL of a 10% glucose polymer solution and then only water during the time trial.

The results are shown in the accompanying graphs. Although the mean muscle glycogen level after the high-CHO diet was not as high as seen in many studies (151 mmol·kg⁻¹ wet weight vs. ~200 mmol·kg⁻¹ wet weight), it was significantly higher than after the normal diet. This enabled the cyclists to maintain a significantly higher power output and a faster overall pace during the time trial. Interestingly, muscle glycogen levels after the time trial were similar between groups despite different starting values and different performances. This indicates that the additional stored glycogen was indeed utilized during the time trial. Furthermore, although the term glycogen depletion is often used, glycogen concentrations do not actually reach zero at exhaustion.



Source: Rauch, H. G. L., A. St. Clair Gibson, E. V. Lambert, & T. D. Noakes: A signaling role for muscle glycogen in the regulation of pace during prolonged exercise. *British Journal of Sports Medicine*. 39:34–38 (2005).

Current recommendations for precompetition fueling for endurance, team, and/or explosive power events lasting more than 1 hour are to ingest 1–4 g·kg⁻¹ of primarily CHO food 1–4 hours before the start. Fiber, protein, and fat should be minimized to reduce the risk of gastrointestinal issues during the event (ACSM et al., 2016; Burke et al., 2011). The absence or presence of gastrointestinal distress can make the difference between a PR (personal record) or a DNF (did not finish). In addition, the ingestion of a small amount of CHO in the form of gel, sports beans, or chews within 5 minutes prior to the start can be beneficial (Volpe et al., 2013). For example, when Neuffer et al. (1987) fed participants 45 g of either liquid CHO, a candy bar, or flavored water containing no CHO 5 minutes before exercise, total work performed on a cycle ergometer was significantly improved after both the liquid CHO and candy bar compared with the flavored water. When a meal of 200 g of CHO (cereal, bread, and fruit) was eaten 4 hours before exercise in addition to a candy bar 5 minutes before the exercise, total work performed on the cycle ergometer was significantly greater compared with

all other trials.

Theoretically, ingestion of CHO from 1 hour to 5 minutes before exercise could be detrimental to performance for the following reasons. The ingestion of CHO causes an increase in blood glucose. The increase of blood glucose causes a concomitant increase in insulin. Insulin favors the removal of blood glucose from the bloodstream, inhibits the release of glucose from the liver, and inhibits the mobilization of free fatty acids, which are used, along with CHO, as fuel for exercise. These actions can cause hypoglycemia and may cause a greater dependency on muscle glycogen stores, depleting them at a faster rate. Despite the popular acceptance of this reasoning, the research literature has not supported the detrimental effects of meals within 1 hour of competition ([Burke and Read, 1989](#); [Burke et al., 2011](#); [Coyle, 1991](#); [Hawley and Burke, 1997](#); [Ormsbee et al., 2014](#); [Williams and Serratos, 2006](#)).

What type of CHO should be ingested in the preevent meal has been extensively investigated. Some studies have shown that low glycemic index meals have a beneficial impact. The common denominator in those studies is a combination of a greater rate of fat oxidation and more stable plasma glucose concentrations during exercise than after a HGI preexercise meal ([O'Reilly et al., 2010](#); [Williams and Serratos, 2006](#); [Wu and Williams, 2006](#)). Additionally, a LGI pre-event meal produces a longer-lasting feeling of satiety than a HGI meal. However, other studies have shown that the achieved metabolic differences did not translate into improved performances. In addition, when CHO is consumed during the event, it negates the effect of the glycemic characteristics of the pre-event meal ([Donaldson et al., 2010](#); [O'Reilly et al., 2010](#)). Therefore, the ingestion of a low glycemic index pre-event meal is likely to be most effective when it is difficult to consume CHO during exercise (such as in some team sports) or the individual is sensitive to a hyperinsulinemic response to CHO ([Burke et al., 2011](#)).

This pre-event meal should consist of foods that the individual likes and tolerates well and should have been practiced during training. **Table 6.7** summarizes this information.

TABLE 6.7 Recommended Eating on Game Day

| Sport | -1 to -4 hr | -2 hr | -5 to 0 min | Event Begins | +15 to +30 min Repeated for Duration of Event | Recovery (+15 to +30 min Post) |
|-----------------------|--|---|-------------------------|--------------|---|---|
| Team and Intermittent | Light meal of 1-4 g·kg ⁻¹ CHO -4 hr, fluid ingestion ~5-7 mL·kg ⁻¹ * | Fluid ingestion of ~3-5 mL·kg ⁻¹ | — | — | 30-60 g·hr ⁻¹ G Fluid ingestion as needed to prevent >2% BW loss | <8 hr to recover: 1.0-1.2 g·kg ⁻¹ CHO; 0.25 g·kg ⁻¹ PRO Fluid ingestion ~1.5 L·kg ⁻¹ BW fluid loss with Na ⁺ |
| Explosive power | Light meal of 1-4 g·kg ⁻¹ CHO -4 hr, fluid ingestion ~5-7 mL·kg ⁻¹ * | Fluid ingestion of ~3-5 mL·kg ⁻¹ | — | — | Fluid ingestion as needed to prevent >2% BW loss | 1.2-1.5 g·kg ⁻¹ CHO; 0.25 g·kg ⁻¹ PRO Fluid ingestion ~1.5 L·kg ⁻¹ BW fluid loss with Na ⁺ |
| Endurance | 1-4 g·kg ⁻¹ CHO -4 hr, fluid ingestion ~5-7 mL·kg ⁻¹ * | Fluid ingestion of ~3-5 mL·kg ⁻¹ | CHO ~50 g (optional) | — | 30-75 min: mouth rinse with CHO or no fuel; 1-2.5 hr: 30-60 g·hr ⁻¹ G; >2.5 hr: 90 g·hr ⁻¹ G; 2:1 G:F Fluid ingestion + CHO 6-8% concentration + ~20-30 mEq·L ⁻¹ Na ⁺ ~2-5 mEq·L ⁻¹ K ⁺ | <8 hr to recover: 1.0-1.2 g·kg ⁻¹ CHO; 0.25 g·kg ⁻¹ PRO Fluid ingestion ~1.5 L·kg ⁻¹ ·BW loss + electrolytes |

*If not already adequately hydrated.

G, glucose; F, fructose; CHO, carbohydrate; PRO, protein.

Feeding during Exercise

When exercise begins, insulin release is suppressed, and catecholamine secretion increases. Both theoretically and experimentally, there is support for the ingestion of CHOs during exercise provided the activity is prolonged (>60 minutes) and at moderate to high intensities (50-85% $\dot{V}O_2 \text{ max}$). Even small amounts of CHOs taken at 15- to 30-minute intervals have been found to prevent a decline in blood glucose and to delay fatigue during the latter portion of an endurance event longer than 1 hour. Note that fatigue can be caused by many factors, and CHO feeding merely delays but does not prevent fatigue. CHO feedings during intermittent exercise such as soccer or basketball have also been shown to be beneficial (Burke and Read, 1989; Cermak and van Loon, 2013; Costill, 1988; Coyle, 1991; Phillips et al., 2011; Williams and Serratos, 2006) although the opportunity to do so is difficult other than at half time.

As stated in the training portion of this chapter, the longer the endurance event, the more important the inclusion of a variety of CHO forms is. Recall that glucose is transported across the membrane of the small intestinal epithelium using the sodium-

dependent glucose transporter SGLT1. This transporter becomes saturated when glucose intake is around 60 g·hr⁻¹. Fructose is transported by GLUT5 and is independent of the saturation of SGLT1. Therefore, when glucose and fructose are ingested in combination using a 2:1 ratio of glucose to fructose, total delivery of CHO can be increased (Baker and Jeukendrup, 2014; Cermak and van Loon, 2013; Jeukendrup, 2013; Marieb and Hoehn, 2018). It does not matter whether the CHO is ingested as a liquid beverage (although this has the added advantage of providing hydration), solid, or gel. Thus, in general:

- For endurance events lasting 30–75 minutes or explosive power events, either no exogenous CHO supplementation is recommended or at most a mouth rinse of a CHO beverage.
- For endurance or intermittent sports lasting 1–2.5 hours, the optimal amount of CHO is approximately 30–60 g·hr⁻¹.
- For events lasting longer than 2.5 hours, 90 g·hr⁻¹ should be ingested. Single or multiple transportable CHOs may be used for sessions under 2.5 hours. Over 2.5 hours, only multiple transportable CHOs are recommended (Jeukendrup, 2014).

Research shows that beverages containing 2.5–10% CHO are absorbed by the body as rapidly as water. A concentration of 6–8% is thought to be optimal (ACSM et al., 2009; Kreider et al., 2010). Drinks containing less than 5% CHO do not provide enough energy to enhance performance, and drinks exceeding 10% CHO (such as most soft drinks/soda pop) are often detrimental since they tend to cause abdominal cramps, nausea, and diarrhea (Coleman, 1988). One high-CHO energy bar, 24 oz. of sports drink, and two packets of gel are equivalent. Depending on its composition, any of these can provide at least 30–60 g of CHO per hour during endurance exercise.

In general, HGI foods are probably best for ingestion during exercise. They are rapidly digested and absorbed and are thus likely to maintain blood glucose levels. LGI CHOs are digested more slowly and thus may cause gastric distress and fail to maintain blood glucose levels (Walton and Rhodes, 1997). The type of GI consumed during exercise has no bearing on performance outcome, per se. The GL may be a better predictor of

glycemic responses than GI alone. Some research suggests that it is the amount of CHO rather than the GI type that is the most crucial factor influencing subsequent endurance performance (Donaldson et al., 2010; O'Reilly et al., 2010).

A combination of CHO and protein does not improve performance beyond the ingestion of the CHO alone (van Loon, 2014).

Fluid Ingestion during and after Exercise

The background and guidelines for fluid ingestion during and after exercise are discussed in Chapter 14. Suffice it to say here that fluid intake is important for preserving thermoregulation and avoiding both dehydration and dilutional hyponatremia (loss of sodium). Individuals should develop a customized fluid replacement program during training to prevent dehydration. The goal is a body weight loss of less than 2% during activity. Table 6.7 summarizes general recommendations as a starting point for an individualized hydration program (ACSM, 2007).

Nutrient Timing

Although consuming adequate amounts of key nutrients like protein and CHOs is of utmost importance for individuals trying to maximize their physical performance, nutrient timing, which is a nutritional strategy to manipulate the time that specific nutrients are consumed to help achieve a desirable outcome, may be another important consideration. The International Society of Sports Nutrition suggests that strategically planning when certain nutrients are consumed may improve exercise recovery and mood, and increase muscle protein synthesis, which should ideally result in improved performance (Kerksick et al., 2017). CHO timing strategies mostly revolve around glycogen resynthesis following exercise as well as providing intra-workout fuel for prolonged exercise (>60 min). For rapid glycogen resynthesis, high-glycemic CHOs are recommended following exercise at doses near $1.2 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ coingested with caffeine, which has been shown to improve glycogen resynthesis. For prolonged aerobic work, consuming dilute solutions of mixed

CHO and electrolyte drink may be an effective strategy to fuel exercise.

Protein recommendations for individuals trying to maximize skeletal muscle mass have been covered in a previous section. However, consuming your total daily protein requirement, ideally evenly spaced throughout the day in 20–40 g amounts, is optimal for maximally stimulating rates of muscle protein synthesis, which should result in greater muscle accrual and improved physical performance. Furthermore, studies have indicated that consuming protein (~40 g) prior to sleep may be an effective strategy to elevate muscle protein synthesis overnight and does not negatively impact measures of metabolism (Kerksick et al., 2017; Leyh et al., 2018; Reis et al., 2021; Saracino et al., 2020).

The importance of consuming nutrients directly after a workout has been debated widely in the literature. An “anabolic window” was previously proposed as a short duration of time following exercise where ingestion of nutrients, particularly protein, was essential for maximizing training adaptations. However, the body of literature does not support this hypothesis. Data show that the response to exercise is more strongly influenced by an individual total daily caloric and protein intake, as well as the proximity of their last meal to exercise, rather than protein consumption immediately following training. On the other hand, a recently published review argues that nutrient timing around exercise should not be viewed as an “anabolic window,” rather a “garage door of opportunity” to fuel an athlete and improve performance. The authors argue that if nutrition is analogous to baking a cake, total daily energy and protein intake would be the base, and the nuances of nutrient timing would be analogous to the frosting and decorations (Arent et al., 2020).

Nutrient Timing A nutritional strategy to manipulate the time that specific nutrients are consumed to help achieve a desirable outcome.

Eating Disorders

Definitions and Diagnostic Criteria

Most individuals participating in fitness programs or athletics view nutrition as a partner with exercise to help them achieve their goals of health or successful competition. Unfortunately, this is not always the case. There appear to be circumstances under which exercise and sport participation constitute a risk factor for disordered eating. Disordered eating exists on a continuum from abnormal eating behaviors to clinically diagnosed eating disorders. Clinically diagnosed **eating disorders** are disturbances of eating habits or weight-control behavior that can result in significant impairment of physical health or psychosocial functioning (Smolak et al., 2000; Sudi et al., 2004).

Eating Disorders (ED) Disturbances of eating habits or weight-control behavior that can result in significant impairment of physical health or psychosocial functioning.

Of course, eating disorders do not occur only in physically active individuals, nor do all, or even most, physically active individuals have eating disorders. However, symptoms of eating disorders are more prevalent among adolescent and adult elite athletes than nonathletes (Martinsen and Sundgot-Borgen, 2013). In addition, a higher prevalence of eating disorders has been found among athletes competing in sports where leanness is advantageous compared with both athletes competing in sports where leanness is not particularly relevant and controls (Joy et al., 2016; Torstveit et al., 2008). Female athletes struggle more with eating disordered behaviors than do male athletes, but this may simply be because eating disorders are an underrecognized problem in male athletes. Male athletes are particularly vulnerable for eating disorders in the same categories of sports as females: (1) sports that emphasize aesthetics (gymnastics, dance, figure skating, diving, and bodybuilding); (2) gravitational sports where low body fat is advantageous (climbing, long-distance running, road cycling, and ski jumping); and (3) sports in which there is a need to make weight (wrestling, some martial arts, rowing, and horse racing). The culture of the latter weight class

sports, in particular, encourages bingeing and purging (Baum, 2006). In the past, males were more likely to actually have been overweight or obese before their eating disorders, whereas females more often simply perceived themselves as such (Sundgot-Borgen, 1993b, 1994a; Thompson and Sherman, 1993). More recently, there has been a rise in the number of young males who are preoccupied with their body image. This preoccupation has been labeled “*muscle dysmorphia*” or bigorexia and is characterized by a preoccupation with being lean and muscular to the point where it interferes with normal food intake, normal life activities, and reasonable exercise levels. Muscle dysmorphia is not listed by the American Psychiatric Association (2013) as an eating disorder and has no formal diagnostic criteria. That does not make it any less of a concern, however. Muscle dysmorphia in males often involves both disordered eating and anabolic steroid abuse (Baum, 2006; Goldfield et al., 2006). Not all males with eating disorders exhibit muscle dysmorphia. Pathological weight control and eating disorders are also evident in fitness instructors, including personal trainers (Höglund and Normén, 2002; Thompson and Sherman, 1993). This not only puts these individuals at personal risk but also jeopardizes the healthy attitudes and sound body ideals they should be communicating to their clients.

The American Psychiatric Association (2013) recognizes three major categories of clinically diagnosed eating disorders:

1. Anorexia nervosa (AN)
2. Binge eating disorder (BED)
3. Bulimia nervosa (BN)

The diagnostic criteria for AN and BN are listed in **Table 6.8. Anorexia nervosa**, often referred to as the self-starvation syndrome, is characterized by marked self-induced weight loss and an intense fear of fatness. **Binge eating disorder (BED)** was added as a condition in the Diagnostic and Statistical Manual of Mental Disorders, 5th ed. (DSM-5) (American Psychiatric Association, 2013) and is defined as recurring (at least once a week over a 3-month period) episodes of eating significantly more food in a short period of time than most people would

under similar circumstances and feeling out of control and/or guilty about it. Obviously, this would not lead to loss of weight. **Bulimia nervosa** is marked by an unrealistic appraisal of body weight and/or shape and is manifested by alternating bingeing and purging behavior. Other specified and/or unspecified feeding or eating disorders can be clinically diagnosed as well. Note that these definitions are from a psychiatric association, not from a nutritional or exercise professional association. Clinical eating disorders are psychiatric conditions that go beyond body weight/shape dissatisfaction and involve more than abnormal weight-control behaviors (Beals and Meyer, 2007). They are complex and multidimensional. Specifically, perfectionism, obsessiveness, harm avoidance, and low self-esteem have been found to accompany AN (Bachner-Melman et al., 2006). Such individuals have trouble identifying and expressing their emotions and have difficulty forming close interpersonal relationships (Beals and Meyer, 2007).

TABLE 6.8 Diagnostic Criteria and/or Characteristics for Selected Eating Disorders

| Anorexia Nervosa | Bulimia Nervosa | Anorexia Athletica* |
|---|---|--|
| Refusal to maintain minimally normal body weight for height and age; marked self-induced weight loss Intense fear of weight gain or becoming fat despite being underweight that includes persistent behavior that interferes with weight gain Severe body dissatisfaction and body image distortion, denial of seriousness of current low body weight | Recurrent episodes of binge eating (rapid consumption of large quantities of calorie-dense food, often secretly), feeling of lack of control while bingeing Purging or compensating for bingeing by self-induced vomiting, use of diuretics or laxatives, vigorous exercise, and strict food restriction or fasting Exhibiting bingeing or inappropriate compensatory behavior at least once a week for 3 months Severe body dissatisfaction, self-evaluation unduly influenced by body shape and weight | Fear of weight gain although lean Weight is $\geq 5\%$ below specified Muscular development maintains weight above AN threshold Distorted body image Restricted calorie intake often broken by planned binges Excessive or compulsive exercise above normal training needs Menstrual dysfunction Delayed puberty Secondary amenorrhea or oligomenorrhea Gastrointestinal complaints |

*Absolute criteria have not been agreed upon.

Sources: Based on American Psychiatric Association (2013); Currie and Morse (2005); Sundgot-Borgen (1994a, 1994b).

Anorexia Nervosa (AN) An eating disorder characterized by marked self-induced weight loss and an intense fear of fatness.

Binge Eating Disorder (BED) Recurring episodes of excessive eating and feeling out of control and/or guilty about it.

Bulimia Nervosa (BN) An eating disorder marked by an unrealistic appraisal of body weight and/or shape that is manifested by alternating bingeing and purging behavior.

Other eating problems are considered subclinical: they are a problem but do not meet formal diagnostic criteria for an eating disorder or show significant psychopathology. Athletes with eating disorders have been clinically observed to display less psychopathology than do nonathletes with eating disorders. For this reason, eating disorders in athletes may differ from eating disorders in nonathletes. The term **anorexia athletica (AA)** has been proposed to describe a subtype of anorexia, or subclinical disorder that affects athletes or other active individuals (Bachner-Melman et al., 2006; Sudi et al., 2004; Sundgot-Borgen, 1994a, 1994b). Anorexia athletica is characterized by a food intake less than that required to support the training regimen and a body weight at least 5% below normal. Additional characteristics of AA are included in **Table 6.8**. Despite years of research in this area, no definite criteria have been established for AA.

Anorexia Athletica (AA) An eating disorder that is characterized by a food intake less than that required to support the training regimen and by a body weight less than 95% of normal.

Individuals suffering from AA may exhibit a variety of symptoms, some of which are common to AN or BN (Bachner-Melman et al., 2006; Sudi et al., 2004; Sundgot-Borgen, 1994a, 1994b). Typically, the initial reduction in body mass/fat by dieting and/or excessive exercising in AA is based on the performance goals, not body appearance or shape issues. Appearance concerns, however, can develop, especially if the

athlete is not as successful in an aesthetic sport as anticipated or desired. The drive to lose weight can become a goal in itself, regardless of the negative impact on athletic performance. Weight cycling (repeated weight loss and regain) may occur, based on different seasonal degrees of training or the necessity to make weight weekly for competition. Finally, the abnormal eating behavior generally disappears when the athlete ends participation (Sudi et al., 2004).

The possible lack of a consistent underlying psychopathology and the possibility of reversal on cessation of competition do not mean that AA is not a serious problem or that it can just be ignored. Nor does it mean that an athlete cannot have AN, BED, or BN or underlying psychological issues where exercise dependence becomes an addiction, and obsessive-compulsive traits, body dysmorphic disorder, and substance dependence become apparent (Currie and Morse, 2005). Any and all of these conditions may overlap and coexist. AA is serious, and it needs to be dealt with.

Risk Factors

The specific causes of eating disorders are unknown (Arthur-Cameselle and Quatromoni, 2014; Sundgot-Borgen, 1994a, 1994b). Risk factors are multifactorial and can be divided into predisposing factors such as genetics, psychological traits, and sociocultural factors; trigger factors such as comments about weight; and perpetuating factors, such as approval by the coach when weight is lost (Bratland-Sanda and Sundgot-Borgen, 2013). Individuals who are conscientious and achievement oriented, who seek perfection, but who, at the same time, have a low self-esteem and a high need for approval seem psychologically predisposed for eating disorders. Eating disorders appear to run in families and are more strongly associated in identical than fraternal twins. By the same token, a cultural obsession with thinness may create a social predisposition, although it is not known why some young girls succumb to the pressure while most do not (Leon, 1991; Sundgot-Borgen, 1994a; Sundgot-Borgen et al., 2013). Sports and exercise for fitness are not to blame for eating disorders (Currie and Morse, 2005). However, the sports/fitness environment emphasizes performance and often demands,

in fact or perception, an ideal body size, shape, weight, or composition both as a means to achieving high performance and, in aesthetic sports, as a major part of the performance itself (Thompson and Sherman, 1993). Once a predisposed individual begins a very restrictive diet, a cycling, self-perpetuating, self-reinforcing process begins, and the individual is at great risk for developing an eating disorder.

Although no long-term prospective studies have been conducted, the following are suggested as possible risk factors for developing an eating disorder (Bratland-Sanda and Sundgot-Borgen, 2013; Sundgot-Borgen, 1994b; Sundgot-Borgen et al., 2013).

1. *Dieting at an early age.* Dieting is a risk factor, especially if recommended by a coach. There is a continuum for dieting that can progress from healthy dieting for a gradual weight loss, to a restrictive diet, chronic dieting, frequent weight fluctuations, fasting, dehydration, and the use of laxatives, diuretics, vomiting, and pills that ultimately become disordered eating behavior or an eating disorder.
2. *Unsupervised dieting.* Athletes rarely receive guidance from someone trained in nutrition and knowledgeable about their sports' requirements. Those who are given such assistance often do not follow the advice.
3. *Lack of acceptance of pubertal changes.* Young female athletes are often distressed at the bodily changes that occur as they mature—particularly menarche and the increasing levels of body fat. Many young athletes are also aware that a delayed menarche is often associated with athletic success (see Chapter 16).
4. *Early sport-specific training.* Children often specialize in a specific sport at an early age. Being a generalist as a child athlete allows more options later—although realistically, that is hard to do if one wishes to compete on an elite national or international level. An individual's somatotype (body type) greatly influences which sports she can successfully compete in. If this influence is disregarded, a young child may select a sport she enjoys (such as gymnastics) only to find that her body outgrows it.

5. *A large increase in training volume accompanied by a significant weight loss.* Athletes do not always spontaneously increase their energy intake when energy expenditure increases. The athlete sees weight loss as good and has no incentive to eat more. Thus, a pattern of eating less and less to lose more and more weight is initiated.
6. *Traumatic events.* Any traumatic event may be a risk factor, but typically, it is associated with an illness or injury that prevents training (and increases the fear of gaining weight) and/or the loss of a coach who the athlete sees as being vital to her career.
7. *Personality.* Some traits desired by coaches such as high achievement orientation potentially manifested as a desire to be leaner to improve sports performance and/or overcompliance involving excessive exercise can be confounding factors. Unfortunately, if some (weight loss or training) is good, more (weight loss or training) is not necessarily better.
8. *Coaching behavior and real or perceived pressure from coaches and judges.* The environment/team culture provided or allowed by the coach can either reduce or increase the risk of an eating disorder. In addition, comments on body composition or belief that judge's evaluations are influenced by body composition have a great impact on athletes.
9. *Weight regulations in some sports.* The goal of athletes competing in weight-class sports is often to have the lowest possible weight with the greatest possible strength. Extreme weight loss practices often occur before weigh-ins, and recovery before actual competition depends to some extent on the amount of time between the weigh-in and the competition. Frequent weight fluctuations could be a trigger for an eating disorder.

The Consequences of Eating Disorders

Energy availability (EA) is the amount of dietary energy remaining for all other metabolic processes after the energy cost of exercise training (relative to fat-free mass [FFM]) is subtracted from the daily energy intake. A value of $45 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{d}^{-1}$

generally equates to energy balance in an adult. Low EA means that all other bodily systems must adjust to a reduced energy expenditure. Disordered eating or diagnosed eating disorders often underlie low EA. Low EA has negative physiological results. The term relative energy deficiency in sport (RED-S) syndrome has been coined to emphasize the consequences of low relative energy availability. Thus, **relative energy deficiency in sport (RED-S)** is defined as a syndrome of impaired physiological function caused by low energy availability ([Mountjoy et al., 2014, 2018](#)).

Relative energy deficiency in sport (RED-S) A syndrome of impaired physiological and/or psychological function caused by low energy availability.

The consequences of RED-S and/or of an eating disorder can be dire. At the very least, the individual's nutritional status is compromised. **Figure 6.10** shows a representative sample of nutrient intake in a Norwegian study of elite female athletes ([Sundgot-Borgen, 1993a](#)). The control group (C) consisted of 30 athletes not classified as at risk for developing an eating disorder. However, 27% of those athletes were on a diet. Except for the bulimic group, all other groups had a lower total energy intake than recommended. The bulimic group's diet also differed in composition from that of the other groups. They ate more fat (30% vs. 20%) and less protein (10% vs. 20%) than did the other groups but an equal amount of CHOs. Despite the same percentage of CHOs, only the bulimic group took in sufficient amounts of CHOs in grams per kilogram per day. Those with AN had the greatest nutritional disturbance. They were simply not ingesting enough of anything.

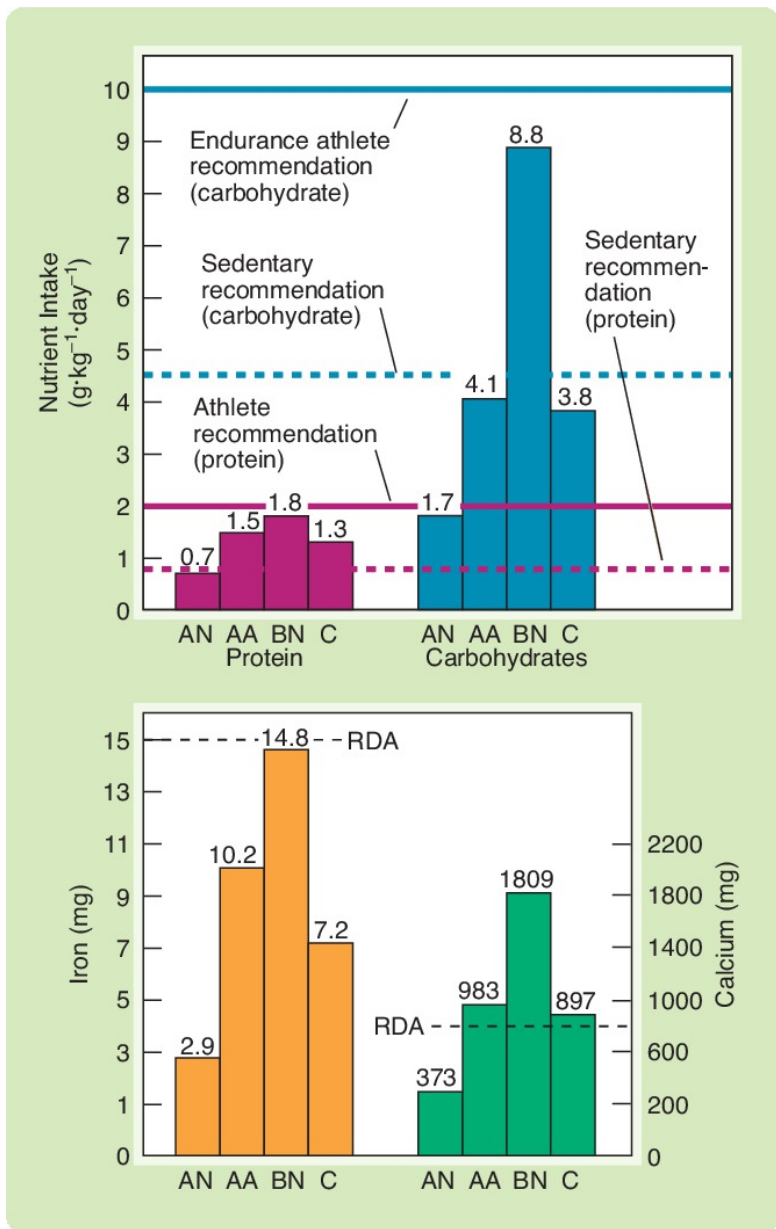


Figure 6.10 Nutrient Intake in Elite Norwegian Female Athletes.

AN, anorexia nervosa (N = 7); AA, anorexia athletica (N = 43); BN, bulimia nervosa (N = 92), and C = control (N =

30). **Source:** Based on data from [Sundgot-Borgen \(1993a\)](#).

Although the bulimic group appears to be getting sufficient nutrients in terms of CHOs, protein, calcium, and iron, note that these values are prepurging intakes. Most of the BN group and one third of the AA group regularly vomited within 15 minutes of eating. Thus, the nutrients were ingested, but they were not absorbed. Individuals who purge by using diuretics or laxatives, in contrast, have minimal caloric loss but lose electrolytes and can become dehydrated.

Figure 6.11 presents more of the consequence of RED-S although it is not exhaustive. The middle series of circles identify systems and functions that are impacted by insufficient energy availability; the outer series of circles presents some specific symptoms or results. Psychological, physiological/health, and fitness/performance characteristics can be negatively impacted. The impact of low energy availability on menstrual function and bone health (indicated by the Triad in the diagram) is detailed in “The Female Athlete Triad” section in [Chapter 16](#). At times, the medical complications of eating disorders can become severe enough to cause death ([Brownell et al., 1992](#)). At the very least, athletes with eating disorders tend to have shorter careers characterized by inconsistent performances and recurrent injuries ([Currie and Morse, 2005](#)). Though the majority of research on RED-S focuses on women, low energy availability negatively affects men as well. Male endurance athletes have been shown to have significantly lower total and free testosterone ([Wheeler et al., 1984](#); [Hooper, 2019](#)) as a result of low energy availability. Evidence even suggests that a small percentage of male endurance athletes have testosterone concentrations below 1 ng·mL⁻¹, which may lead to infertility ([Hooper, 2019](#); [Schofield et al., 2020](#)).

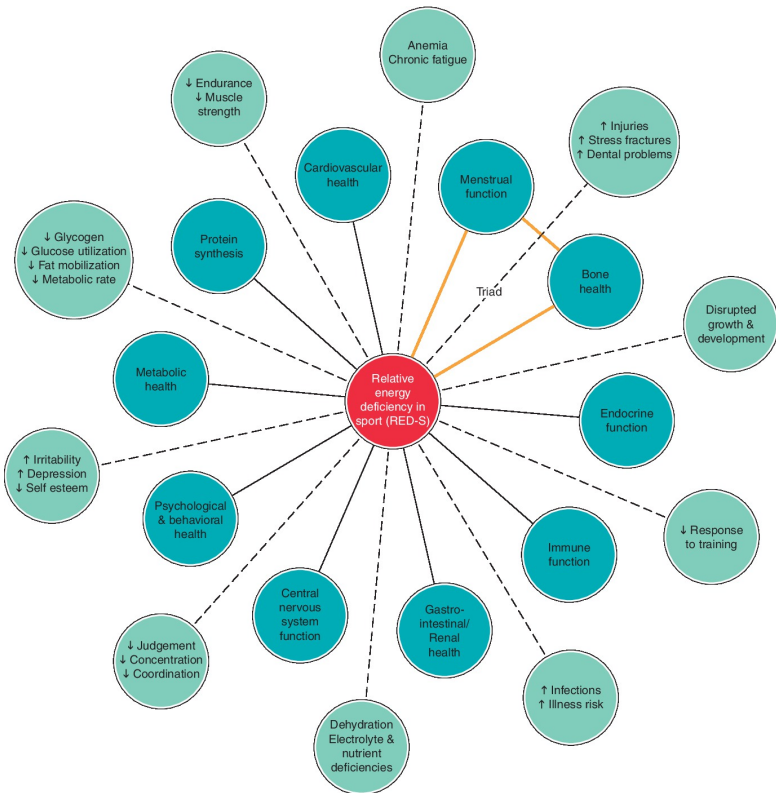


Figure 6.11 Selected Psychological and Physiological/Health and Fitness/Performance Manifestations of the Relative Energy Deficiency in Sport (RED-S) Syndrome.

The middle series of circles identify systems and functions that are impacted by insufficient energy availability; the outer series of circles presents some specific symptoms or results. Psychological, physiological/health, and fitness/performance characteristics can all be negatively impacted by low energy availability. **Source:** Modified from Mountjoy M., J. K. Sundgot-Borgen, L. M. Burke, K. E. Ackerman, C. Blauwet, N. Constantini, C. Lebrun, B. Lundy, A. K. Melin, N. L. Meyer, R. T. Sherman, A. S. Tenforde, M. Klungland Torstveit, & R. Budgett: IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *British Journal of Sports Medicine*. 52(11):687–697 (2018) and

Constantini, N. W.: Medical concerns of the dancer. Book of Abstracts, XXVII FIMS World Congress of Sports Medicine, Budapest, Hungary, 2002:151. Reprinted with permission from Prof. Naama W. Constantini, MD, DFM, FACSM, Dip. Sport Med. (CASM).

Prevention and Treatment

As a coach, physical educator, athletic trainer, or exercise leader, your role is to be part of the solution, not part of the problem. To this end, it is important to adhere to the following:

1. Become educated in all aspects of disordered eating/eating disorders and trained in how to communicate and intervene in an appropriate manner.
2. Be knowledgeable about how to optimize nutrition, body composition, and performance as well as the principles of growth, development, and maturation for both males and females.
3. Encourage youngsters to try a variety of sports. As much as possible, try to guide them into more than one sport experience where they can potentially be successful.
4. Provide access to a trained health professional or dietician who can supply realistic, healthy weight goals appropriate for each individual's stage of maturation. If weight goals are called for at all, target weight ranges that allow for growth and development should be the norm. Allow for individual differences. Male exercise leaders, teachers, or coaches, in particular, must accept that females naturally have a higher percentage of body fat than males, even when both sexes train equally ([Thompson and Sherman, 1993](#)).
5. If weight and body composition are to be monitored, do it privately to prevent embarrassment and competitive weight loss. Do not post weights. Avoid all derogatory or teasing remarks about body size, shape, or weight. Do not always equate weight loss with good. Coaches should not be directly involved in any decision regarding weight or in actually weighing athletes ([Sangenis et al., 2006](#); [Sundgot-Borgen et](#)

al., 2013; Thompson and Sherman, 1993). However, it is critical that they build into the athletic program a system for assessment of athletes by other qualified personnel, not ignore eating disorder behavior, and unconditionally support athletes who need counseling or treatment. Treatment should be delivered by health professionals (Arthur-Cameselle and Quatromoni, 2014).

6. Provide proper nutritional guidance—if need be, in conjunction with a nutritionist—and emphasize nutrition for performance, not for weight and fat control.
7. Provide a realistic, progressive training program to which students, athletes, or fitness participants can gradually adjust in terms of energy input and energy output and avoid overtraining, illnesses, and injuries.
8. Monitor the relationship between any weight loss and performance. In the early stages of an eating disorder, performance may improve, which in turn may spur the individual on to greater weight loss. However, good performance does not always equate with good health and with too much weight loss, performance will eventually decline. The most common impetus to seek help with an eating disorder is experiencing the negative consequences of such behavior, including a drop in performance (Arthur-Cameselle and Quatromoni, 2014).
9. Provide an atmosphere that accepts and supports pubertal changes so that young participants will see them in a positive light.
10. Provide an atmosphere that values the individual and his or her health and well-being above athletic performance or appearance.
11. Be aware of the signs and symptoms of eating disorders and react to them. Do not assume, however, that merely talking to or educating individuals about eating disorders will be enough to prevent or cure them. Precisely the opposite can occur (Thompson and Sherman, 1993).
12. Seek professional help when dealing with any individual you suspect of having an eating disorder. The first response to being questioned is often denial, and this is best handled by a professional trained in this area. Do not delay. The sooner

the treatment is started, the better the chances are for a full recovery (Steen, 1994; Sundgot-Borgen et al., 2013). Confrontation and/or intervention from a truly concerned individual, willing to listen and assist, is ultimately appreciated (Arthur-Cameselle and Quatromoni, 2014).

13. Never permit treatment to become secondary to an individual's participation in sports or fitness activities.

Several proactive programs aimed at preventing disordered eating have been studied and shown encouraging results. One such program for fourth to sixth graders called "Healthy Body Image: Teaching Kids to Eat and Love Their Bodies Too!" consists of 11 lessons. Compared to peers who did not take this class, participants had improved body image, self-image, lifestyle behaviors, awareness of the thinness culture message in the media, understanding of the biology of size and dieting, and awareness of body size prejudices (Kater et al., 2002). Two student-led curricula (Athletes Training and Learning to Avoid Steroids, and Athletes Targeting Healthy Exercise and Nutrition Alternatives) for high school males and females, respectively, have shown improved behaviors and intentions related to eating patterns and body-shaping drug use (Elliot et al., 2006; Gabel, 2006). All of these were short-term programs, and follow-up was needed, but they did show promise.

One study (Martinsen et al., 2014) examined the effect of a yearlong school-based intervention program that was intended to prevent the development of new cases of eating disorders in adolescent male and female elite athletes. The intervention covered six themes: motivation, self-esteem, nutrition, physiology, sport sciences, and illness prevention organized into lectures, practical and theoretical assignments, and teamwork exercises. At the end of the intervention year, for females, there were no new cases of eating disorder in the intervention high schools, while 13% of females developed an eating disorder in the control schools. Among males, there were also no new cases of eating disorder in the intervention schools but only one in a control school. Coaches also attended an intervention program emphasizing knowledge and strategies for healthy nutrition, healthy eating behavior, prevention, symptoms, identification, and management of eating disorders. Intervention coaches

exhibited higher scores on total knowledge, weight regulation, and recognition and management of eating disorders than did control coaches (Martinsen et al., 2015).

To protect the health and well-being of athletes suffering from RED-S and/or specific eating disorders, sport risk assessments and return-to-play guidelines have been developed (El Ghoch et al., 2013; Mountjoy et al., 2014, 2018; Sundgot-Borgen et al., 2013). **Table 6.9** provides the primary elements of these guidelines. Obviously, those athletes exhibiting both healthy eating habits and appropriate energy availability are fully cleared to play sports and are considered to be at low risk. Individuals who exhibit one or more of the characteristics in the middle column are considered to be at moderate risk. These individuals should be under treatment and may only train according to their treatment plan. Return to competition requires medical clearance. High-risk individuals have either diagnosed eating disorders, serious physical/psychological complications, extreme weight loss practices or meet at least three of the criterion in the moderate risk category. These individuals require supervision for training (when medically acceptable) and should not be competing.

TABLE 6.9 RED-S Risk Assessment/Play Model

| Low Risk | Moderate Risk | High Risk |
|---|---|--|
| <ul style="list-style-type: none">• Healthy eating habits• Appropriate EA* | <ul style="list-style-type: none">• Body fat <5% M; 12% F; BMI <18.5 kg m⁻² (M & F)• >5–10% weight loss in 1 month• Stunted growth or development• Abnormal hormonal profile (M); abnormal menarche/ menstrual profile (F)• Reduced BMD*; history of stress fractures• Physical/physiological complications related to low EA• Prolonged RED*• Disordered eating behavior negatively affecting other team members• Lack of progress/noncompliance in treatment• May train according to treatment plan• May compete under supervision when medically cleared | <ul style="list-style-type: none">• AN, BN, AA*• Serious physical/psychological complications related to low EA• Extreme weight loss techniques leading to life-threatening conditions• At least three criteria in moderate-risk category• Only supervised training when medically cleared• Written contract• No competition |
| <ul style="list-style-type: none">• Full sports participation | | |

*AA, anorexia athletica; AN, anorexia nervosa; BMD, bone mineral density; BN, bulimia nervosa; EA, energy availability; RED, relative energy deficiency.

Based on El Ghoch et al. (2013); Mountjoy et al. (2014, 2018); Sundgot-Borgen et al. (2013).

Summary

1. The five goals in an optimal training diet are to
 - a. Provide caloric and nutrient requirements
 - b. Incorporate nutritional practices that promote good health
 - c. Achieve and maintain optimal body composition and playing weight
 - d. Promote recovery from training sessions and for physiological adaptations
 - e. Try variations of precompetition and competition fuel and fluid intake to determine bodily responses
 - f. Implement nutritional strategies that have been shown to improve physical performance
2. A balanced nutritional diet for an adult is composed of sufficient calories to maintain acceptable body weight and composition, with 10–35% protein, 20–35% fat, and 45–65% CHO; the Dietary Reference Intake/Recommended Daily Allowance for vitamins and minerals; and sufficient fluid for hydration. This is also adequate and probably optimal for all but hard-training elite, competitive athletes.
3. Active individuals who are satisfied with their body weight and composition must ingest sufficient calories to balance those expended. This diet should proportionally increase the nutrient intake.
4. In order to maximize adaptations from training sessions, the exercise needs to rehydrate, replenish (muscle glycogen), and repair (muscle protein). This involves both CHO and protein ingestion.
5. Recommended daily CHO ingestion for training is on a sliding scale based on the duration and intensity of the training: for low-intensity, short duration exercise = $4.5\text{--}5\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$; for moderate exercise $1\text{ hr}\cdot\text{d}^{-1} = 5\text{--}7\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$; and for extreme endurance training $4\text{--}5\text{ hr}\cdot\text{d}^{-1} = 8\text{--}12\text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$.
6. Selection of the type of postexercise CHO should be based on

the time available for replenishment and GI. The immediate postexercise feedings should consist of HGI foods to facilitate glycogen replenishment, with the following meals containing both high-glycemic and mixed-glycemic menus when time is short, while the GI of the CHO consumed during the immediate postexercise period might not be as important as simply ingesting a sufficient amount of CHO if a full 24 hours are available. When CHO intake is insufficient ($<1.2 \text{ g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$), the addition of amino acids and/or protein can be beneficial.

7. Recommended daily protein ingestion for training depends on the sport and the individual. These values include $1.2\text{--}1.4 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for endurance training, $1.2\text{--}1.7$ (or even 2.0) $\text{g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for team/intermittent sports, and $1.5\text{--}1.7 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for power sports. Older individuals need $1.0\text{--}1.5 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ up from the RDA of $0.8 \text{ g}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ for younger sedentary adults.
8. Protein ingestion should begin immediately postexercise at a rate of $0.25 \text{ g}\cdot\text{kg}^{-1}$, continue every 4–5 hours, and be topped off with approximately 40 g prior to sleep.
9. A high-fat diet has no performance advantage for most individuals. However, active individuals should be ingesting 20–30% of their daily calories as fat.
10. Vitamins are important for the production of energy. However, there is no evidence that vitamin supplementation in an adequately nourished individual improves performance, speeds up recovery, or reduces injuries. Where deficiencies are present, supplementation to normal physiological levels can improve performance.
11. Supplementation of zinc, chromium, selenium, copper, iron, or any other micronutrient above normal levels does not appear to increase exercise performance. Iron supplementation given to individuals with iron deficiency anemia consistently improves performance, but supplementation should not be done unless needed clinically.
12. Supplementation of the macrominerals calcium and magnesium does not increase exercise performance. Phosphate loading may delay the onset of anaerobic metabolism effects.

13. A complete nutritional analysis by a trained nutritionist should precede any supplementation to determine need and safety.
14. The five goals of an optimal competitive diet are to
 - a. Ensure adequate fuel supplies in the pre-event time span
 - b. Ensure adequate fuel supplies during the event, regardless of its duration
 - c. Facilitate temperature regulation by prevention of dehydration
 - d. Achieve desired weight classifications while maintaining fuel and water supplies
 - e. Avoid gastrointestinal discomfort during competition
15. CHO loading is beneficial for individuals competing in endurance events of at least 60–90 minutes at 65–85% $\dot{V}O_2 \text{ max}$.
16. In the 1-week CHO-loading technique, an individual increases the percentage of CHO ingested from 50 to 70% while gradually tapering training the week before competition. The simplified CHO-loading technique requires only a short supramaximal exercise bout followed by approximately 90% CHO ingestion for 24 hours to achieve glycogen supercompensation. Both techniques are safe and effective.
17. A pre-event meal composed primarily of CHO and accompanied by fluids is recommended 1–4 hours before competition; a small amount of CHO may be ingested within 5 minutes of the event. During prolonged exercise, either moderate or HGI foods are suggested.
18. CHO beverage ingestion at 15- to 30-minute intervals during endurance activity can prevent a decline in glucose and delay fatigue. For endurance or intermittent sports lasting 1–2.5 hours, the optimal amount of either glucose or glucose plus fructose is 30–60 g·hr⁻¹. For events lasting longer than 2.5 hours, 90 g·hr⁻¹ of glucose plus fructose should be ingested. Beverages containing 6–8% of mixed CHO type are recommended for optimal benefit. All CHOs ingested during an event should have a high-glycemic index.

19. Aside from daily nutritional requirements, nutrient timing may be another important consideration to improve physical performance and exercise induced adaptations. Nutrient timing pre-, during, and postexercise should be viewed as a “garage door of opportunity,” which may positively impact performance, recovery, and athlete availability.
20. Three types of eating disorders are of great concern for personnel working with active individuals: anorexia nervosa, bulimia nervosa, and anorexia athletica. Although there are technical differences among the three disorders, all involve a restriction of food intake or a purging of food ingested in a binge, a desire for more and more weight loss, and a denial of having a problem.
21. The exact cause of eating disorders is unknown. More athletes than nonathletes suffer from eating disorders, and most cases occur in sports where low weight gives a competitive advantage, in aesthetic sports, or in sports requiring weight classifications. Far more females than males have eating disorders, but prevalence is increasing in males.
22. Personnel working with active individuals between the ages of 10 and 25 years should be aware of situations that are probably risk factors for an eating disorder, avoid practices that increase the risk, identify problems at an early state, and facilitate appropriate therapy.

Review Questions

1. List the goals for nutrition during training and the goals for nutrition during competition. Explain why they are different.
2. Make a table comparing a balanced diet for a sedentary individual and one for an active individual training for an endurance event, team or intermittent sport, or a power event. Include caloric intake, percentages, and grams per kilogram per day recommendations for the major nutrients, as well as similarities or differences in vitamin, mineral, and fluid ingestion.

3. Define the GI, and describe how foods are divided into high, moderate, and low categories. Using the GI, design an ideal snack to be eaten approximately 30 minutes after a century bike ride (100 mi), and design an ideal snack to be eaten during a day of hiking on the Appalachian Trail. Explain your choices.
4. Discuss the positive and negative aspects of a high-CHO diet.
5. Discuss the situations in which an increase in protein above the RDA is advisable and situations in which such an increase is not advisable.
6. Describe a training situation and the theory behind when fat intake can be too low in that situation.
7. Compare the theory behind the use of CHO loading for endurance athletes with the theory behind the use of CHO loading for bodybuilders.
8. Discuss the role of nutrient timing with regard to protein and CHO consumption for improved physical performance (focus on postexercise and presleep recommendations).
9. Make a table of a comprehensive fluid and nutrient intake for pre-event, during-the-event, and postevent diets for a football player and a triathlete.
10. Define and list the criteria/characteristics of anorexia nervosa, bulimia nervosa, and anorexia athletica.
11. Identify the risk factors for developing an eating disorder. Prepare a set of guidelines that could be used to counteract these risk factors or deal with the disorder early in its progression.
12. Discuss possible consequences of eating disorders.

Literature Search

1. We discussed nutritional considerations for fitness and athletics in this chapter. One topic we explored was protein needs and how they change as we age and when we might consume protein to best achieve certain goals. To explore this topic further, do a literature search using a search engine

such as PubMed, Google Scholar, or Web of Science.

- a. Search protein timing, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Protein and resistance training
 - ii. Protein and fat metabolism
 - iii. Protein and presleep feeding
 - iv. Protein and animal versus plant sources
 - v. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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7 Body Composition: Determination and Importance



CHAPTER OUTLINE

Introduction

Body Composition Assessment

Laboratory Techniques

Field Tests of Body Composition

Overweight and Obesity

What Happens to Adipose Cells in Obesity? The Cellular Basis of Obesity

Fat Distribution Patterns

Health Risks of Overweight and Obesity

Summary

OBJECTIVES

After studying the chapter, you should be able to:

- Detail the extent of the “obesity epidemic.”
- List the laboratory techniques for the assessment of body composition and both fully describe the technique of hydrostatic weighing (densitometry) and explain its theoretical basis.
- Describe the theory behind Dual X-Ray Absorptiometry (DXA) and how it is used to determine body composition.
- Calculate body density and percent body fat.
- Discuss variations in the basic assumptions of densitometry related to children, adolescents, and older adults, and describe the practical meaning of these variations.
- List and identify the strengths and weaknesses of the field estimates of body composition.
- Compare the percent body fat estimated by skinfold and bioelectrical impedance measurements with the percent body fat determined by hydrostatic weighing.
- Contrast the percent body fat and the patterns of fat distribution in an average adult male and female and the health implications of these differences.
- Differentiate between overweight and obesity.
- Describe what happens to adipose cells in obesity and the major fat distribution patterns.
- List and discuss the health risks of being overweight or obese and the impact of physical fitness on these risks in general and in relation to the obesity paradox.

Introduction

Answer the following questions to yourself. Is your body weight

just right, a little high, very high, a little low, or very low? Is your percent body fat (%BF) just right, a little high, very high, a little low, or very low? Do you like how you look in a bathing suit? What mental images did you have as you answered these questions?

If you are a female, chances are that regardless of the reality of your body weight, composition, and shape, you feel that your values are too high and you are dissatisfied with how you look. You are probably incorrect in your assessment, but you are certainly not alone (Novella et al., 2015; Quittkat et al., 2019; Schneider et al., 2013). For example, in one study (Lutter, 1994), active women were shown images of five individuals who were 20% underweight, 10% underweight, average weight and size, 10% overweight, and 20% overweight. The subjects were asked to select which image they would like to look like. Only 14% wanted the average shape, 44% wanted to be 10% underweight, and 38% wanted to be 20% underweight!

These feelings are, of course, largely a result of cultural expectations. There have been times in Western civilization (e.g., in the 17th century) when plumpness was the norm for feminine attractiveness. In more modern times, great changes have occurred in the cultural ideal of the female body. An analysis of *Playboy* centerfolds and Miss America Pageant contestants from 1960 to 1980 showed that the percentage of average weight (average weight being based on mean population values at the time) for these individuals declined steadily from approximately 90% to almost 80%. At the same time, average hip and bust sizes for these icons of beauty declined, while waist and height values increased. Thus, the ideal shape changed from the hourglass curve to a more tubular profile (Garner et al., 1980). The concern for the shrinking size of models' figures is so great that multiple countries (Italy, Spain, France, and Israel) have banned from its fashion shows any model with a body mass index (BMI) less than 18.5 kg-m^{-2} (a standard for underweight). Efforts to improve the health of models by placing limitations on BMI and bodyweight are underway in the United States, but no concrete measures have been taken.

In our current society, social media can have a large influence on individual's confidence, feelings of attractiveness, and body

satisfaction. Data show that women who were exposed to images of “attractive” peers and celebrities increased negative mood and body dissatisfaction compared to those who were exposed to travel images (e.g., images of landscapes) (Brown and Tiggemann, 2016). Another study found that uploading “selfies,” or the act of taking a picture of oneself, to social media platforms has been shown to increase anxiety, decrease confidence, and decrease feelings of attractiveness among women (Mills et al., 2018). The effects of social media on body image are not exclusively affecting female users. Just look at the images portrayed by male role models on social media as well. A recently published study demonstrated that among male social media users, those who view images of shirtless male figures have lower body satisfaction compared to those who viewed clothed male figures (Tiggemann and Anderberg, 2020). However, the authors did conclude that the negative effects of social media on body image may be more pronounced in female rather than male users. This is because male users reported no difference between clothed and bare-chested images of influencers regarding other measures of body satisfaction, such as inspiration to exercise.

What body shape did you compare yourself with mentally as you considered the opening questions above? Hopefully, you visualized standards that are consistent with good health, but many males as well as females compare themselves to images present in the media that do not represent healthy weights.

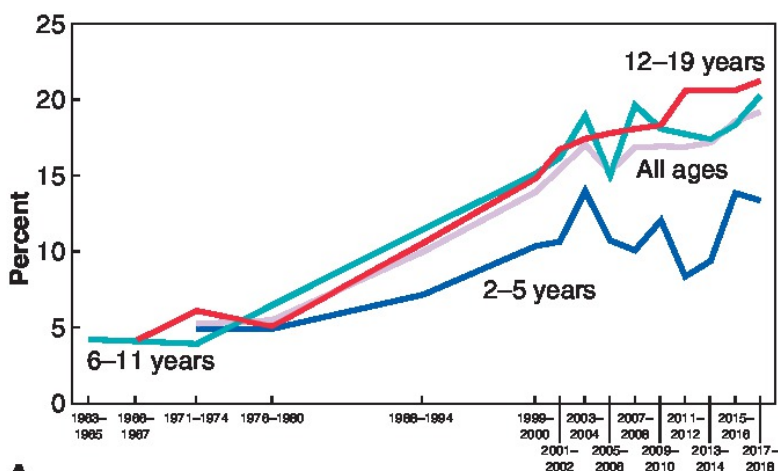
Two studies of adolescents suggest that the perception of being overweight leads to unhealthy weight control behaviors. The first study (Talamayan et al., 2006) examined data from the 2003 Youth Risk Behavior study of a total of 9,714 normal-weight U.S. high school students. A significant portion (25.3% of females and 6.7% of males) misperceived themselves as overweight and engaged (16.8% of all females and 6.8% of all males) in unhealthy weight control behaviors (use of diet pills, laxatives, and fasting) in the 30 days before the study. The second study (Neumark-Sztainer et al., 2006), a 5-year longitudinal study, showed that lower satisfaction with body weight does not serve as a motivator for engaging in healthy weight management behaviors. Rather, it predicts the use of behaviors (unhealthy dieting, disordered eating, smoking, lower levels of physical activity, and less fruit and vegetable intake) that place the

adolescents at risk for further weight gain and poorer overall health.

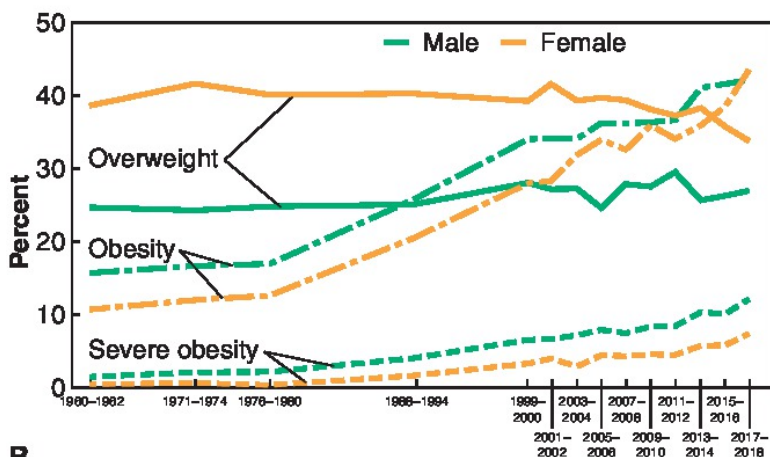
An interesting twist regarding body image is evident. In a study of 310 college students, [Neighbors and Sobal \(2007\)](#) confirmed that women are still more dissatisfied with their bodies than men even though in this sample, the prevalence of overweight was higher in males (44%) than in females (11%). Most (87%) of the “normal”-weight females wanted to weigh less, whereas considerably fewer (24%) of the “not overweight” males wanted to weigh less. Females classified as underweight expressed almost no body weight or shape dissatisfaction. The surprising result was that although both the overweight males and females wanted to lose weight, they did not select an ideal body weight that would move them into a healthier BMI category. The authors concluded that these results may suggest a shift in body size ideals to more acceptance by many individuals of larger body sizes in an era of prevalent obesity, despite the linkage of obesity to health problems.

Regardless of all of these somewhat conflicting and varied perceptions, the fact is that the prevalence of overweight and obesity is a major worldwide problem. Sixty-five percent of the world’s population lives in a country where overweight and obesity kill more people than does underweight ([World Health Organization, 2020](#)). The World Health Organization states that the number of overweight and obese individuals worldwide was 1.9 billion in 2016, of whom 50 million were obese. At all ages, prevalence was higher in developed countries than in developing countries. The overall percentages were for males 19% (children) and 42.4% (adults) and for females 18% (children) and 39.7% (adults) were overweight or obese in 2018–2019. No country has shown significant decreases in obesity in the past three decades; however, the increase in adult obesity in developed countries has slowed down ([Ng et al., 2014](#)). The prevalence of obesity is lower in Canada in both adults and children/adolescents over the age of 6 years than in the United States ([Carroll et al., 2015](#); [Ng et al., 2014](#); [Shields et al., 2011](#)). **Figure 7.1A** shows the trend in obesity among children in the United States from the early 1970s to 2017–2018. In the early 1970s, the prevalence of obesity was 5% for children 2–5 years, 4% for children 6–11 years, and 6% for adolescents 12–19 years. Data for 1963–1970 are not included

because they are not available across the age span, but what data are available showed values less than 5% for youth 6–17 years (Fryar et al., 2014a). In 2017–2018, based on measured NHANES data, 19.3% of U.S. children and adolescents 2–19 years were obese. In addition (not shown), 13.4% of infants and toddlers were obese. The rates for boys were 14.79% at 2–5 years, 21.3% at 6–11 years, and 22.5% at 12–19 years. The comparable values for girls were 12.2% (2–5 years), 19.2% (6–11 years), and 19.9% (12–19 years) (Hales et al., 2020). These are meaningful increases; however, the increase appears to be plateauing, at least for girls.



A



B

Figure 7.1 Trends in Childhood and Adolescent Obesity and Adult Overweight, Obesity, and Extreme Obesity.

Panel A shows the trends in obesity among children and adolescent aged 2–19 years by sex in the United States from 1963–1965 to 2017–2018; **Panel B** shows trends in 20- to 74-year-old adult overweight, obesity, and extreme obesity by sex from 1960–1962 through 2017–2018. **Sources:** Fryar et al. (2014a, 2014b); Hales et al. (2020).

Figure 7.1B presents the trend in overweight, obesity, and extreme obesity among adults 20–74 years from 1960–1962 through 2017–2018 (Fryar et al., 2014b). While the values for overweight for both males and females exhibit little variation over this time span, both obesity and extreme obesity percentages have slowly and then sharply increased. In the late 1970s, 15% of adults were obese. In the early 1990s, zero states had an adult obesity rate of more than 25% (Dietary Guidelines for Americans, 2010). In 2017–2018, based on measured National Health and Nutrition Examination Survey (NHANES) data, 42.8% of U.S. adults were obese. For adult males, the values were 40.3% at 20–39 years, 46.4% at 40–59 years, and 42.2% ≥ 60 years. The comparable values for adult females were 39.7% (20–39 years), 43.3% (40–59 years), and 43.3% (≥ 60 years) (Fryar et al., 2014b). In 2019, by self-reported data for adults as part of the Behavioral Risk Factor Surveillance System (BRFSS), no states had an obesity prevalence less than 20%; only Colorado and the District of Columbia had a prevalence of obesity between 20 and 25%; 13 states (Guam and Puerto Rico) had a prevalence of obesity between 25% and less than 30%; 23 states had an obesity prevalence of between 30 and 35%; and 12 states had a prevalence of obesity $\geq 35\%$. The Midwest (33.9%) and South (33.3%) had the highest prevalence followed by the Northeast (29.0%) and then the West (27.4%) (Centers for Disease Control and Prevention, 2021).

When the results of the NHANES study are broken down by race/ethnicity (Hales et al., 2020), the data indicate that there is a disparity. For males and females combined, the prevalence of obesity is lowest in non-Hispanic Asian youth 2–19 years (8.7%)

followed by non-Hispanic white (16.1%), non-Hispanic black (24.2%), and Hispanics (25.6%). For adults ≥ 20 years, the order of obesity prevalence varies slightly: non-Hispanic Asian (17.4%), non-Hispanic white (42.4%), Hispanic (44.8%), and non-Hispanic black (49.6%).

There is an insidious problem with overweight and an *obesity epidemic*. In fact, obesity has risen approximately 5% in adults and 2% in children between 2013 and 2018.

As an exercise specialist/physical educator/rehabilitation therapist/athletic trainer, you will be dealing with a public that has more questions and concerns about body composition and weight control than just about anything else we deal with. This and the next chapter are designed to present you with an understanding of these crucial areas.

Body Composition Assessment

Laboratory Techniques

The two major approaches to body composition assessment are chemical and anatomical. The chemical (molecular) approach yields the amounts of water, fat, protein, and mineral in the body (Ellis, 2000; Fosbøl and Zerahn, 2015). The anatomical (tissue/system) approach partitions the body into components that can be separated by dissection, that is, skin, muscle, adipose tissue, bone, and organs (Ackland et al., 2012; Clarys et al., 1984). Both of these approaches may be direct or indirect. **Table 7.1** provides a listing of laboratory and field techniques for body composition assessment. Note that two of the laboratory techniques have an “R” beside the name indicating that these are “Reference” techniques. Those are cadaver analysis and magnetic resonance imaging (MRI). By definition, these two methods and a compilation of several methods (typically densitometry, dual-energy x-ray absorptiometry [DXA], and hydrometry combined into a multicomponent model) are considered the most accurate techniques for measuring body composition and are the references against which other techniques should be compared. The two-component model partitions the body into fat mass and fat-free mass; the three-component model partitions the body into

fat mass, total body water, and fat-free dry solid; and the four-component model partitions the body into fat mass, total body water, bone mineral, and residual (**Figure 7.2**). The three-component model greatly increases validity over the two-component model, and the four-component model marginally improves on the three-component model ([Withers et al., 1999](#)). This does not mean, however, that the multicomponent models are the most applicable or practical, nor are they considered true criterion “gold standard” measures. Many of these techniques are expensive, cumbersome, and/or time-consuming, requiring sensitive instrumentation operated by highly trained technicians. None of the approaches to estimating body fat have been validated against cadaver dissection ([Clarys et al., 1999](#)).

TABLE 7.1 Techniques for Assessing Body Composition

| Technique | Approach | D/I | Primary Measures |
|---|--|-----|--|
| Laboratory | | | |
| 1. Cadavers (R)* | Anatomical dissection | D | Tissue masses |
| Densitometry | | | |
| A. Air displacement plethysmography (ADP) | Chemical BOD POD® body volume | I | %BF; %FFW |
| B. Hydrostatic (underwater) weighing (UWW) | Chemical | D | Whole-body density |
| | Archimedes' principle body volume | I | %BF; %FFW |
| 3. Hydrometry | Chemical Deuterium oxide dilution | D | % Water; %BF; %FFW |
| Imaging | | | |
| A. Dual-energy x-ray absorptiometry (DXA) | Chemical X-ray | D | Tissue masses |
| B. Magnetic resonance imaging (MRI) (R)* | Anatomical | D | Tissue thickness/area |
| | Nuclear | I | Tissue thickness/volume |
| C. Peripheral quantitative computed tomography (pQCT) | Anatomical X-ray | D | Tissue layer thickness |
| D. Ultrasound | Anatomical High-frequency sound waves | D | Tissue layer thickness |
| Field | | | |
| 1. Anthropometry | Anatomical Skinfolds/circumferences | I | Thickness; %BF; %FFW |
| 2. Bioelectrical impedance (BIA) | Chemical Electrical current | I | Total body water; %BF; %FFW |
| 3. Body mass index (BMI) | Anatomical | I | Ponderosity: weight relative to height |

*R, reference; D, direct; I, indirect; %BF, percent body fat; %FFW, percent fat-free weight.

Based on [Ackland et al. \(2012\)](#); [Heymsfield et al. \(2005\)](#); [Lohman et al. \(2013\)](#).

| | | | | | | |
|---|----------------------------------|-------------|-------------------|-------------|--------------------|----------------------------|
| A | Total body weight (mass) (TBW) | | | | | |
| B | Fat-free weight 55–96% of TBW | | | | | Fat 4–45% of TBW |
| C | Muscle 48% | Bone 16% | Skin 14% | Blood 9% | Organs 13% | Storage + essential fat |
| D | Water 72–74% | | Protein 19–21% | | Bone mineral 7% | Fat |

Figure 7.2 Models of Body Composition.

A represents total body weight, B is the two-compartment model, C lists the tissue components of FFW and of total fat, and D indicates the chemical components of FFW and represents a four-component model. **Source:** Based on Lohman (1986).

The most common laboratory technique for measuring body composition in a multicomponent manner is the DXA. However, the two-compartment hydrostatic method (underwater weighing) will be described first in detail here because of its historical importance and prevalence in a large body of research (Behnke and Wilmore, 1974; Ellis, 2000; Fosbøl and Zerahn, 2015; Going, 2005; Goldman and Buskirk, 1961; Lohman and Chen, 2005; Ratamess, 2010). In addition, due to the ease of use, another two-compartment method, air displacement plethysmography (BOD POD®) will be discussed. **Figure 7.3** panel A shows underwater weighing; panel B shows air displacement plethysmography in a BOD POD®; panel C shows the DXA technique. Although BOD POD® and underwater weighing are examples of two-component analysis, densitometry remains a useful technique in laboratory settings.



Figure 7.3 Densitometry.

Panel A shows the determination of body density by hydrostatic (underwater) weighing that is based on Archimedes' principle; **panel B** shows the determination of body density by air displacement in a BOD POD®; **panel C** shows the DXA technique®.

Hydrostatic (Underwater) Weighing: Densitometry

Historically, **hydrostatic**, or underwater, **weighing** (UWW) (**Figure 7.3A**) was used to determine body composition through the calculation of body density (Behnke and Wilmore, 1974; Going, 2005; Goldman and Buskirk, 1961; Ratamess, 2010). It has been documented that Archimedes, a Greek mathematician who lived in the 2nd century BC, discovered this technique. When King Hieron of Syracuse commissioned a new crown, he suspected that the jeweler substituted silver for pure gold inside the crown. The king asked Archimedes to determine the composition of the crown without harming it in any way. Legend has it that as Archimedes was pondering this question at the public baths, he solved the problem and went running through

the streets naked, shouting, “Eureka!” (“I have found it!”).

Hydrostatic Weighing The historical criterion measure for determining body composition through the calculation of body density.

What Archimedes observed was that an amount of water was displaced from the bath equal to the volume of the body entering the bath. Archimedes reasoned that the volume was proportional to the mass of the object and that the object's loss of weight in water equaled the weight of the volume of water displaced. We now define the mass of an object divided by its loss of weight in water as the *specific gravity* of that object. Archimedes also reasoned that the body (or any other object floating or submerged) is buoyed up by a counterforce equal to the weight of the water displaced. Thus, **Archimedes' principle** states that a partially or fully submerged object experiences an upward buoyant force equal to the weight or the volume of fluid displaced by the object. Based on this principle, the volume of any object, including the human body, can be measured by determining the weight lost by complete submersion underwater. In the case of the human body, dividing the mass of the body by its volume defines *body density*. When Archimedes compared the amount of water displaced by a mass of pure gold and a mass of pure silver equal to the mass of the king's crown, he found that the crown displaced more water than did the gold, but less than did the silver. He confirmed this result by weighing, in air and underwater, masses of gold and silver equal to the weight of the crown in air and found the crown to have an intermediate specific gravity value. Thus, the crown was not pure gold (only about 75%), and we can only speculate as to the fate of the jeweler ([Behnke and Wilmore, 1974](#)).

Archimedes' Principle The principle that a partially or fully submerged object will experience an upward buoyant force equal to the weight on the volume of fluid displaced by the

object.

Even if you have never been weighed underwater, you probably have a basic understanding of the principle just described. Just think about swimming. Do you float easily, or did you seem to sink? How would you describe floaters and sinkers in terms of being lean or fat? Whether one floats or sinks depends on one's body density, and body density (mass per unit volume) is largely determined by the amount of body fat present. The density of bone and muscle tissue is greater than the density of fat tissue. Thus, the leaner, more muscular individual weighs more underwater and tends to sink, whereas an individual with a large amount of fat weighs less and tends to float.

Whole-body **densitometry**, which is the measurement of mass per unit volume, is the foundation of hydrostatic weighing. It is based on dividing the total body weight (**Figure 7.2**, row A) into two compartments: fat and fat-free weight (FFW) (row B). Row C lists the component parts of the **fat-free weight** (all of the tissues of the body minus the extractable fat) as muscle, bone, skin, blood, and organs; this represents a second model. The fat compartment includes both storage and essential fats. *Storage fat* is the fat in the subcutaneous adipose tissues and the fat surrounding the various internal organs (visceral fat). *Essential fat* includes the fat in the bone marrow, central nervous system, cell membranes, heart, lungs, liver, spleen, kidneys, intestines, and muscles.

Densitometry The measurement of mass per unit volume.

Fat-Free Weight The weight of body tissue excluding extractable fat.

Note that although the terms *lean body mass* (LBM) and FFW (sometimes called the fat-free body, FFB, or fat-free mass, FFM) are used interchangeably, they are slightly different. Technically,

LBM includes the essential fat, but FFW does not. Chemically, FFW is composed of water, proteins, and bone mineral (**Figure 7.2**, row D); this is a third model of compartmentalization or composition ([Lohman, 1986](#)). Although each of these models (B, C, and D) describes the composition of the body, when the term body composition is used in exercise physiology, it generally refers to model B. That is, **body composition** is defined as the partitioning of body mass into FFM (weight or percentage) and fat mass (weight or percentage).

Body Composition The partitioning of body mass into FFM (weight or percentage) and fat mass (weight or percentage).

Compartmentalizing the body into only fat and FFW (not water, mineral, protein, and fat) and using this two-compartment model to determine percent body fat depends on the following assumptions:

1. The densities of the fat and the FFW are known and additive.
2. The densities of water, bone mineral, and protein that make up the FFW are known and are relatively constant from individual to individual.
3. The percentage of each fat-free component is relatively stable from individual to individual.
4. The individual being evaluated differs from the assumptions of the equation being used only in the amount of storage fat.

Once body density has been measured, %BF can be calculated. The two most widely used formulas for converting body density to %BF were developed by Siri and Brozek. They have been derived differently but, within the density units of 1.09 and $1.03 \text{ g}\cdot\text{cc}^{-1}$, agree within 1% on the %BF values calculated. At lower densities, the Siri formula gives increasingly higher %BF values than the Brozek formula ([Lohman, 1981](#)).

$$7.1 \quad \text{Brozek: } \%BF = \left[\left(\frac{4.570}{D_b} \right) - 4.142 \right] \times 100$$

$$7.2 \quad \text{Siri: } \%BF = \left[\left(\frac{4.950}{D_B} \right) - 4.5 \right] \times 100$$

Brozek's formula assumes that the individual has neither lost nor gained substantial amounts of body weight recently.

Once %BF has been determined, body weight at any selected fat percentage can be calculated using the following sequence of formulas. The first formula simply determines the amount of fat-free weight an individual currently has (WT₁).

$$7.3 \quad \begin{aligned} &\text{fat-free weight} = \text{current body weight} \\ &(\text{lb or kg}) \times \left(\frac{100\% - \text{percent body fat}}{100} \right) \\ &\text{or} \\ &FFW = WT_1 \times \left(\frac{100\% - BF\%}{100} \right) \end{aligned}$$

The second formula calculates the desired weight (WT₂).

$$7.4 \quad \begin{aligned} &\text{body weight at the selected percent of body} \\ &\text{fat (lb or kg)} = [100 \times FFW(\text{lb or kg})] \\ &\div (100\% - \text{selected \%BF}) \\ &\text{or} \\ &WT_2 = \frac{100 \times FFW}{100\% - \%BF} \end{aligned}$$

The third formula calculates the amount of weight to be gained or lost.

$$7.5 \quad \begin{aligned} &\text{weight to gain or lose (lb or kg)} \\ &= \text{body weight at selected \%BF} \\ &\quad - \text{current body weight} \\ &\text{or} \\ &\Delta WT = WT_2 - WT_1 \end{aligned}$$

Example

For example, an individual who currently weighs 150 lb at a body fat of 25% wishes to reduce her body fat to 17%. Equation 7.3 is used to calculate her current fat-free weight.

$$\text{FFW} = 150 \text{ lb} \times \left(\frac{100\% - 25\%}{100} \right) = 112.50 \text{ lb}$$

Her current fat-free weight and selected %BF are then substituted into Equation 7.4 to obtain her weight goal.

$$\text{WT}_2 = \frac{100 \times 112.50 \text{ lb}}{100\% - 17\%} = 135.54 \text{ lb}$$

Comparing her current weight to her goal weight in Equation 7.5, we get

$$\Delta \text{WT} = 135.5 \text{ lb} - 150 \text{ lb} = -14.5 \text{ lb}$$

This means that to be 17% BF, this individual must reduce her current weight by 14.5 lb. Of course, these calculations assume that in the process of losing weight, muscle mass is maintained. This assumption is not always true.

When the measuring technique is properly conducted, the error of %BF determined by densitometry is approximately $\pm 2.7\%$ for adults. This error range is primarily due to variations in the composition of the FFM (Lohman, 1981). The error is always lowest when the individual being tested closely matches the sample on which the equation was developed (Heyward and Stolarczyk, 1996).

CHECK YOUR COMPREHENSION 1

Phyllis Elizabeth Major is an 18 years old female who currently weights 132 lbs. Her body fat = 27.2%; she wishes to reduce that to 22%.

Using [equations 7.3, 7.4, and 7.5](#), calculate the amount of weight she needs to attain her desired %BF.

Check your answer in [Appendix C](#).

Air Displacement Plethysmography (BOD POD®)

Air displacement plethysmography is a 2-compartment model of body composition assessment, providing a measure of body fat mass and fat free mass. The most common tool used to measure body composition using air displacement plethysmography is called the BOD POD® (**Figure 7.3B**). The BOD POD® is an egg-shaped chamber that measures body volume, which is directly related to air displaced from the chamber. Using body mass and volume, BOD POD® calculates total body density as a product of body mass per unit of volume. The BOD POD® offers advantages such as comfort, ease of use, and time efficiency over traditional reference methods such as UWW.

The measure of total body volume is calculated based on *Boyle's law*, which states that the relationship between pressure and volume is inversely related. The volume of the BOD POD® chamber is first measured while empty. The test is then run with a person inside the chamber, which changes the pressure in the chamber and reduces the total volume of air in the chamber. The difference in air volume between the empty chamber and chamber with a person inside is indicative of total body volume and is used to calculate total body density. Once density is measured, then body composition is estimated with formulas as shown above in the UWW section and using identical known densities of fat and FFM. The major difference is that UWW measures water displacement whereas air displacement plethysmography measures air displacement. The validity of air displacement has been reported as $\pm 4\%$ from UWW ([Wingfield et al., 2014](#)).

Dual-Energy X-Ray Absorptiometry (DXA)

Dual-energy x-ray absorptiometry (DXA) is the most used laboratory multicomponent method for measuring body composition (**Figure 7.3C**). DXA was originally developed to diagnose bone-related issues such as osteoporosis ([Fosbøl and Zerahn, 2015](#)). It uses an x-ray beam to measure three compartments: (1) fat mass, (2) lean soft tissue (also called lean body mass), and (3) bone mineral density. Each component has a characteristic ability to absorb energy, which the DXA will measure to distinguish how much of each tissue is present in the body. The DXA technique is pictured and described in more detail in [Chapter 16](#) because it is considered the “gold standard,” or criterion measure, for the assessment of bone mineral density.

Dual-Energy X-Ray Absorptiometry The new criterion measure for determining body composition in a three-compartment model that use x-ray technology.

The DXA is often used in conjunction with methods of determining total body water and are added to specific equations to calculate four-compartment body composition values. In general, body composition measured by DXA has good correlation with four-compartment models ([Fosbøl and Zerahn, 2015](#)). The validity of DXA for determining body fat has approximately a $\pm 4.0\%$ error; however, the technique is quite reliable at less than 1.0% error ([Moon et al., 2008](#)).

The Four-Compartment Model

As mentioned earlier, multicompartment models are used to assess body composition. Of these, 4-compartment (4C) models are currently the most accurate methods for assessing body composition. 4C models categorize total bodyweight into 4 compartments (total body water, protein, bone mineral, and fat), which is more accurate than more commonly used 2- or 3-compartment models. A major reason why 4C models are more accurate is because they include fewer assumptions about the

composition of different tissues. By directly measuring some of these assumed variables (e.g., body water), 4C models account for some of the error presented by the models using fewer compartments and thus providing more accurate measurements. Unfortunately, 4C models has not been widely used because they can be expensive and time consuming. As an example, previous methods to measure body composition using a 4C model included the use of hydrostatic weighing or air plethysmography to determine total body volume, used isotope dilution methods to determine total body water, and required the use of dual x-ray absorptiometry (DXA) to determine bone density. However, Smith-Ryan et al. have developed a 4C model using only DXA to determine bone density and body volume measurements and bioimpedance spectroscopy (BIS; see below for details) to determine total body water (Smith-Ryan et al., 2017; [Wang et al., 2004](#)). These developments are expected take over as the gold standard for body composition measurements as these laboratory tools are refined.

Densitometry: Children and Adolescents and the Older Adult

The basic assumptions underlying hydrostatic weighing (densitometry) appear to not be universally applicable. In particular, research has challenged these assumptions with regard to children and adolescents ([Lohman et al., 1984](#)).

The values for the FFW or the FFB components for adults (males and females) are assumed to be approximately 73% for water and 7% for mineral content, with an overall density of FFW of $1.100 \text{ g}\cdot\text{cc}^{-1}$ (**Figure 7.4**, dashed lines) ([Boileau et al., 1984](#); [Lohman, 1986](#)). Protein, not shown on the graph, makes up about 20% of the FFW of adults.

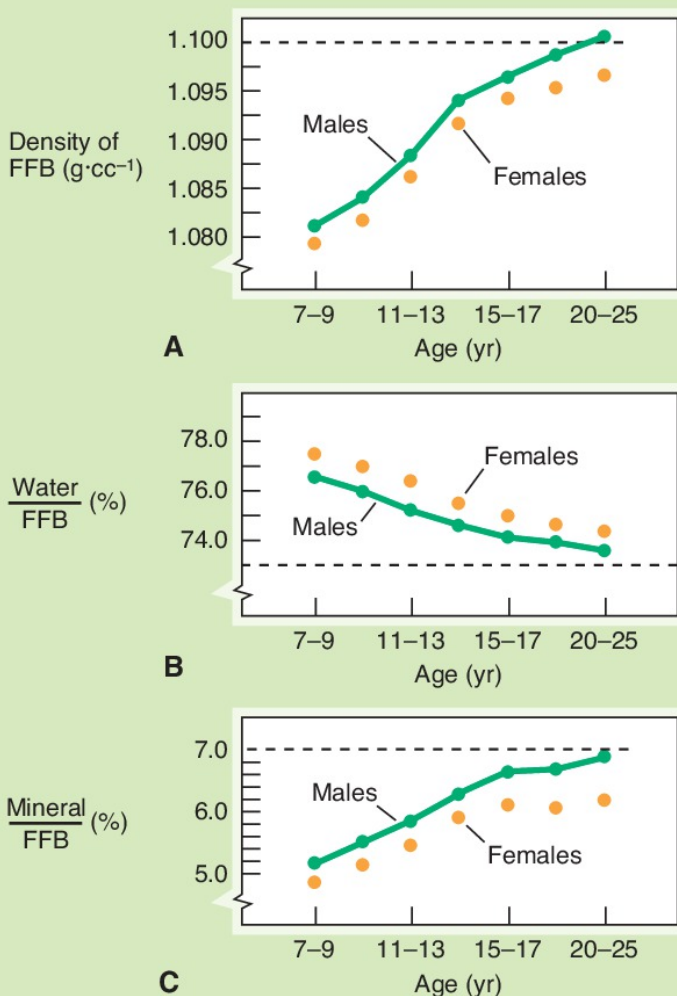


Figure 7.4 Estimated Changes in Fat-Free Body (FFB) Composition as a Function of Age.

A. The density of the fat-free body, also known as the FFW, is much lower in both male and female children than in adults. FFB density increases as the child matures, but the female density is lower at each age than the male density and never reaches the assumed adult values of 1.1. **B.** The percentage of FFB that is composed of water is higher in

both male and female children than in adults. As the child matures, the percentage of water in the FFB declines, but the female percentage is always higher than the male even after adulthood is reached. C. The percentage of FFB that is composed of minerals is lower in both male and female children than in adults. As the child matures, the percentage of minerals in the FFB increases, but the female percentage is always lower than the male percentage even after adulthood is reached. **Source:** Reprinted with permission from Lohman, T. G.: *Applicability of body composition techniques and constants for children and youth*. In Pandolf, K. B. (ed.): *Exercise and Sport Sciences Reviews*. New York, NY: Macmillan, 325–357 (1986).

Boileau et al. (1984) have shown that the percentage of water in FFW decreases (**Figure 7.4B**) from 77 to 78% at ages 7–9 years to 73% at approximately age 20 in a steady but slightly curvilinear fashion. Across the age span, females have a slightly higher percentage of water in FFW than do males. Conversely, the percentage of mineral content in FFW (**Figure 7.4C**) increases from approximately 5% at ages 7–9 to 7% at age 20. This change is also slightly curvilinear, and the values for females are consistently lower than those for males. The protein change is minimal (and therefore not presented in **Figure 7.4**), varying only about 1% (from 19% to 20%) over the age span. The result of these changes is that body density also increases in a curvilinear fashion from approximately 1.08 to 1.10 g·cc⁻¹ for adult males and from 1.08 to 1.095 g·cc⁻¹ for adult females. This means adult females never meet the 1.10·g·cc⁻¹ assumed value (**Figure 7.4A**) (Lohman, 1986).

Because the components are constantly changing as children mature, no single formula can be used for children of different ages. Nor can one formula be used for boys and for girls. **Table 7.2** shows the array of age and sex formulas needed.

TABLE 7.2 Formulas for Converting Body Density to Percent Body Fat in Children and Adolescents,

by Sex

| Age (Y) | Male | Female |
|---------|---|---|
| 7-9 | $\frac{5.38}{D_B} \times 4.97 \times 100$ | $\frac{5.43}{D_B} \times 5.03 \times 100$ |
| 9-11 | $\frac{5.30}{D_B} \times 4.89 \times 100$ | $\frac{5.35}{D_B} \times 4.95 \times 100$ |
| 11-13 | $\frac{5.23}{D_B} \times 4.81 \times 100$ | $\frac{5.25}{D_B} \times 4.84 \times 100$ |
| 13-15 | $\frac{5.07}{D_B} \times 4.64 \times 100$ | $\frac{5.12}{D_B} \times 4.69 \times 100$ |
| 15-17 | $\frac{5.03}{D_B} \times 4.59 \times 100$ | $\frac{5.07}{D_B} \times 4.64 \times 100$ |
| 17-20 | $\frac{4.98}{D_B} \times 4.53 \times 100$ | $\frac{5.05}{D_B} \times 4.62 \times 100$ |

Note: Calculated from the density FFW constant (D1) reported by [Lohman \(1986\)](#) and a density of fat constant (D2) of 0.9 g·cc⁻¹ according to the formula.

$$\%BF = \frac{1}{D_B} \left[\left(\frac{D_1 D_2}{D_1 - D_2} \right) - \left(\frac{D_2}{D_1 - D_2} \right) \right] \times 100$$

Using the equations developed with assumptions about the composition of adult components will overestimate the %BF of the child or the adolescent. This can be illustrated as follows. If the DB of a 9-year-old girl is determined to be 1.065 g·cc⁻¹ by hydrostatic weighing, the %BF calculated by the Brozek formula ([Equation 7.1](#)) is

$$\left(\frac{4.570}{1.065} \right) - 4.142 \times 100 = 14.9\%$$

The [Lohman \(1986\)](#) age- and sex-specific formula yields

$$\left(\frac{5.350}{1.065} \right) - 4.95 \times 100 = 7.3\% \quad (\text{see Table 7.1})$$

Most research published to date for children and adolescents has been on normally active nonathletes. Further work is in progress to determine the effects of physical activity and/or athletic participation on bone mineral, hydration, and body density values throughout the growth years. %BF values of young athletes, even if the appropriate age and sex formulas are used, must be interpreted cautiously. Although the pediatric formulas are better than adult formulas, even the pediatric formulas may need to be revised for young athletes. In addition, research in the area of body composition, on both sedentary and active youths, should directly measure water (W) by hydrometry and bone mineral content (M) by DXA or pQCT of the FFW as well as body density to more accurately account for all body compartments. These variables are used in a more complex formulas that account for the multiple components of body composition as shown in **Figure 7.2**.

At the other end of the age continuum, the older adult, the effect of bone mineral density loss (termed osteopenia) should be considered in the determination of %BF. Theoretically, a loss of bone mineral density would cause a decrease in body density and, thus, an overestimation of %BF if not accounted for ([Ballor et al., 1988](#); [Brodie, 1988a](#); [Lohman, 1986](#)). Indeed, the multicomponent model is preferable for all ages when equipment, time, and resources allow it.

Field Tests of Body Composition

Field methods to determine body composition can be classified as anthropometry (measurement of the human body) or bioelectrical impedance analysis (BIA). Anthropometric techniques include skinfolds, height and weight, body mass index (BMI), diameters, and circumferences. These techniques are generally practical, require a minimum of equipment, and (if properly applied) can provide useful, reasonably accurate information. They vary in the

degree of skill needed by the tester. Since field methods for assessing body composition introduce user error, the International Society for the Advancement of Kinanthropometry (ISAK) has developed specific courses and accreditations to educate health and fitness professional on the proper and standardized use of anthropometric equipment to help provide precise and accurate body composition measurements for students/clients/patients.

Skinfolds

A widely used anthropometric estimation of body size or composition involves the measurement of skinfolds at selected sites. **Skinfolds** (sometimes called fat folds) are the double thickness of the skin plus the adipose tissue between the parallel layers of the skin (**Figure 7.5**). Because skin thickness varies only slightly among individuals, skinfold measures generally indicate the thickness of the subcutaneous fat ([Behnke and Wilmore, 1974](#)). Technically, however, adipose tissue (and thus the subcutaneous fat fold) has both a fat component and a fat-free component. The fatfree component is composed of water, blood vessels, and nerves. As the fat content of the adipose tissue increases (as in obesity), the water content decreases ([Roche, 1987](#)).



Figure 7.5 Measurement of Triceps Skinfold Using a Calibrated Skinfold Caliper.

Skinfolds The double thickness of skin plus the adipose tissue between the parallel layers of skin.

The use of skinfold thickness to estimate body composition is based on two assumptions. The first is that selected skinfold sites are representative of the total subcutaneous adipose tissue mass. In general, evidence supports this assumption (Lohman, 1981; Roche, 1987). The second assumption is that the subcutaneous tissue mass has a known relationship with total body fat. **Table 7.3** shows the distribution of total body fat and the relative percentages of each storage site for a reference male and a reference female 20–24 years old.

TABLE 7.3 Percentage of Fat Distribution in a Reference Male and a Reference Female

| Distribution Site | 70-kg Male | | 56.8-kg Female | |
|-------------------------|------------|-------|----------------|-------|
| | Fat (kg) | Fat % | Fat (kg) | Fat % |
| Total body | 10.3–10.5 | 15 | 13.4–17.2 | 24–30 |
| Essential | 2.1–3.5 | 3–5 | 4.5–6.8 | 8–12 |
| Storage | 8.2–8.4 | 12 | 8.5–10.4 | 15–18 |
| Subcutaneous | 3.1 | 4 | 5.1 | 9 |
| Intermuscular | 3.3 | 5 | 3.5 | 6 |
| Intramuscular | 0.8 | 1 | 0.6 | 1 |
| Abdominothoracic cavity | 1.0 | 1 | 1.2 | 2 |

Sources: Modified from Behnke and Wilmore (1974); Going and Davis (2001); Lohman (1981).

Most of the values in **Table 7.3** are estimates. They indicate that approximately one third of the total fat for both males (3.1 kg subcutaneous fat/10.3–10.5 kg total fat) and females (5.1 kg subcutaneous fat/13.4–17.2 kg total fat) is estimated to be subcutaneous. Other estimates put this value as high as 70% and as low as 20%. With advancing age, a proportionally smaller amount of fat is stored subcutaneously. Thus, any given skinfold would then represent a smaller percentage of total body fat. It may also be that lean individuals and fat individuals store their fat in proportionally different ways. Additionally, females may store more or less fat subcutaneously than males (Lohman, 1981). There is little disagreement regarding the differences between essential fat amounts for males and females. The higher essential fat values in females are generally explained as additional storage needed to meet the energy requirements of pregnancy and lactation.

These variations in the estimation of subcutaneous fat percentage mean that the second assumption about the use of skinfolds to estimate body composition is not as firmly based as the first (Brodie, 1988a). One way of dealing with this problem is to use equations that are age-adjusted or generalized for sedentary individuals and population specific for various athletic groups (Going, 2006).

When skinfolds are taken by trained professionals and the appropriate equations are used, the variability between appropriate equations and accuracy of skinfold prediction of %BF compared with underwater weighing is approximately $\pm 3\text{--}5\%$

(Jackson and Pollock, 1978; Kaminsky and Dwyer, 2006; Lohman, 1992). Compared to 4 compartment models using UWW and DXA, skinfold measurements tend to slightly underpredict %BF by 3–5% (Nickerson et al., 2020). In the absence of complex body composition assessment tools, skinfolds may provide a fairly accurate estimate of %BF. Improper techniques can result in large prediction errors. The greatest source of error is improper location of the skinfold site. Anyone wishing to use this method to predict %BF should locate the site precisely and practice the technique repeatedly before using it. **Table 7.4** presents equations that can be used to predict %BF from two or three skinfold sites. These equations are specific for sex and age. Also included is an equation developed using a multicomponent reference model for athletes (Evans et al., 2005).

TABLE 7.4 Calculation of %BF from Skinfolds

| Group | Equation |
|--|--|
| Adult males | $\%BF = 0.39287(X_1) - 0.00105(X_1)^2 + 0.15772(X_2) - 5.18845$, where X_1 = sum of abdominal, suprailiac, and triceps skinfolds and X_2 = age |
| Adult females | $\%BF = 0.41563(X_1) - 0.00112(X_1)^2 + 0.03661(X_2) + 4.03653$, where X_1 = sum of abdominal, suprailiac, and triceps skinfolds and X_2 = age |
| Male children and adolescents (8–18 y) | $\%BF = 0.735(X_1) + 1.0$, where X_1 = sum of triceps and calf skinfolds |
| Female children and adolescents (8–18 y) | $\%BF = 0.610(X_1) + 5.1$, where X_1 = sum of triceps and calf skinfolds |
| Collegiate athletes | $\%BF = 8.997 + 0.24658(X_1) - 6.343(X_2) - 1.998(X_3)$, where X_1 = sum of abdominal, thigh and triceps skinfolds; X_2 = sex (M = 1; F = 0); X_3 = race (black = 1; white = 0) |

Note: The last three equations were developed utilizing a multicomponent reference model incorporating body density, percentage of water in FFW, and bone mineral content variations by age.

Sources: Based on information in Evans et al. (2005); Golding et al. (1989); Jackson and Pollock (1985); Slaughter et al. (1988).

Skinfold thickness values may be used in ways other than to predict %BF. First, the millimeter values of several sites (usually 5–7 from anatomically diverse locations) can be added to form a *sum of skinfolds*. Such a sum indicates the relative degree of fatness among individuals. It can also be used to detect changes within a given individual if measurements are taken repeatedly over time. Second, skinfolds may be used to determine the pattern of distribution of subcutaneous fat. Such a pattern has

emerged as an important predictor of the health hazards of obesity (Harrison et al., 1988; Roche, 1987; Van Itallie, 1988). Fat distribution patterns and the associated health problems are discussed later in this chapter. Skinfold measurements are minimally affected by the ingestion of a meal or daily changes in hydration status. Due to the simplicity, ease of use, and time efficiency of skinfold measuring, they may be the most practical tool for measures changes over time in %BF in the field (Kasper et al., 2021).

Body Mass Index

In some situations, such as when large numbers of individuals are being evaluated, the only measures of body size that can easily be obtained are height and weight. In and of themselves, these measures have little value for an adult. However, if height and weight are then used to calculate **body mass index (BMI)**, the ratio of the total body weight to height, this is an indication, albeit imperfect, of adiposity. Several ratios have been proposed, but the one used most frequently is weight (in kilograms) divided by height (in meters) squared [$WT \div HT^2$ ($kg \cdot m^{-2}$)]. This ratio is also known as the Quetelet Index (Brodie, 1988a; Reviscki and Israel, 1986; Satwanti et al., 1980). Calculated BMI can then be compared against standard values to determine whether the individual has acceptable body weight, is overweight, or is obese (Table 7.5).

TABLE 7.5 Commonly Used Body Mass Index ($kg \cdot m^{-2}$) Standards for Acceptable Body Weight, Overweight, and Obesity in Nonathletes

| Age | Acceptable Body Weight | | Overweight | | Obesity | |
|-------|------------------------|--------|--------------------|--------|---|--------|
| | Male | Female | Male | Female | Male | Female |
| <18 y | 5–84th percentile | | 85–94th percentile | | ≥95th percentile or BMI ≥ 30 99th percentile = severe obesity or BMI = 30–32 (10–12 y) BMI ≥ 34 (14–16 y) | |
| ≥18 y | 18.5–24.9 BMI | | 25.0–29.9 BMI | | Class I = 30–34.9 BMI Class II = 35–39.9 BMI Class III = ≥40 BMI | |

Sources: Based on American College of Sports Medicine (2022); Barlow and Expert Committee (2007); U.S. Department of Health and Human Services (2000); Weir & Jan (2021); World

Body Mass Index (BMI) The ratio of the total body weight to height.

Example

If a female subject (age = 30 years) weighs 165 lb and is 5 ft 8 in. tall, calculation of her BMI first requires conversion to metric units. Dividing 165 lb by 2.2 lb·kg⁻¹ gives a weight of 75 kg. Multiplying 68 in. by 2.54 cm in.⁻¹ gives 172.7 cm (1.73 m). Height value squared is 2.98 m². Thus, BMI = 75 kg ÷ 2.98 m² = 25.2 kg·m⁻². Check **Table 7.5** to see whether this is an acceptable, overweight, or obesity value for this individual.

The selection of BMI values at the upper limit of acceptable is based on the data relating BMI to morbidity (disease occurrence, particularly cardiovascular disease [CVD]), and mortality (death). For adults, there is a J-shaped curvilinear increase in excess mortality (a greater number of deaths than expected in a given population) with an increasing BMI as initially determined in the now classic study by [Bray \(1985\)](#). He found that BMI values from 15 to 25 kg·m⁻² represented no excess mortality risk. Significant increases in risk were shown to begin at a BMI of 27.3 kg·m⁻² for females and 27.8 kg·m⁻² for males. Therefore, in 1985, the National Institute of Health defined obesity as a BMI of 27.3 kg·m⁻² for females and 27.8 kg·m⁻² for males.

In 1988, the World Health Organization (WHO) adopted BMI standards of less than 18.5 for underweight, 18.5–24.9 for normal weight, 25–29.9 for overweight, and ≥30 for obesity. Obesity was further divided into three classes. Class I obesity is indicated by a BMI of 30–34.9, class II by a BMI of 35–39.9, and class III by a BMI of ≥ 40. All values are in kg·m⁻². In 1998, the National

Health, Lung, and Blood Institute and the Centers for Disease Control and Prevention (CDC) in the United States concurred with these standards and they remain current in 2021.

Using these WHO cut points, [Meyer et al. \(2002\)](#) also found a J-shaped curvilinear association between BMI and total mortality (**Figure 7.6**) even when stratified based on smoking habits and physical activity, although their data were limited to males. So few deaths occurred in those with a BMI of less than 18.5 kg·m⁻² over the length of the study that its influence on the curve has been omitted in **Figure 7.6**.

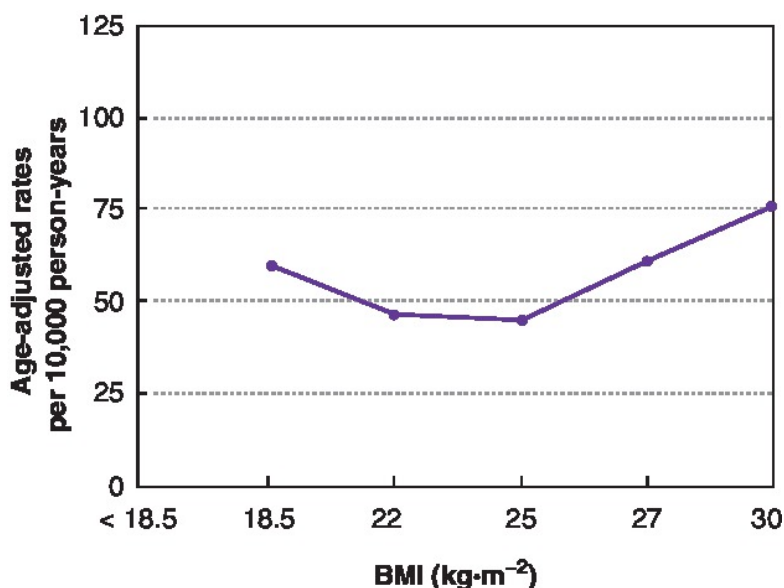


Figure 7.6 The Relationship between BMI and Mortality.

Age-adjusted rates of death from all causes based on BMI in 22,304 males without recognized cardiovascular diseases, diabetes mellitus, or cancer at the initial screening followed for an average of 16.3 years. The increasing values for BMI in kg·m⁻² are described as a J-shaped curve. **Source:**

Source: Reprinted with permission from Meyer, H. E., A. J. Søgarrd, A. Tverdal, & R. M. Selmer: Body mass index and mortality: The influence of physical activity and smoking. *Medicine & Science in Sports & Exercise*. 34(7):1065–1070

BMI has been shown to correlate highly with %BF derived from skinfold measures ($r = 0.74$) and hydrostatic weighing ($r = 0.58$ – 0.85). However, when compared with cadaver adipose tissue weight, the relationship to BMI is lower, that is, $r = 0.55$ ($r = 0.63$ for M and 0.53 for F) between the two variables accounting for only approximately 30% of the variance (**Figure 7.7**) (Clarys et al., 1999). Thus, these correlations indicate a moderate relationship, but they also suggest that there is considerable measurement error in using BMI as an estimate of body adiposity ($>5\%$). In situations where the same group of individuals was evaluated, skinfolds generally predicted %BF better than BMI did. Individuals with a normal BMI can actually have high body fat levels, and individuals with a high BMI (especially those in the 25 – $27 \text{ kg}\cdot\text{m}^{-2}$ range) may actually have normal, acceptable body fat levels. Individuals who are obviously active and/or muscular should have %BF more directly measured (Gallagher et al., 1996). The Focus on Research box reports a study showing the difficulty with using BMI to classify collegiate athletes and nonathletes as overweight. At any given BMI, females have a higher %BF than do males. Older individuals have a higher %BF at any given BMI than do younger individuals, and the association with clinical outcomes and mortality is weaker (Chang et al., 2012; Gallagher et al., 1996; Sardinha and Teixeira, 2005). The degree of adiposity associated with a given level of BMI varies by racial group/ethnicity as well. At the same BMI, black males and females tend to have higher lean body mass and lower fat mass than do white males and females. Conversely, Asian populations may have higher %BF at a given BMI (although this is still under debate) (Carpenter et al., 2013; Flegal et al., 2010), and health risks may begin at a lower BMI among Asians (Ogden et al., 2014). For this reason, a cut point of $\geq 23.0 \text{ kg}\cdot\text{m}^{-2}$ for overweight and $\geq 25.0 \text{ kg}\cdot\text{m}^{-2}$ for obesity are suggested for Asian individuals (ACSM, 2022). Finally, BMI gives no indication of fat distribution. The importance of fat distribution is discussed later in this chapter. A new description for those with low BMI but high %BF is **normal weight obese** or “skinny fat”

(Ding et al., 2016; Oliveros et al., 2014). There is evidence that the prevalence of normal weight obese individuals is higher among college males compared to females, which may increase the risk of adverse cardiometabolic health (Anderson et al., 2020). Nevertheless, BMI is often (and acceptably) used as indication of overweight or obesity and risk stratification across the age span in the absence of more specific methods to estimate total body fat, particularly when mass screening is done (Sardinha and Teixeira, 2005).

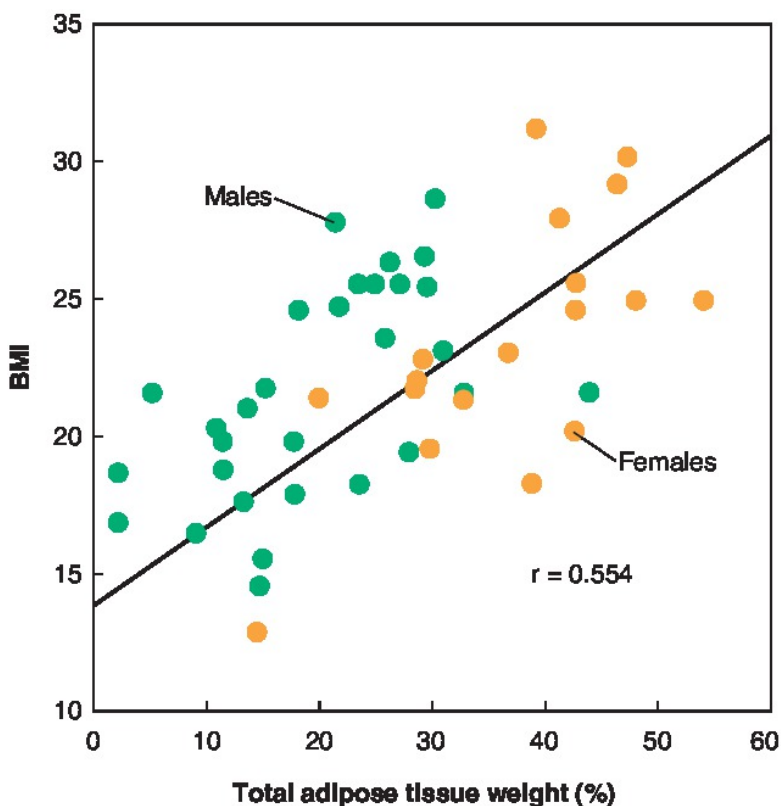


Figure 7.7 The Relationship between BMI and Total Adipose Tissue Weight Determined by Cadaver Analysis.

The relationship between directly measured adipose tissue weight from cadaver dissection expressed in percentage and body mass index (BMI). The correlation was higher for males (0.63) than females (0.53). The 0.55 value in the

figure is the combined M + F correlation. These results indicate that BMI was a limited predictor of adiposity.

Source: From Clarys, J. P., A. D. Martin, M. J. Marfell-Jones, V. Janssens, D. Caboor, & D. T. Drinkwater: Human body composition: A review of adult dissection data. *American Journal of Human Biology*. 11(2):167–174 (1999). Copyright © 1999 Wiley-Liss, Inc. Reprinted by permission of John Wiley & Sons, Inc.

Fat Free Mass Index (FFMI)

The fat free mass index (FFMI) is a measure of fat free mass (FFM) per unit of volume. Similar to BMI, the FFMI can be calculated by measuring FFM in kg and dividing it by height in meters squared. Of course, this index is different than BMI because it is specific for FFM and not total bodyweight. Therefore, the FFMI is a useful tool for comparing lean mass between individuals without the confounding factor of height. FFMI may be used alongside BMI to determine an individual's risk for developing disease. Nonetheless, calculating FFMI is a bit more complex than BMI, since equipment capable of measuring FFM is necessary.

While age does not drastically change FFMI standards ([Schutz et al., 2002](#)), there is a difference between men and women. Standards for men and women ages 18–34 years have been developed. The average FFMI for men and women is 18.9 and 15.5 kg·m⁻², respectively. A study of nearly 6,000 men and women between the ages of 15 and 98 years reported that that men categorized as “normal weight” based on BMI standards have a FFMI of 16.7 to 19.8 kg·m⁻². The value for women was 14.6 to 16.8 kg·m⁻² ([Kyle et al., 2003](#)). This is because men tend to have more FFM than women even at similar heights. Standard values for FFMI may be used in a clinical setting to help diagnose certain diseases. For example, someone with below average FFMI but normal BMI may have sarcopenic obesity. Sarcopenia is the age-associated loss of muscle mass. When sarcopenia is accompanied by increased adiposity, it is termed sarcopenic obesity. Sarcopenic obese individuals may have a low BMI due to the loss of lean mass but also be categorized as obese due to an

increase in fat mass. Likewise, in some sport settings, FFMI is being used to identify to coaches about how much muscle a player may be able to add to meet the standards for a particular sport or position. For example, in division 1 college football players, a FFMI of 24.5 to 27 $\text{kg}\cdot\text{m}^{-2}$ was reported in offensive and defensive linemen (Trexler et al., 2017). Linemen are obese by BMI standards (mean BMI of about 36) but have a %BF of only 21.5% (Provencher et al., 2018). These data indicate that these athletes carry an enormous amount of FFM. Furthermore, Lineman, both offensive and defensive, had higher FFMI when compared to other positions. Therefore, standardized values for different positions can be developed to assess player potential relative to the normative values.

Normal Weight Obese The term used for an individual with a normal or low BMI but a high %BF.

FOCUS ON RESEARCH

BMI as a Predictor of Percent Fat in College Athletes and Nonathletes

The purposes of this study were to describe the relationship between %BF and BMI in college athletes and nonathletes and to determine the accuracy of the standard of $\geq 25 \text{ kg}\cdot\text{m}^{-2}$ BMI as defining excessive body fatness in these populations. Data from varsity athletes (149 M and 77 F) and 213 undergraduate students majoring in kinesiology and enrolled in an exercise physiology lab class at a major Midwest university were used in the analyses. The male athletes were involved in basketball, wrestling, ice hockey, and football. The female athletes competed in basketball, crew, and softball.

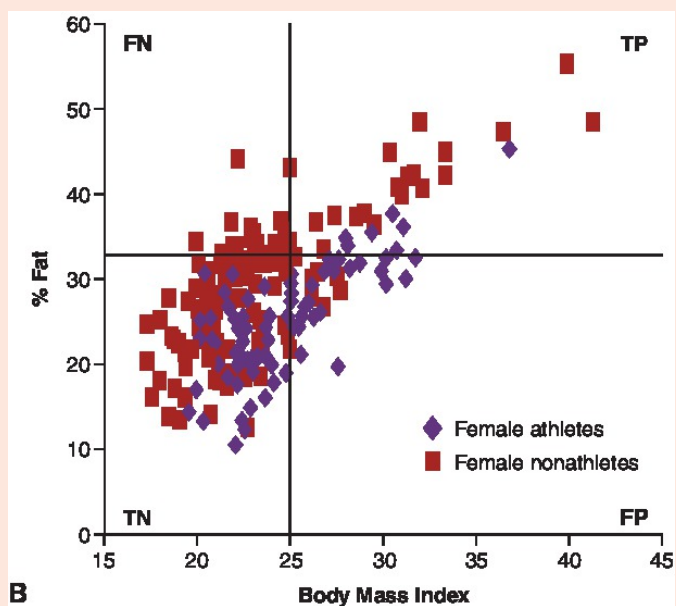
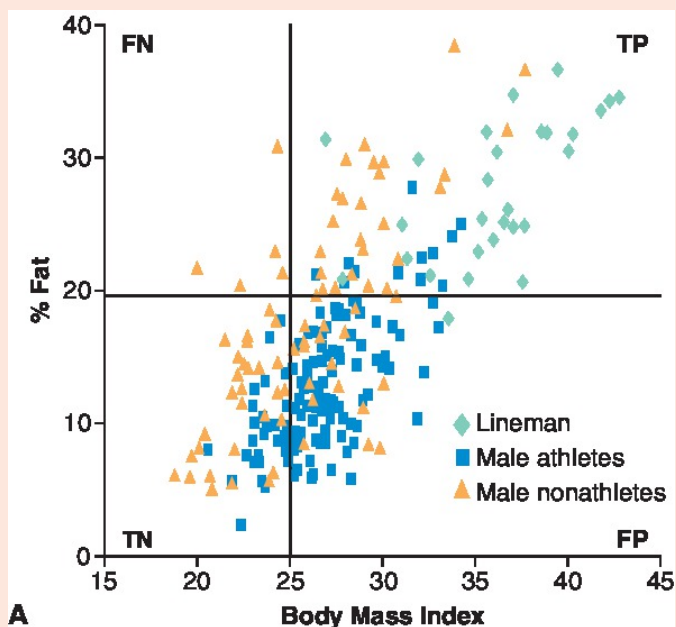
Overweight was defined as $\text{BMI} \geq 25 \text{ kg}\cdot\text{m}^{-2}$ but less than $30 \text{ kg}\cdot\text{m}^{-2}$; obesity was defined as $\geq 30 \text{ kg}\cdot\text{m}^{-2}$.

Overfat was defined as $\geq 20\%$ BF for males and $\geq 33\%$ BF for females. No football lineman had a BMI less than $25 \text{ kg}\cdot\text{m}^{-2}$, and only one had a %BF less than 20%. Therefore, the linemen were separated out from the rest of the male athletes. The results are presented in the figure accompanying: A for males and B for females. FP means false positive—individuals who were classified as overweight but had normal %BF; FN means false negative—individuals who were classified as normal weight but were overfat; TP means true positive—individuals who were classified as overweight and who were overfat; TN means true negative—individuals who were both normal weight and normal %BF. The concern is for those individuals who are incorrectly classified, that is, the FPs and the FNs.

The scatterplot in Figure A shows that 67% of the male athletes and 25% of the male nonathletes fell within the FP quadrant. The scatterplot in Figure B shows that 31% of the female athletes and 7% of the female nonathletes fell within the FP quadrant. There were no FN in either the male or the female athletes, but 44% of the female nonathletes and 6% of male nonathletes fell within the FN quadrant. These results indicate that BMI misclassifies normal fat individuals a large percentage of the time. Conversely, a large percentage of overfat female nonathletes were classified as normal weight by BMI.

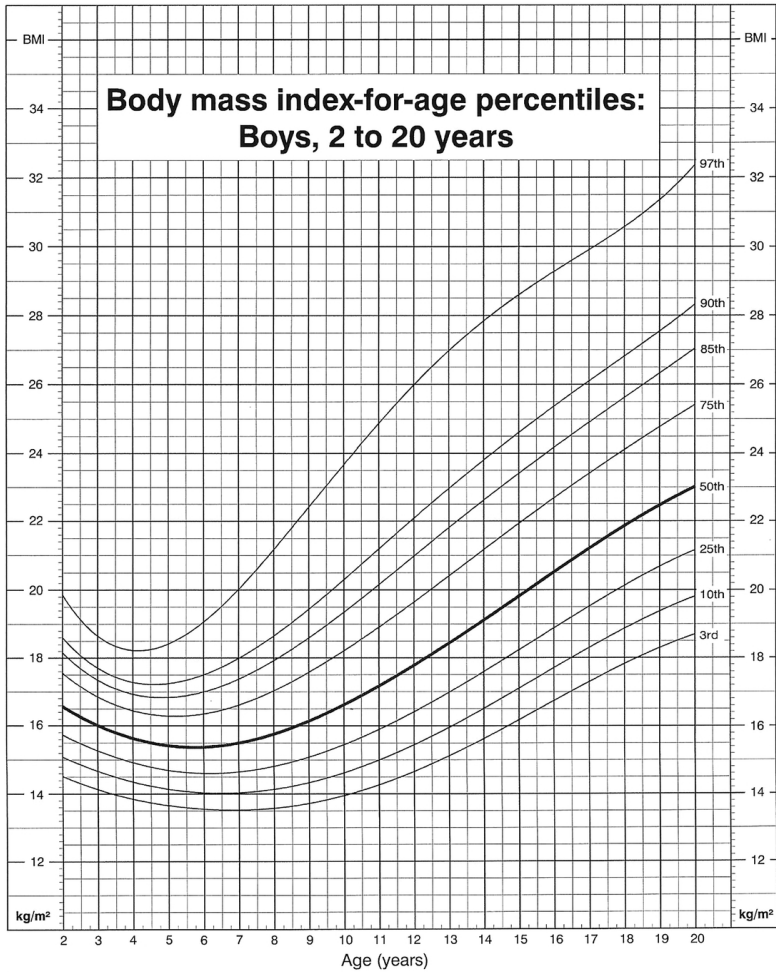
Statistically determined optimal BMI cut points to minimize the false classifications and maximize the specificity (identification of normal-weight individuals as such) and sensitivity (identification of overfat individuals as overweight by BMI) were determined to be $27.9 \text{ kg}\cdot\text{m}^{-2}$ for male athletes, $34.1 \text{ kg}\cdot\text{m}^{-2}$ for football linemen, $26.5 \text{ kg}\cdot\text{m}^{-2}$ for male nonathletes, $27.7 \text{ kg}\cdot\text{m}^{-2}$ for female athletes, and $24.0 \text{ kg}\cdot\text{m}^{-2}$ for female nonathletes. Interestingly, these optimal cut points are consistent with the 1985 NIH recommendations of $\geq 27.3 \text{ kg}\cdot\text{m}^{-2}$ for females and $\geq 27.8 \text{ kg}\cdot\text{m}^{-2}$ for males to define overweight (the 85th percentile of BMI distribution in young adults 20–29 years). Optimal cut points for the nonathletes are lower than those for the athletes at least in part because of less muscle mass in the nonathletes. These results illustrate the limitations in the BMI

classification when using one standard for all adults.



Source: Reprinted with permission from Ode, J. A., J. M. Pivarnik, M. J. Reeves, & J. L. Knous: Body mass index as a predictor of percent fat in college athletes and nonathletes. *Medicine & Science in Sports & Exercise*. 39(3):403–409 (2007). Copyright ©2007 The American College of Sports Medicine.

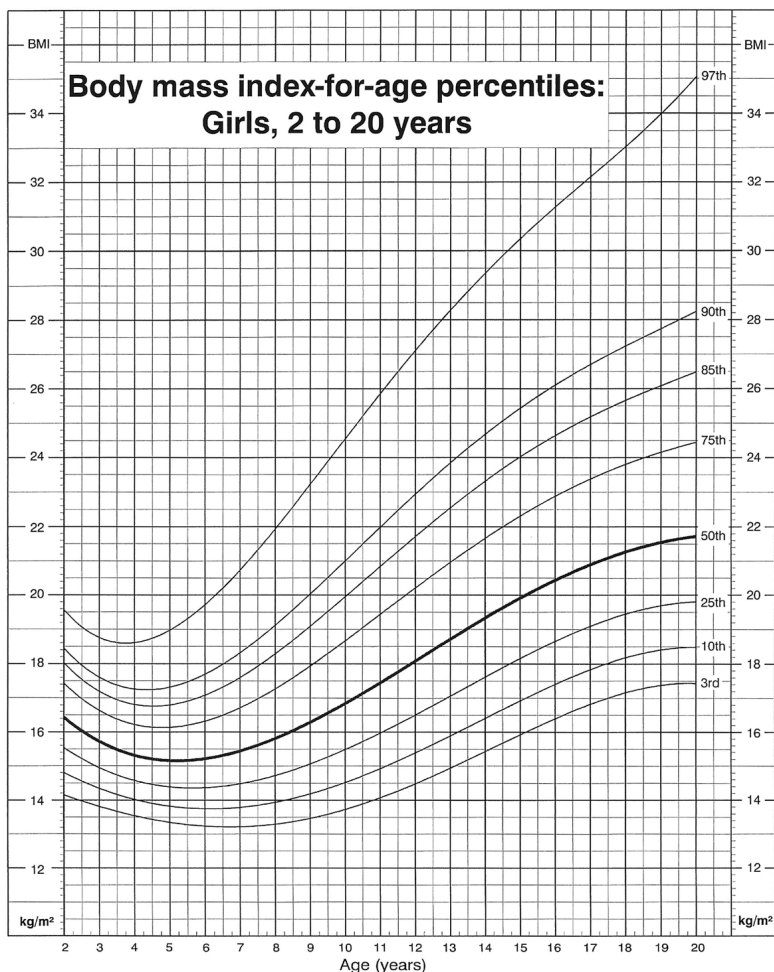
Table 7.5 presents commonly used BMI standards for acceptable body weight, overweight, and obesity in nonathletic individuals. Note that for adults, the values are those presented in the paragraph above. However, for children and adolescents, specific values are not given; instead, age- and sex-specific percentiles are used. These require the use of charts such as the CDC BMI Growth charts presented in **Figure 7.8 A (boys) and B (girls)**.



A

Figure 7.8. CDC BMI Growth Charts.

A. BMI percentiles by age for boys 2–20 years.



B

B. BMI percentiles by age for girls 2–20 years.

Waist-to-Hip Ratio/Waist Circumference

Waist-to-hip (W/H) ratio is another way to estimate health risk based on the individual's pattern of fat distribution. Research has shown that the W/H ratio is a stronger predictor for diabetes, coronary artery disease, and overall death risk than body weight, BMI, or %BF in adults (Brownell et al., 1987; Folsom et al., 1993). In the absence of large total fat stores, the W/H can aid in the identification of certain hormonal and metabolic

abnormalities associated with a relative increase in abdominal size but not total fat mass. However, for any given W/H ratio, there can be considerable variability in total body fat. Additionally, increases in body fat including intraabdominal fat may not be detected if a corresponding increase occurs in hip circumference ([Sardinha and Teixeira, 2005](#)).

Waist circumference is measured with a tape measure, to the nearest centimeter, at the level of the natural indentation or at the navel if no indentation is apparent. Hip circumference is measured at the largest site. Both measures should be taken while the individual is standing and without clothes. The average value for females aged 17–39 years is 0.80, and this value increases with age to above 0.90. Comparable averages for males range from 0.90 to 0.98. Values ≥ 0.84 for females and ≥ 0.99 for males 20–60 years and ≥ 0.90 and ≥ 1.03 for females and males, respectively, 60–69 years indicate a high risk for metabolic disease ([Ratamess, 2010](#); [Stamford, 1991](#)). Additionally, waist-to-hip ratio is a significant determining factor of all-cause mortality among highly functioning older adults (70–79 years old) ([Srikanthan et al., 2009](#)).

In 1998, the National Heart, Lung, and Blood Institute's expert panel on obesity concluded that waist circumference (also known as waist girth) alone was more strongly related to visceral abdominal adipose tissue and more predictive of disease risk than the W/H ratio in adults. Similarly, W/H ratio is not a good index of intraabdominal fat deposition in children and adolescents ([Sardinha and Teixeira, 2005](#); [Semiz et al., 2007](#)).

A high waist circumference is associated with an increased risk for Type 2 diabetes, a poor blood lipid profile, high blood pressure, breast cancer, endometrial cancer, and CVD. It is a stronger predictor of diabetes than is BMI; it can identify those who are at greater cardiometabolic risk better than can BMI; it is consistently related to the risk of developing coronary heart disease; and it is strongly associated with all-cause mortality and selected cause-specific mortality rates ([Aune et al., 2015](#); [Carmienke et al., 2013](#); [Klein et al., 2007](#); [van Dijk et al., 2012](#); [White et al., 2015](#)). A 2019 study showed that men and women who were at “high risk” based on waist circumference has significantly lower cardiorespiratory fitness compared to those

who had “normal” waist circumferences (Dyrstad et al., 2019). The values of greater than 102 cm (40 in.) for males and 88 cm (35 in.) for females are used as cutoff points for increased disease risk (ACSM, 2022; National Heart, Lung, and Blood Institute, 1998; U.S. Department of Health and Human Services, 2000; World Health Organization, 2008).

Bioelectrical Impedance (Impedance Plethysmography and Spectroscopy)

Determining body composition by Bioelectrical Impedance (BIA) has gained a great deal of acceptance in fitness facilities and schools primarily because the procedure can easily be done. In many BIA analyzers, four electrodes are attached to a quietly resting supine subject's hands and feet (two per limb either ipsilaterally or contralaterally) (**Figure 7.9A**). Some BIA equipment simply requires the individual to either stand on an electronic digital platform scale with built-in footpad electrodes or to hold the analyzer in both hands (**Figure 7.9B**). In all types of BIA equipment, a harmless, sensationless, low-amperage (80 mA), radio frequency (50 kHz) electrical current is passed between the electrodes, and the resistance (in ohms) to the current is recorded. Body volume is assumed to be a cylinder of constant cross-sectional area with uniform density distribution. Body volume is more often defined as height squared divided by resistance ($HT^2 \div R$) (Baumgartner et al., 1990; Brodie, 1988b; Van Loan, 1990).

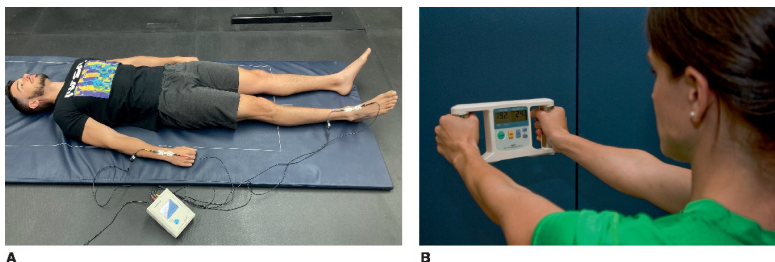


Figure 7.9 A, B. Determination of body composition by bioelectrical impedance.

The ability to conduct the electrical current is directly related to the amount of water and electrolytes in the various body tissues. Electrical current flows more easily in fat-free tissue (which offers less resistance) than in fat tissue (which acts as an *impedance*) because the fat-free tissue has a higher percentage of water and electrolytes. Therefore, individuals with more fat-free weight (FFW) have lower resistance values, and those with more fat weight have higher resistance values.

Because the BIA technique actually measures total body water (TBW), estimates can be made of body density and FFW on the basis of the obtained TBW and the assumed known percentages of water in the body and in the FFW. Another technique is to derive regression equations from height, resistance, and/or other anthropometric variables (such as weight, age, sex, skinfolds, or circumferences) to directly predict FFW or %BF. Hydrostatic weighing was traditionally used as the criterion measure but now DXA is most commonly used. As with skinfolds, a number of equations have been generated in this manner.

As you might suspect, the accuracy of BIA measurement depends highly on a normal level of hydration. Either too much fluid (hyperhydration) or too little fluid (hypohydration or dehydration) will affect the readings. The exact effect is controversial. Theoretically, an increase in body water should decrease resistance and %BF. Some experimental evidence supports this theory, but other studies have shown the opposite result. That is, dehydration caused by exercise has decreased resistance, and hyperhydration from ingestion of replacement supplements has increased resistance. This variance may be related to whether the electrolyte content changes proportionally to water and to the shift in water between compartments. What is clear is that individuals should be measured 3–4 hours after the ingestion of a meal or after an exercise session and should not be under the influence of substances such as caffeine that might act as a diuretic ([Baumgartner et al., 1990](#); [Deurenberg et al., 1988](#); [Stump et al., 1988](#); [Van Loan, 1990](#)).

The accuracy of BIA also depends on both ambient and skin temperatures. Resistance is higher in cool temperatures than warm ones. Thus, if an individual is tested repeatedly over a period of 15–20 minutes in an environment where body cooling

occurs, the %BF estimate will go up as the skin temperature goes down. Care must therefore be taken that testing is done at a neutral temperature (27–29°C; 80–84°F) when the subject is neither overheated nor chilled (Caton et al., 1988; Stump et al., 1988).

The accuracy of BIA estimates of body composition is also influenced by the equation used. These equations include those for body geometry, cross-sectional area, and current distribution, as well as those used to convert resistance to TBW, body density, and %BF, and are generally closely guarded secrets by the manufacturers. Each of the electrical assumptions is violated by the human body, but the discrepancies are apparently not enough to rule out their use in BIA. However, these violations do limit the accuracy for estimating TBW, FFW, and %BF. If the criterion measure upon which the equation was based did not take into account the age-related differences in TBW, these inaccuracies would be confounded (Baumgartner et al., 1990; Brodie, 1988a; Chumlea and Sun, 2005; Hodgdon and Fitzgerald, 1987). One recent study found that when compared to DXA, FM was underestimated and FFM overestimated by BIA, more so in males than females, and in normal weight than in overweight and obese individuals (Berstad et al., 2012).

BIA values under standard conditions will be consistent, but the accuracy is probably no better than that achieved by skinfolds (Baumgartner et al., 1990). The error of the estimate has been reported to range from approximately 3–6% BF (Ratamess, 2010). Other bioimpedance techniques, such as bioimpedance spectroscopy (BIS) have been developed. Unlike BIA methods, BIS measures intra- and extra-cellular water compartments without the necessity of population-based equations to calculate the quantity of fluid in different compartments (Earthman et al., 2007).

In summary, %BF and fat-free mass can be estimated adequately for screening and tracking moderate changes in body composition through field measures.

Complete the case studies in the Check Your Comprehension box to demonstrate your understanding of body composition evaluations.

CHECK YOUR COMPREHENSION 2—CASE STUDIES 1–5

Given the following information, classify individuals 1-5 as “underweight,” “normal weight,” “overweight,” “obese (I, II, III),” and/or as exhibiting abdominal obesity or not. Document your calculations and defend your classifications.

1. Enrico: sex = M; age = 27 y; height = 5 ft. 11 in.; weight = 198 lb; waist circumference = 44 in.
2. Zoe: sex = F; age = 20 y; height = 5 ft. 4 in.; weight = 105 lb; waist circumference = 25 in.; hip circumference = 30 in.
3. Abdullah: sex = M; age = 12 y; height = 4 ft. 7 in.; weight = 120 lb.
4. Rudie: sex = F; age = 66 y; height = 5 ft. 6 in.; weight = 187 lb; waist circumference = 36 in.; hip circumference = 39 in.
5. Yourself

For individual 6, calculate BMI and FFMI. Using all of your information, is his BMI accurate?

6. Joseph: sex = M; age = 27; height = 6 ft. 5 in.; weight = 220 lbs.; body fat = 13%.

Check your answers in [Appendix C](#).

Overweight and Obesity

Although being overweight is what bothers most people, it is really the amount and location of fat (%BF, abdominal fat mass) that should be of concern. Excess weight can be caused by high levels of lean muscle mass, but additional muscle mass is beneficial. Except in rare instances, such as providing protection from the cold water for an English Channel swimmer or certain wasting diseases, excess fat is generally not beneficial.

There are no universally agreed upon acceptable %BF standards. The most typically used normal values for young adults (20–29 years) are 12–15% for males and 22–25% for females with an allowance of an additional 2% for each decade of age. Using these ranges, obesity is then defined as +5% BF above the normal value (Kaminsky and Dwyer, 2006).

Figure 7.10 shows a wider range of standards based on age, sex, and athletic participation (Lohman et al., 1997). Note that some increase in %BF with age has also been built in for individuals ≥ 35 years. This was based on studies showing that low or reduced body fat (especially in middle-aged females) is associated with lower bone mineral content. Thus, to maximize the goal of overall health, a balance must be reached between protecting against heart disease (associated with excessive fatness) and osteoporosis and bone fractures (associated with excessive leanness) (Kaminsky and Dwyer, 2006). Note also that there is no category representing overweight in this figure (because this is based on body fat not weight), but there is a break between each recommended range and the start of the corresponding obesity range. Individuals whose %BF falls in this area (as well as the obese range) should reduce %BF.

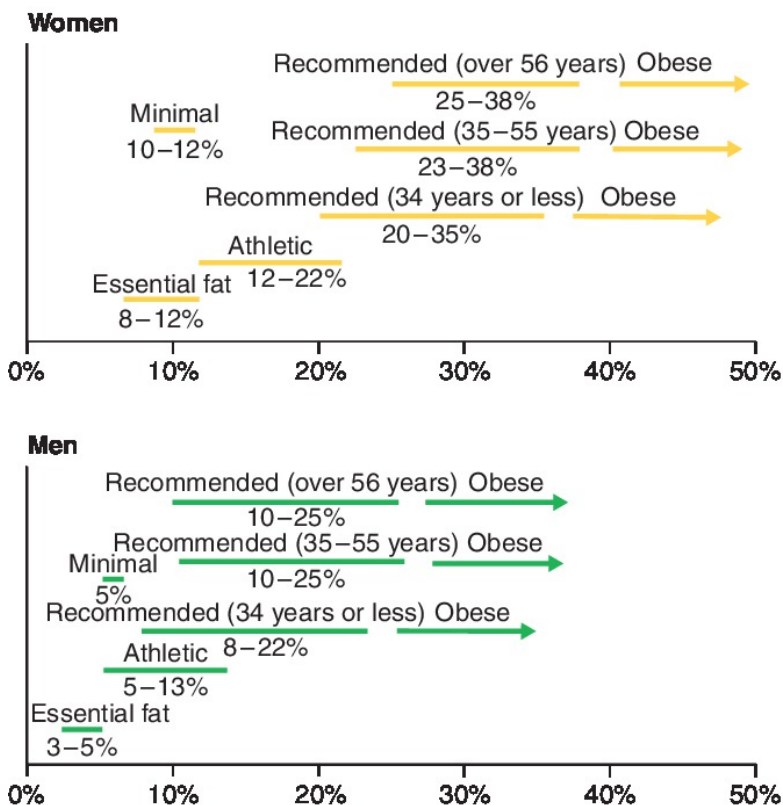


Figure 7.10 Percent Body Fat (%BF) Standards for Adult Males and Females.

Ranges of acceptable %BF and %BF values representing obesity are presented for adult women and men by age and athletic status. Note that the athletic standards begin at the upper limit of essential fat: 5% for men and 12% for women. This coincides with the acceptable minimal value for men. Women have an acceptable minimal range that encompasses part of the essential fat range. **Source:**

Reprinted with permission from American College of Sports Medicine: *ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription* (7th ed.). Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 304 (2014).

What Happens to Adipose Cells in Obesity?

The Cellular Basis of Obesity

Human adipose cells can either be white, brown, or beige. White adipose tissue is composed of a matrix of connective tissue in which white adipose cells (adipocytes) appear singularly or in small clusters. A typical cell (**Figure 7.11**) looks something like a signet ring, a metal band with a stone or jewel at the top. The nucleus of the cell appears as the stone or jewel of the ring in the cell membrane, which forms the band of the ring. The triglyceride droplets are stored in the space within the confines of the cell that provides nutrients mainly for other cells. Brown and beige adipose cells (**Figure 7.11**) also store triglycerides but in many small fat droplets. Brown and beige adipose cells contain abundant mitochondria with brown cells having more and larger mitochondria than beige cells. These abundant mitochondria use the lipid fuels as well as glucose to warm the body rather than produced ATP. Brown and beige adipose cells per se are not involved in overweight or obesity as most of the fat stored in the human body is stored in white adipose cells. Brown and beige adipose cells are discussed more fully in [Chapter 8](#).

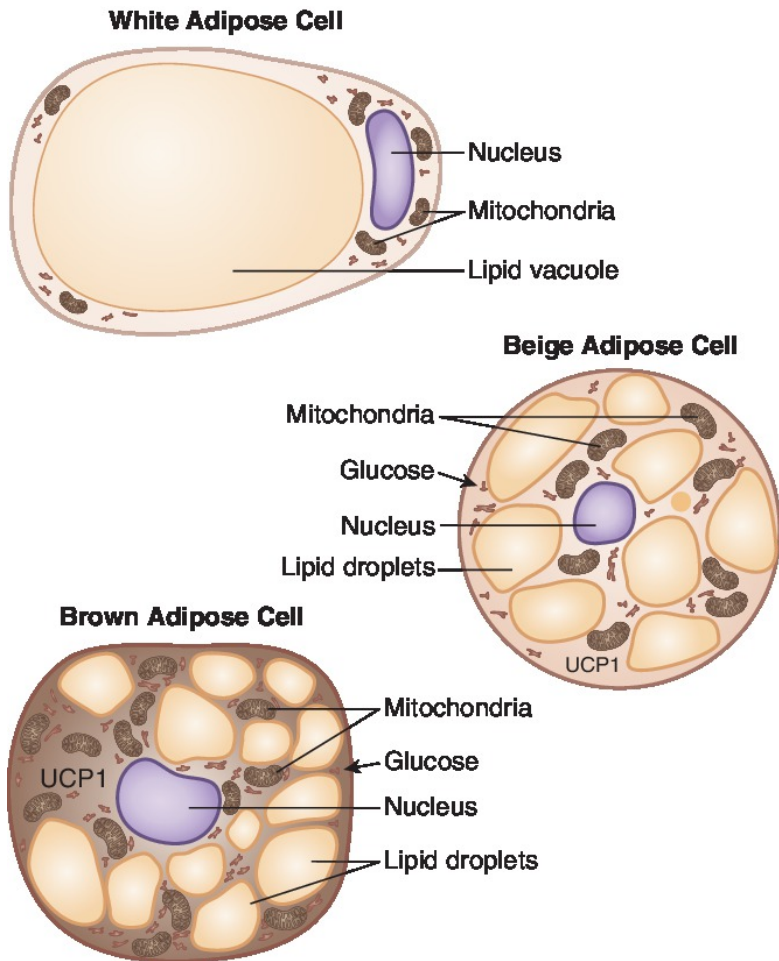


Figure 7.11 Adipose Cells.

White, brown, and beige adipose cells all store lipid as triglyceride, but do so in slightly different ways. *White* adipose cells contain one large fat droplet; *brown* adipose cells contain many small fat droplets; *beige* adipose cells contain fewer adipose droplets than *brown*, but they are somewhat larger. *Brown* adipose cells have the largest number of mitochondria, followed by *beige*; *white* adipose cells have the fewest mitochondria. **Source:** Adapted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth, Maturation and Physical Activity* (2nd ed.).

Champaign, IL: Human Kinetics (2004); Lee et al. (2013); and Wu et al. (2013).

An adult of acceptable weight has about 30–50 billion white fat cells. Mature adipocytes are among the largest cells in the body. They become plumper or more wrinkled as they take up or release fat. Females have approximately 50% more fat cells than do males. Adipocytes can become up to 10 times larger if needed to store triglycerides. Apparently, this increase in size (hypertrophy) is how increasing levels of fat are first stored. Sometimes, when the fat cell size is enlarged, the increased size causes a bulging between the fibrous tissue strands, causing a dimply, waffled appearance. These lumpy areas are often referred to as *cellulite*. Cellulite, however, is simply fat (Björntorp, 1987, 1989).

Once the upper limit of fat storage by hypertrophy is approached (somewhere around 30 kg of fat), fat cell hyperplasia occurs. **Hyperplasia** in general is growth in a tissue or organ through an increase in the number of cells. Fat tissue hyperplasia involves the development of new adipocytes from immature precursor cells. Adipocytes themselves do not divide and multiply, but hypertrophy in adipocytes stimulates cell division and maturation in precursor cells (Malina et al., 2004). Thus, a newly overweight adult is likely to have the same number of fat cells as when he or she was of normal weight, but these adipocytes will be larger than before. An obese individual may have enlarged adipocytes, an increased number of adipocytes, or both. Obese individuals may have as many as 75–80 billion fat cells. Once created, fat cell numbers are not naturally reduced, even if body weight and body fat are lost (Sjöström and Björntorp, 1974). Liposuction—the surgical removal of adipose tissue—is the only way to get rid of adipocytes. The maintenance of large numbers of adipocytes may be one reason why it is so difficult for obese individuals to maintain a weight/fat loss once it has been achieved.

Hyperplasia Growth in a tissue or organ through an increase in the number of cells.

These facts emphasize the importance of preventing the maturation of extra fat cells. Overweight or obese infants, children, or adolescents tend to become overweight or obese adults, although adolescent obesity is more predictive of adult obesity than is obesity at birth or in infancy (Charney et al., 1975; Dietz, 1987; Lohman, 1989; Simmonds, 2015).

Figure 7.12 indicates the typical pattern of changes in adipose cell size (**Figure 7.12A**) and number (**Figure 7.12B**) that occurs in normal (nonobese) children and adolescents (Malina et al., 2004). From birth to young adulthood, the average adipose cell size doubles or even triples. Most of this increase in size happens during the first year after birth. From 1 year to the onset of puberty, there is no significant increase in size and no sex difference. At puberty, adipose cell size increases in females but remains fairly constant in males. Not all adipose cells are of the same size; internal (visceral) fat cells are generally smaller than subcutaneous fat cells. Furthermore, not all subcutaneous cells are equal in size. For example, gluteal adipocytes tend to be larger than abdominal adipocytes, which in turn are larger than subscapular cells.

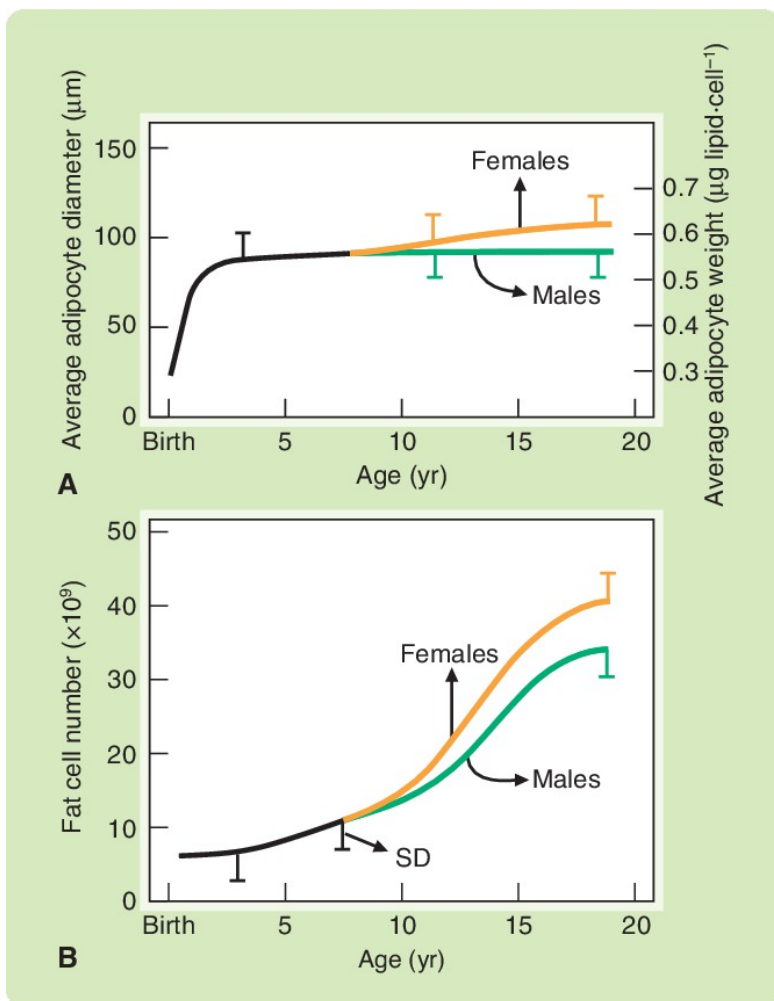


Figure 7.12 Adipose Cell Size and Number Changes with Growth.

A. The greatest change in adipose cell size occurs between birth and 1 year. Until approximately age 10, cell size is similar in males and females. After age 10, the difference between males and females gradually widens, and individual variations (indicated by the *vertical lines*) become more apparent. **B.** The number of adipose cells gradually increases through the childhood years in males and females at a similar rate. At approximately age 10, in both sexes, the

rate of increase in fat cell number accelerates, but females far exceed males. Individual differences indicated by vertical bars (labeled SD for standard deviation) increase considerably with age. **Source:** Reprinted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth, Maturation and Physical Activity*(2nd ed.). Champaign, IL: Data from Bonnet and Rocour-Brumioul (1981); Boulton et al. (1978); Chumlea et al. (1981); Hager (1981); Hager et al. (1977); Knittle et al. (1979); Sjöström (1980); Soriguer Escofet et al. (1996).

At birth, the number of adipocytes is approximately 5 billion. For the number to increase to the average adult value of 30 billion, considerable change must occur. However, little increase in number occurs during the first year after birth when the cell size is changing so drastically (**Figure 7.12B**). From 1 year to the onset of puberty, there is a gradual but steady increase in the number of adipocytes, with no difference appearing between the sexes. This gradual increase can double or even triple the number of fat cells. At puberty, the number of adipose cells increases greatly in both males and females, but the female increase far exceeds the male increase. This increase in fat cell number plateaus in late adolescence/early adulthood and ideally remains at that level. In reality, however, hyperplasia can and often does occur in adulthood (Malina et al., 2004). Thus, although there are two critical periods—infancy and adolescence—in the development of adipocytes, this should not be interpreted as meaning that fat cells cannot be added during adulthood.

Some individuals produce more adipocytes than do others during growth, and this can amount to billions of cells. During the growth changes, males tend to accumulate more subcutaneous fat on the trunk and females on the extremities.

Fat Distribution Patterns

The location of fat storage varies among individuals. In general, fat is distributed in three basic patterns: android, gynoid, and intermediate (**Figure 7.13**). The *android pattern* (**Figure 7.13A**),

also known as the abdominal or apple pattern, is predominately found in males. It is characterized by the storage of fat in the nape of the neck, shoulders, and abdomen (upper part of the body). In this pattern, the largest quantity of fat is stored internally, not subcutaneously, and the trunk area has the greatest amount of subcutaneous fat. The result is the classic potbelly shape. Individuals with potbellies often make the claim that they are not fat and occasionally challenge others to hit them in the stomach as hard as possible to prove their superior musculature. In fact, the hardness of the abdominal region is caused by the excess fat in the abdominal cavity pushing against the abdominal muscles and stretching them taut, not by muscle tone or hypertrophy. The amount of intra-abdominal (visceral) fat is twice as high in android obesity as gynoid obesity (Blouin et al., 2008; Campaigne, 1990; Stamford, 1991).



Figure 7.13 Patterns of Fat Distribution.

A. Android, B. gynoid, and C. intermediate.

The *gynoid pattern* (**Figure 7.13B**), also called the gluteofemoral or pear pattern, is found predominantly in females. It is characterized by the storage of fat in the lower part of the

body, specifically, in the thighs and buttocks, with the largest quantity being stored subcutaneously. No pseudohardness is apparent. These sites tend to be soft and tend to jiggle (Campaigne, 1990; Stamford, 1991).

It is thought that the deposition of fat in the gluteal-femoral region in females is linked to the reproductive function. In particular, gluteal-femoral fat may furnish energy for the development of the fetus, primarily during the latter stages of pregnancy, and for the newborn child during lactation. As would be expected, these fat deposits are controlled by the steroid hormones.

The third type of fat pattern is known simply as the *intermediate pattern* (Figure 7.13C). In this pattern, fat is stored in both the upper and the lower parts of the body, giving a somewhat rectangular cubic appearance. Note that all three patterns are found in both males and females, despite the sex-specific predominance associated with android and gynoid shapes (Campaigne, 1990; Stamford, 1991). With menopause, female fat distribution typically changes from a gynoid to an android pattern (Després and Lamarche, 2000; Rosano et al., 2007).

Abdominal adipose tissue (in subcutaneous and visceral depots) is the primary site for immediate storage of diet-derived fat. During weight gain, these adipose cells undergo hypertrophy, and because their number does not expand, they have a finite storage capacity. The life span of abdominal adipocytes is approximately 10 years with a 10% turnover each year (Karpe and Pinnick, 2015). Abdominal fat deposits are easily mobilized; therefore, it is possible to reduce fat accumulation in this area relatively easily (assuming a caloric deficit). Conversely, gluteal-femoral fat deposits respond to weight gain by hyperplasia, and their contents are not easily mobilized. Thus, it is not possible to reduce fat accumulation in these areas easily. The potential for reshaping the gluteofemoral fat pattern is extremely limited (Campaigne, 1990; Stamford, 1991). In one study that measured areas of fat loss from caloric restriction in overweight young females, those with an android distribution of fat lost more weight and showed a greater decrease in waist circumference and a greater intra-abdominal fat loss than did those with a gynoid fat distribution (Jones and Edwards, 1999).

The reason behind the variation in the fat deposit mobilization is hormonally based (Wong et al., 2003). Two different receptors, alpha and beta, have been identified in fat cells; they vary in their ability to facilitate or inhibit fat incorporation into the cell or fat mobilization out of the cell. Alpha-receptors inhibit fat transfer to and from the adipocytes, and beta-receptors enhance these transfers. Enzyme activity is concomitantly increased or decreased. Alpha-receptors predominate in the lower body and are thus more abundant in the gynoid pattern. Beta-receptors are concentrated in the upper body and are more abundant in the android pattern. Thus, the adipose cells in the abdominal region are more unstable.

Epinephrine (released from the adrenal medulla) binds to beta-receptors on adipose tissue and causes lipids (fatty acids) to be mobilized from the abdominal cells and released into the circulatory system. If the free fatty acids and glycerol can be used (directly or indirectly) as fuel to support exercise, there is no problem. However, when epinephrine is released in times of emotional stress and there is no need for the excess fuel, the fatty acids and glycerol are then routed to the liver, where they are detrimentally deposited or converted to very low-density lipoproteins and small dense LDL-Cs increasing the risk of coronary heart disease (Després and Lamarche, 2000).

In addition, abdominal fat cells tend to be larger than the fat cells found in other parts of the body. Larger abdominal (android) fat cells are associated with glucose intolerance (the inability to dispose of a glucose load effectively), coupled with insulin resistance, hyperglycemia, and an excess of insulin in the blood (hyperinsulinemia). These conditions are associated with diabetes mellitus (a coronary heart disease [CAD] risk factor) and hypertension (a CAD risk factor). The latter occurs because of the action of insulin in promoting reabsorption of sodium by the kidneys (Brownell et al., 1987; Campaigne, 1990; Stamford, 1991). Conversely, the smaller thigh-hip (gynoid) fat cells are highly insulin sensitive. After a fatty meal when circulating triglycerides are broken down by these cells, the resulting fatty acids tend to be stored immediately. This keeps them out of circulation and avoids the conversion to low-density lipoprotein cholesterol (LDL-C) (McCarty, 2003). Finally, visceral adipose tissue releases over 120 hormones and factors called adipokines

or adipocytokines. Most of these factors are involved in the pathological development of inflammation, atherosclerosis, hypertension, endothelial dysfunction, and insulin resistance. These are important components in the development of the metabolic syndrome (discussed later) (Hutley and Prins, 2005; Lyon et al., 2003; Wong et al., 2003). Between the two major fat distribution types, these factors result in the gynoid pattern being associated with a lower risk and the android pattern with a higher risk of myocardial infarctions, stroke, and metabolic syndrome components (Kang et al., 2011; Toss et al., 2011; Wiklund et al., 2010). Data suggest that the profound functional differences between gynoid and android fat are controlled by site-specific developmental genes (Karpe and Pinnick, 2015). **Table 7.6** summarizes the differences between android and gynoid fat patterns.

TABLE 7.6 Patterns of Fat Distribution

| Factor | Android | Gynoid |
|-------------------------------|--|------------------------------------|
| Sex it predominates in | Males | Females |
| Regional fat storage | Upper body (neck and abdomen) | Lower body (thighs and buttocks) |
| Fat storage site | Internal | Subcutaneous |
| Characteristic of fat deposit | Hard | Soft |
| Adipose tissue receptors | Beta | Alpha |
| Mobilizing hormone | Epinephrine | Reproductive, especially prolactin |
| Adipose cell size | Large | Small |
| Fat mobilization | Easy | Difficult |
| Major risk | Coronary artery disease, glucose intolerance, diabetes, and hypertension | Psychological |

Simply looking in the mirror while naked is probably the best way to determine whether you are android, gynoid, or intermediate in terms of fat distribution, although the calculation of the waist circumference and W/H ratio might also be useful. High ratios reflect a larger waist and thus more upper body fat deposition (android shape) than lower body hip fat deposition.

Figure 7.14 presents an image of visceral abdominal tissue. This particular image was produced by a CT scan. MRI is also frequently used in laboratory studies. These imaging techniques directly measure fat tissue. As previously mentioned, measuring waist circumference is the best field technique for estimating abdominal obesity.

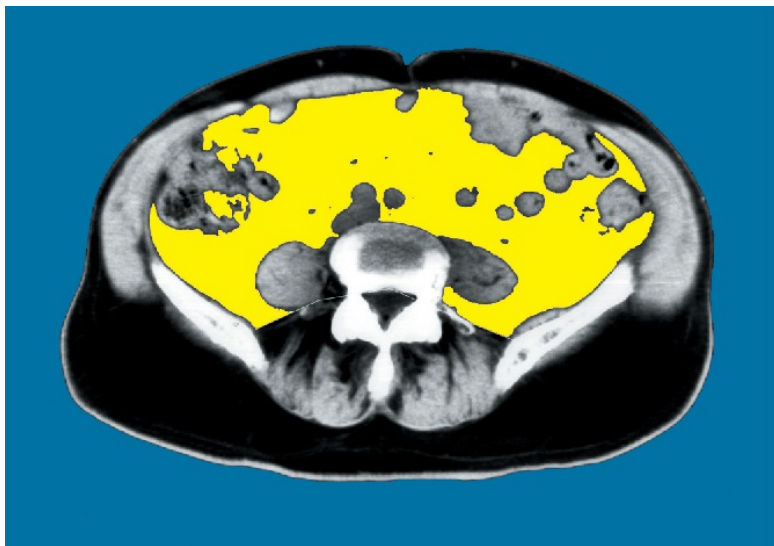


Figure 7.14 Visceral Abdominal Tissue.

CT scan at the L4–L5 intravertebral space. The abdominal visceral fat depot is in *yellow*; the subcutaneous fat depot (both outside the muscle wall and below the skin) is in *black*.

Figure 7.15 presents the results of National Health and Nutrition Examination Surveys (NHANES) conducted on adults aged 20–79 years indicating the mean waist circumference of age-adjusted abdominal obesity. Abdominal obesity was defined as greater than 102 cm for males and greater than 88 cm for females. Both males and females show a steady increase in the average waist circumference from 1960–1962 to 1999–2000 and then more moderate increases. The male average has remained below the cutoff for abdominal obesity, but the female averages from 1988–1994 through 2015–2016 are above the cutoff. In 2014, among adults 20 years and older, the prevalence of abdominal obesity has increased to 57.2% of the total population with a mean annual increase of 0.62% since 1999–2000 ([Caspard et al., 2018](#)). The prevalence of abdominal obesity in children and adolescents 2–18 years as defined by waist circumference greater than or equal to the sex- and age-specific 90th percentile based on NHANES III (1988–1994) is 18.87%, but this has been

steady since 2003–2004 (Xi et al., 2014).

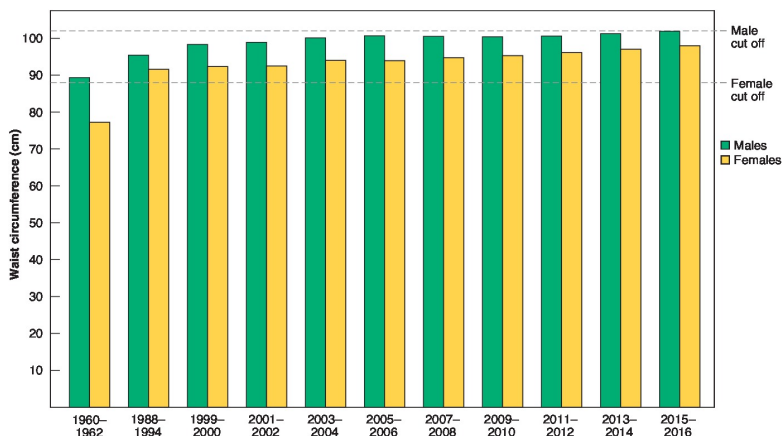


Figure 7.15 Waist Circumference in U.S. Adult Males and Females, 1960–2016.

Average values for males are below the 102-cm cutoff for abdominal obesity over this time span, but average female values are above the cutoff of 88 cm for abdominal obesity. Based on data from Fryar et al. (2018).

Health Risks of Overweight and Obesity

In 2013, the American Medical Association declared obesity a disease in and of itself. There are three common criteria for defining a disease. The first is an impairment of normal function of some aspect of the body. In obesity, there is an impaired functioning of appetite regulation, energy balance, endocrine function, and blood pressure to name a few. The second is the presence of characteristic signs and symptoms. For obesity, the increase in body fat is obvious, but, for example, joint pain, immobility, and sleep apnea are also evident. The third criterion is harm or morbidity. The fat mass of obesity is directly related to hypertension, cardiovascular-respiratory diseases, some cancers, and other comorbidities.

It is estimated that age-adjusted obesity-related mortality rates have increased by 142% from 1999 to 2016 (D'Souza et al.,

2018). The increase is more pronounced in men (173%), than woman (117%), which aligns with obesity statistics showing a larger increase in male populations. Even if mortality is not an immediate concern, the financial costs of being obese is also evident. It was found in a 2017 study that higher BMIs are directly associated with higher medical expenses and being obese raises direct medical costs per person by approximately \$3,500 per year (Biener et al., 2017).

Table 7.7 provides a listing of risk factors and diseases divided into those that result from metabolic changes caused by obesity and those that results from the increased mass of fat per se (Bray, 2000). Among equally obese individuals, those with the highest accumulation of visceral adipose tissue (VAT) show the severest deterioration in metabolic variables (Després and Lamarche, 2000). VAT appears to have a stronger relationship with physiological and pathological processes, metabolic syndrome, and cardiometabolic risk factors than total body fat and/or abdominal subcutaneous adipose tissue do (Demerath et al., 2008; Liu et al., 2010; Sardinha and Teixeira, 2005). Several of these diseases and/or risk factors are discussed below.

TABLE 7.7 Health Risks Associated with Overweight/Obesity

| From Metabolic Changes | From Mass of Fat per se |
|---|-------------------------------------|
| Diabetes mellitus type 2; insulin resistance | Osteoarthritis |
| Gallbladder disease | Sleep apnea |
| Hypertension | Breathlessness/respiratory problems |
| Coronary heart disease; stroke | Low back pain |
| Certain cancers (breast, colon, endometrial, pancreatic, prostate, and uterine) | Skin stretch marks |
| Dyslipidemia/hyperlipidemia | Congestive heart failure |
| (↑LDL-C, ↑TG, and ↓HDL-C) | |
| LDL-C, low-density lipoproteins | |
| TG, triglycerides | |
| HDL-C, high-density lipoproteins | |
| Impaired fertility; other reproductive problems | |
| Gout | |
| Digestive diseases | |
| Impaired kidney function | |
| Liver malfunction | |
| Stroke | |

Hypertension (High Blood Pressure)

Excess body weight and fat has a strong direct relationship with elevated blood pressure (Bray, 1987; Burton et al., 1985; Lee et al., 2007; Pi-Sunyer, 1993). Hypertension is also a very strong and independent risk factor for coronary heart disease.

Cardiovascular-Respiratory Diseases

A longitudinal study has been investigating the risk factors for heart disease in cohorts of more than 15,000 residents of Framingham, Massachusetts, since 1948. Data from years of follow-up have shown that overweight or obesity is a significant predictor of cardiovascular disease (CVD), independent of age, cholesterol levels, systolic blood pressure, cigarette smoking, and glucose tolerance. Obesity has also been shown to be a risk factor for atrial fibrillation in the heart, which in turn is a risk factor for cerebrovascular stroke. Based on this extensive data set, the Framingham investigators have concluded that if everyone were at or within 10% of his or her desirable weight, there would be 25% less coronary heart disease and 35% less congestive heart failure and stroke. The risk is greater for those who become obese early in life, as in childhood, rather than in old age (Bray, 1987; Burton et al., 1985; Lee et al., 2007; Pi-Sunyer, 1993; Simopoulos, 1987; Wang et al., 2004). Worldwide, the trends are similar. Epidemiological studies from not only the United States but also Europe and Asia have found that higher BMI values are significantly associated with increased incidence of coronary artery disease and ischemic stroke (Yatsuya et al., 2014). A recent 2020 meta-analysis found that higher BMI levels are associated with increased rates of cardiovascular events. Furthermore, among those with cardiovascular diseases, overall mortality rates displayed a U-shaped relationship with BMI (Dwivedi et al., 2020).

FOCUS ON RESEARCH | *Clinically Relevant*

Childhood Obesity and CVD Risk

Factors

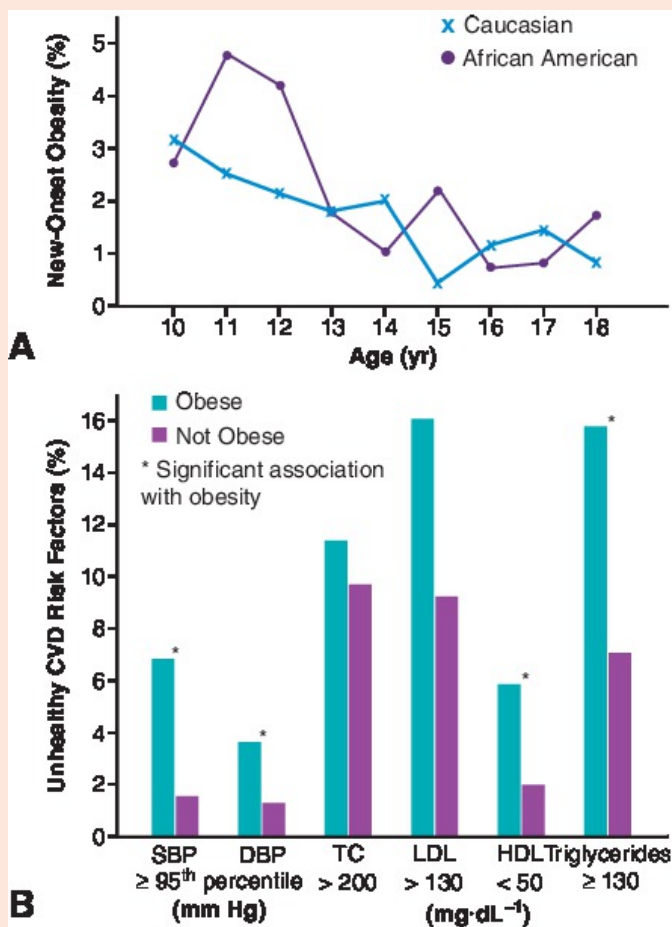
A total of 1,166 White (C) and 1,213 African American (AA) girls were tested in the National Heart, Lung, and Blood Institute Growth and Health Study annually between ages 9 or 10 years and 18 years and were contacted for self-reported measures at age 21–23 years. At the time the study began, BMI values above the CDC's age-specific 95th percentile were labeled as overweight. Because these values are now labeled obese, the term obesity will be used here. Young adult obesity was defined as a BMI ≥ 30 kg-m⁻².

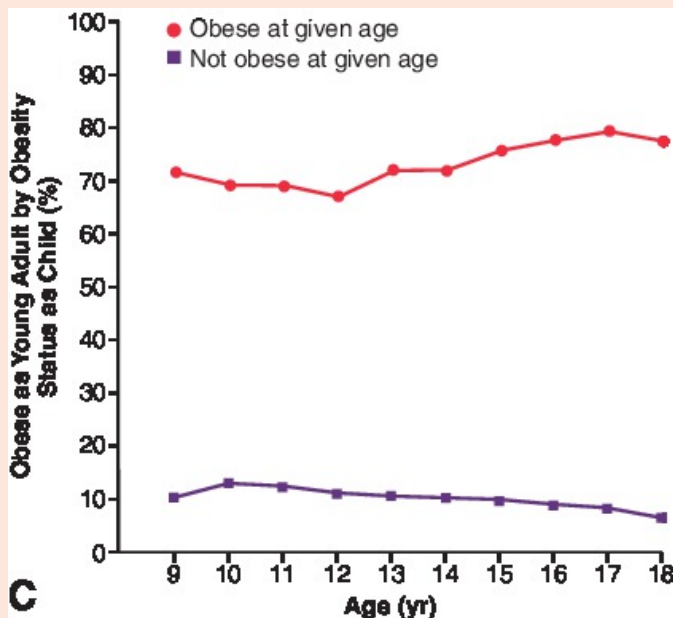
The rate of obesity increased throughout adolescence from 7 to 10% in the C girls and from 17 to 24% in the AA girls (not shown). Graph A shows the percent of new-onset cases (incidence) of obesity by race over the age span. The incidence ranged from 2 to 5% through age 12, after which the annual increase was generally in the 1–2% range. The important part of this information is the fact that it is the so-called tween years (ages 9–12 years) when girls are especially at risk of getting fat. Thus, particular attention and intervention should be directed toward this age group to prevent the increase in body weight and fat.

Graph B shows the percentage of selected cardiovascular risk factors in the C and AA groups combined, based on being or not being obese. Girls who were obese were 3–10 times as likely as those who were not obese to be assessed as “at risk” on four of the six cardiovascular risk factors: unhealthful levels of systolic and diastolic blood pressure (SBP and DBP), high-density lipoproteins (HDL), and triglyceride levels. The increased risk was already evident at age 9 years. Thus, there are meaningful health reasons for not delaying interventions.

Graph C shows the percentage of obese girls in young adulthood who were obese or not at each age in childhood. For example, 71.3% of the obese 9-year-olds were obese at approximately 21 years, but only 10.3% of the nonobese at age 9 were obese at approximately 21 years. Overall, girls who were obese during childhood were 11 to 30 times more likely to be obese in young adulthood. Given that the height and weight values were self-reported by the young adults,

and underreporting weight is a known problem, the risk could be even greater. These data clearly show that obesity tracks from childhood to adulthood and needs to be addressed with children.





Source: Thompson, D. R., E. Obarzanek, D. L. Franko, et al.: Childhood overweight and cardiovascular disease risk factors: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics*. 150:18–25 (2007).

However, several studies have found that waist circumference or waist-to-hip ratio may be a better predictor of CVD than BMI (Ji et al., 2018; Lavie et al., 2018; van Dijk et al., 2012).

A study of 276,835 Dutch children showed that higher BMI values during childhood (7–13 years of age) were associated with an increased risk of coronary heart disease in adulthood. The associations were stronger in boys than girls and increased as the child became older in both sexes (Baker et al., 2007). Among other things, the Focus on Research: Clinically Relevant box provides evidence of increased CVD risk factors in female children and adolescents with BMI values greater than the 95th percentile (Thompson et al., 2007).

As mentioned previously, the risk is higher for those who store their fat in the android pattern than in the gynoid pattern. While coronary heart disease is linked to diabetes mellitus,

hypertension, and hyperlipidemia (primarily metabolic changes), congestive heart failure is more related to the increase in total fat mass per se. A larger body mass increases circulatory demand, including an elevation in the cardiac output often operating against an elevated systemic vascular resistance induced by hypertension (Arciero and Nindl, 2004).

The increased sizes of the chest and abdomen in obese individuals alter respiratory patterns leading to ventilation-perfusion mismatches and predisposing the individual to hypoxia (too little oxygen) and carbon dioxide retention. Airway obstruction often leads to sleep apnea (absence of spontaneous breathing). If uncontrolled, this can lead to sudden cardiac death from ventricular arrhythmias (Arciero and Nindl, 2004).

Gallbladder Disease and Hypercholesterolemia (High Cholesterol)

In the Framingham study, individuals who were 20% or more above the average weight for their height were about twice as likely to develop gallbladder disease as those who were 10% less than the average weight. In another study, the frequency of gallbladder disease was largely explained by weight, age, and, in females, the number of viable pregnancies (parity). Obese females between ages 20 and 30 years had a 600% greater chance of having gallbladder disease than did average-weight females. Within all age groups, the frequency of gallbladder disease increased with body weight. The body weight of males with gallstones has also been shown to be significantly more than the body weight of males without gallstones (Bray, 1987; Pi-Sunyer, 1993).

Increased gallbladder disease in overweight or obese individuals can be at least partly explained by the effect of increased body weight and fat on cholesterol. Fatness has a significant positive relationship with cholesterol level. Cholesterol production is also related to body weight, such that 1 excess kg of body weight increases cholesterol production by 20–22 mg·dL⁻¹. Bile is produced in the liver and stored in the gallbladder and always contains some cholesterol. The bile of obese individuals is more saturated with cholesterol than is that of nonobese

individuals. This increased presence of cholesterol in bile is the likely cause of the increased risk of gallbladder disease ([Arciero and Nindl, 2004](#); [Bray, 1987](#)).

The prevalence of adverse lipid profiles (low high-density lipoprotein cholesterol [HDL-C], high low-density lipoprotein cholesterol [LDL-C], and high triglycerides [TG]) is higher in obese adults and high in overweight adults compared to normal-weight adults. They are highest in adults with elevated abdominal obesity ([U.S. Department of Agriculture, Department of Health and Human Services, 2015](#)).

Diabetes Mellitus

Diabetes is a disorder of carbohydrate (glucose) metabolism. Overweight or obesity appears to cause an increase in insulin resistance and deterioration in glucose tolerance (leading to high levels of blood glucose) and to aggravate the appearance of diabetes. The risk for developing diabetes mellitus increases with the degree of obesity and age of the individual. This is also linked to the increased levels of circulating fatty acids that are evident in the obese. Visceral abdominal tissue is more strongly associated with insulin resistance than abdominal subcutaneous fat ([Arciero and Nindl, 2004](#); [Bray, 1987](#); [Burton et al., 1985](#); [Pi-Sunyer, 1993](#); [Preis et al., 2010](#)). The prevalence of type 2 diabetes among all adults greater than 65 years old was 24.6% in 2014, up from 18.4 in 1999–2000. Among adults greater than 65 years old with abdominal obesity, the prevalence of type 2 diabetes was 30.3%, up from 23.8% in 1999–2000 ([Caspard et al., 2018](#)).

Cancer

The American Cancer Society has published data on 750,000 individuals studied between 1959 and 1972. In these studies, as BMI increased, so did the incidence of death from cancer, even independent of cigarette smoking. Overweight males were particularly susceptible to prostate and colorectal cancer; overweight females showed increased rates of breast, cervical, endometrial, uterine, and ovarian cancer. Waist circumference is independently and positively associated with both premenopausal

and postmenopausal breast cancer risk (White et al., 2015). The suspected link, at least for females, is the level of estrogen. Adipose tissue is a site for estrogen formation in all females and the major site in postmenopausal females. Estrogen formation is increased in overweight and obese individuals owing to the increased number of adipose cells (Bray, 1987; Charney et al., 1975; Pi-Sunyer, 1993; Simopoulos, 1987).

Metabolic Syndrome

The metabolic syndrome (MS) is a cluster of interrelated risk factors of metabolic origin that directly promote the development of atherosclerotic CVD and increase the individual's risk of diabetes. Clinical identification of MS in adults includes three or more of the following: abdominal obesity, high triglycerides, low high-density-lipoprotein cholesterol, hypertension, and insulin resistance. In an analysis of NHANES data from 1999 to 2011, the prevalence of MS increased with increasing BMI for both sexes. That is, only 8.6% of underweight and normal-weight adults were diagnosed with MS, whereas 33.2% of overweight adults and 61.6% of obese adults were diagnosed with MS. Adults with both metabolic syndrome and obesity had a significantly higher risk of all-cause mortality and cancer mortality (Shi et al., 2020).

An analysis of adolescents (12–19 years) in the NHANES database (1999–2008) revealed that the prevalence of metabolic syndrome in obese youth was approximately 7% for overweight and 35% for obese boys and 9.2% for overweight and approximately 25% for obese girls versus less than 2% in the normal-weight groups. The odds of MS for overweight boys was approximately 9 times and for obese boys were 67 times that of normal-weight boys, whereas for overweight girls, the odds were 6 times for overweight and 19 times greater than for normal-weight girls (Al-Hamad and Raman, 2017; Laurson et al., 2014).

Miscellaneous Disorders

In addition to the specific diseases just discussed, overweight or obesity has been linked to kidney and liver dysfunction, joint problems (osteoarthritis) including back pain and gout, endocrine disorders including reproductive problems, problematic response

to anesthetics for surgery, skin infections, and an impaired physical work capacity (ACSM, 1983; Aune et al., 2014; Pi-Sunyer, 1993). The increased prevalence of these diseases and problems applies to adults, but overweight or obese children also show increased risk factors, though not the actual diseases, compared with normal-weight children (Williams et al., 1992).

The diseases discussed in the preceding sections lead to increased mortality or decreased longevity. In and of itself, visceral fat is an independent predictor of all-cause mortality in males (Kuk et al., 2005). The associations between underweight, overweight, and obesity per se with these diseases and mortality are somewhat more complex.

The Obesity Paradox—Misleading Misnomer?

A paradox is something contrary to what is expected, and the *obesity paradox* refers to the fact that while obesity is linked with higher health risk and mortality, in certain chronic disease states, overweight and/or obesity appears to confer a survival advantage. Among these chronic disease states are coronary artery disease (after intervention), heart failure, peripheral artery disease, stroke, chronic obstructive pulmonary disease, HIV/AIDS, specific cancers, and end-stage renal disease/dialysis (Caan et al., 2018; Li and Bu, 2020; McAuley and Blair, 2011). Some obese individuals have normal cardiometabolic risk profiles, and some do not. The obesity paradox has been an evolving concept.

Flegal et al. (2005) estimated the relative risk of mortality (death) associated with different levels of BMI (underweight = BMI < 18.5 kg·m⁻²; overweight = BMI 25 to <30 kg·m⁻²; obese = BMI ≥ 30 kg·m⁻²) using data from NHANES I (1971–1975), II (1976–1980 with follow-up through 1992), and III (1988–1994 with follow-up through 2000). Underweight and obesity were associated with increased mortality relative to normal weight. However, overweight was not associated with excess mortality. The authors speculated that improvements in public health and medical care may have been responsible for this positive outcome for those who are overweight. Two subsequent studies extended the follow-up to 2004 and found similar results. In the first study (Flegal et al., 2007), which used the same databases with a longer follow-up, underweight was

linked with increased mortality from noncancer or non-CVD (primarily respiratory related) but not with cancer or CVD mortality. Obesity was linked with significantly increased mortality from CVD and obesity-related cancers (colon, breast, esophageal, uterine, ovarian, kidney, and pancreatic), but not with other cancers, noncancer, or non-CVD mortality. Overweight and obesity combined were linked with *increased* mortality from diabetes and kidney disease. However, overweight and obesity combined were also linked with *decreased* mortality from noncancer or non-CVD causes. Overweight in and of itself was not linked with cancer or CVD mortality and was linked significantly with decreased mortality from noncancer or non-CVD causes. This time, the authors speculated that the greater nutritional reserves of being moderately overweight may be associated with improved survival during recovery from situations such as infections or surgeries. Additionally, some proportion of the individuals classified as overweight based on BMI could actually be in a healthy range for %BF due to a larger amount of LBM. The second study ([McAuley et al., 2007](#)) using data from military veterans found that obesity was associated with substantially lower mortality risk in non-heart failure individuals.

A 2013 meta-analysis of 97 studies on a combined sample of 2.88 million individuals found that relative to normal weight (as assessed by BMI), all grades of obesity and only grades 2 and 3 combined obesity had an 18 and 29% significantly higher all-cause mortality relative to normal-weight individuals. However, grade 1 obesity alone was not associated with higher mortality, and overweight was associated with 6% significantly lower all-cause mortality ([Flegal et al., 2013](#)). Possible explanations included the greater likelihood of overweight and moderately obese individuals seeking earlier medical assistance and the benefits of higher metabolic reserves. What has been missing in this progression of studies is any measure of cardiorespiratory fitness.

By 2018, Flegal and Ioannidis claimed that the term “Obesity paradox” was a figure of speech and not a scientific term and argued that the use of this term should be abandoned. Part of their reasoning is that science and medicine should be studying why normal weight is not associated with better survival or why being underweight is associated with worse outcome in several

scenarios.

Much of the difficulty in determining whether the obesity paradox is real or not is in design flaws of the studies. Chief among these is the primary use of BMI as the measurement of obesity. As has been described earlier in this text, BMI fails to reflect body composition. It cannot distinguish between muscle and fat, nor the various types of fat (intramuscular, visceral, or subcutaneous). See [Figure 7.16](#) and note the differences in muscle and two of the three types of fat despite equal BMI values. A normal BMI can mask excessive adiposity and, conversely, an abnormal BMI does not always mean levels of adiposity sufficient to increase mortality risk. Perhaps most critically, BMI does not measure muscle. Thus, the apparent paradox may be due to overweight individuals (BMI between 25 and 30 kg·m⁻²) having a sufficient muscle reserve but not adipose levels high enough to increase mortality. In diseases such as cancer, the best evidence supports a survival advantage due to higher muscle reserves in overweight patients ([Caan et al., 2018](#); [Donataccio et al., 2021](#); [Donini et al., 2020](#); [Lee and Giovannucci, 2018](#)). The Focus on Application Box: Physical Activity/Physical Fitness and the Health Risks of Obesity describes several studies on the interaction between fitness, fatness, and mortality. In addition, a study by [McAuley and Beavers \(2014\)](#) dealt specifically with patient populations. Five observational studies of 30,104 patients with cardiovascular disease (87% male) were analyzed, and results indicated that cardiorespiratory fitness significantly altered the obesity paradox. Among patients with high cardiorespiratory fitness, risk of all-cause mortality was lowest for the overweight category in some, but not all, of these studies. This suggested to the authors that higher levels of fitness may modify the relationship between body fatness and survival. Thus, higher levels of fitness may improve the prognosis for survival, and this is no paradox—high fitness should lower mortality rates. High fitness is also typically associated with sufficient muscularity. However, [Carbone et al. \(2019\)](#) have suggested that CVR fitness be expressed as lean peak or maximal oxygen consumption (i.e., mL per kg of lean body mass), not per kg of total body weight.

FOCUS ON APPLICATION

Physical Activity/Physical Fitness and the Health Risks of Obesity

Epidemiological evidence indicates that there is a relationship between high physical activity/physical fitness and lower health risks of overweight/obesity. The health risks of overweight and obesity have been detailed in this chapter. It is well established that one way to reduce these health risks is to lose body weight and, more specifically, body fat. However, it is also well established that this is easier said than done, and the same is true for maintaining a weight loss that has been achieved. So, the question becomes whether physical activity or physical fitness can weaken or blunt the increased risk of morbidity or mortality in overweight/obese individuals who remain overweight or obese.

The compiled results of three studies shed some light on this question. These studies were conducted at the Institute for Aerobics Research, Cooper Clinic (Dallas, TX), and are based on approximately 22,000 males aged 20–60 years and 2,600 males and females over the age of 60 years. The results presented in the accompanying graph for males aged 20–60 years clearly show that regardless of how body composition is measured (BMI—using $27.8 \text{ kg}\cdot\text{m}^{-2}$ as the cutoff between normal and obese males, %BF, or waist girth), overweight or obese males who are fit have a lower risk of all-cause mortality (early death from all causes) than do unfit males (Blair and Brodney, 1999; Welk and Blair, 2000). Indeed, the fit obese males had much less risk of early death than did unfit lean males. This can be seen in the figure by comparing the red bars (fit), which have a relative risk (RR) of approximately 1.0, to the blue bars (unfit), where the RR ranges from 1.62 to 4.88. These higher-risk ratios mean that the unfit are 62% to almost 500% more likely to suffer early death than are the fit, regardless of body composition. In addition, the risk of all-cause mortality is similar for fit individuals, regardless of their body composition: lean,

normal, or obese, that is, the adjusted RR for the fit groups deviated only slightly from 1.0 (0.8 to 1.08, which indicates no excessive risk), regardless of body composition (Blair and Brodney, 1999; Welk and Blair, 2000).

Fitness in these studies was determined by a maximal treadmill test, and only the individuals in the bottom 20% (by age-specific distribution) were classified as unfit. In absolute values, this level of cardiovascular fitness is

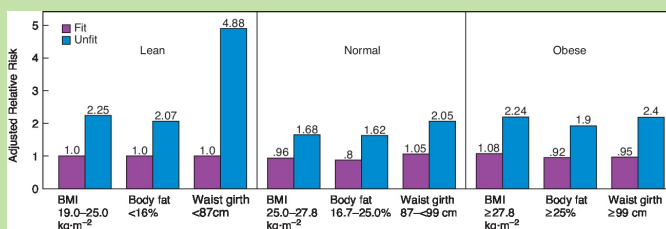
equivalent to a $\dot{V}O_2 \text{ max}$ of $35 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for 20- to 39-year-old males and $33 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for 40- to 59-year-old males. These fitness levels can be achieved by as little as 400–1,650 kcal of activity per week. This energy expenditure is consistent with 30 minutes of moderate activity on most days of the week.

A 2007 study (Sui et al., 2007) from the same laboratory using the same testing procedures extended these results to individuals more than 60 years of age including approximately 20% females. One change was that individuals were divided into four groups by BMI to match the new standards: $18.5\text{--}24.9 \text{ kg}\cdot\text{m}^{-2}$ (normal), $25.0\text{--}29.9 \text{ kg}\cdot\text{m}^{-2}$ (overweight), $30\text{--}34.9 \text{ kg}\cdot\text{m}^{-2}$ (level I obesity), and greater than $35 \text{ kg}\cdot\text{m}^{-2}$ (level II and III obesity). The upper limit of normal %BF was maintained at 25% for males and set at 30% for females. Death rates per 1,000 person-years, adjusted for age, sex, and examination year, were 32.6, 16.6, 12.8, 12.3, and 8.1 from the lowest to the highest quintiles of fitness. That is, the lowest 20% of this population based on

$\dot{V}O_2 \text{ max}$ $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ had the highest death rate. Higher fitness was linked to lower risk of death due to all causes. This was true in both normal and overweight BMI groups, for participants with normal waist size, abdominal obesity, normal %BF, and excessive fat. However, fit individuals who were obese (BMI = $30\text{--}34.9 \text{ kg}\cdot\text{m}^{-2}$, had abdominal obesity, or had excessive %BF) had a lower risk of all-cause mortality than unfit, normal-weight, or lean individuals.

The implications for health promotion are clear. There are health risks associated with overweight and obesity, and the

advice to lose weight remains sound. Weight loss remains one of the primary reasons individuals join a health club or exercise program. Many overweight/obese individuals, however, become frustrated when the desired weight loss does not happen or does not happen rapidly enough for whatever reason, and they quickly drop out of the exercise regimen. These individuals should be encouraged to remain or become more active in order to reap the physiological benefits of regular activity both separate from and linked to increasing physical fitness. The process of being active and becoming physically fit, rather than the product of changes in body weight or percent fat, should be stressed. Emphasizing the process is more likely to motivate overweight/obese individuals because participation in activity is within each person's control (Gaesser et al., 2015; Welk and Blair, 2000). If the process (regular physical activity) is accomplished, the product (physical fitness/lower health risks) will follow.



Sources: Blair and Brodney (1999); Gaesser et al. (2015); Sui et al. (2007); Welk and Blair (2000).

FOCUS ON APPLICATION

The Impact of Fatness on Fitness

While the previous Focus on Application box has shown that cardiovascular fitness positively impacts the health risks of overweight and obesity, the interaction of these two variables

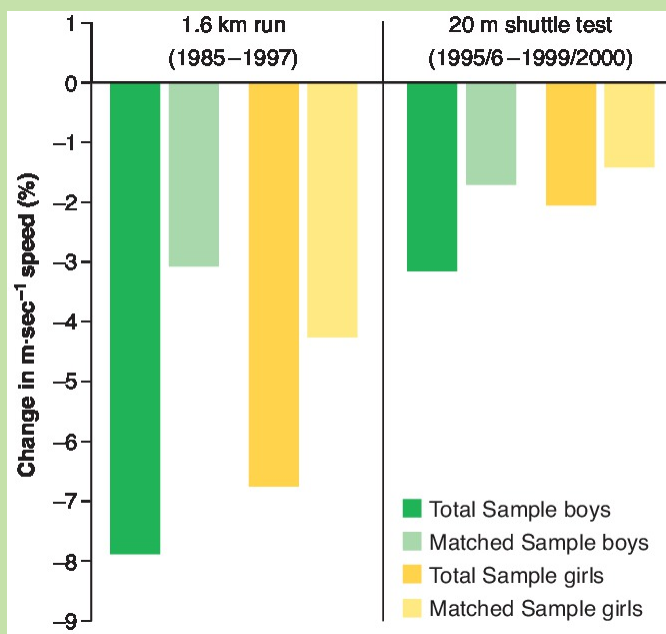
can be viewed in another way: how does fatness impact fitness? More specifically, do the increasing overweight/obesity levels of children and adolescents explain the decline in cardiovascular fitness that is now evident? Considerable evidence worldwide supports the contention that aerobic fitness is decreasing and body fatness increasing in children and adolescents. Variability in fatness is known to account for approximately 20% of the variability in running performance but are the changes in fatness totally responsible for the changes in aerobic fitness?

In this report, Australians aged 10–12 tested in 1985 were matched by age, sex, BMI, and triceps skinfold to children tested in 1997 (N = 279 matched pairs from a total sample of 2,748 participants) on the 1.6-km run (also known as the metric mile). Similarly, 12- to 15-year-olds tested in 1995–1996 were matched with adolescents tested in 1999–2000 on the 20-m shuttle run (20 MST) (also known as the PACER) (N = 2,834 matched pairs from a total sample of 7,938 participants).

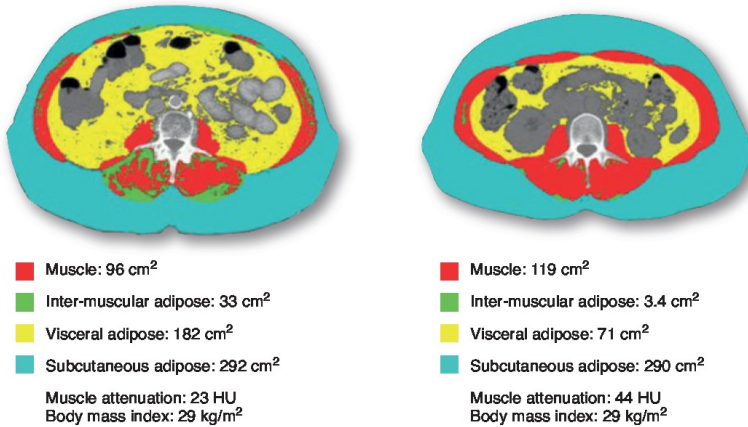
The accompanying graph shows the decrement in performance in the total samples for both of the fitness run tests. The values for the 1.6-km run declined approximately –7% to –8% and those for the 20 MST –2% to –3%. The lesser decline for the 20 MST is undoubtedly due to the shorter time between the testing sessions (4 vs. 12 years) for that test compared with the 1.6-km run. The differences for the matched samples are less than the total samples. In the 1.6-km run, the differences in fitness performance were reduced by 61% and 37% for boys and girls, respectively, by matching for BMI and skinfold thickness. For the 20 MST, the reductions for matching were 46% for boys and 29% for girls. In the matched sample statistical technique, if the declines in running performance seen in the total group had been eliminated in the matched sample, then these fitness declines could have been attributed solely to increases in fatness. However, this clearly did not happen.

These results are of more than just theoretical interest. If the goal is to reverse the decline in fitness (which it certainly should be), it is important to know whether it is due only to

increases in fatness, in which case weight reduction strategies alone are called for, or whether other factors are also important. The data indicate that other factors, such as reduced physical activity and a subsequent detraining effect, are likely to have contributed to the decline. This suggests that increasing physical activity levels in conjunction with dietary strategies is needed to improve aerobic fitness.



Source: [Olds et al. \(2007\)](#).



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Figure 7.16 Images often reveal differences in body composition that may not be apparent otherwise.

This image shows two female patients with identical body mass index but with very different body compositions.

Source: Reprinted from Caan, B. J., E. M. Cespedes Feliciano, & C. H. Kroenke: The importance of body composition in explaining the overweight paradox in cancer-counterpoint. *Cancer Research*. 78(8):1906–1912 (2018) with permission from AACR.

A meta-analysis on the impact of fitness versus fatness on all-cause mortality found that the unfit individuals had twice the risk of death as the fit individuals, regardless of BMI. Conversely, fit, overweight, and obese individuals all had similar mortality risk as their normal-weight counterparts. The risk of death was dependent on cardiovascular fitness and not BMI ([Barry et al., 2014](#)).

In general, lean unfit patients with CVD have a worse prognosis than do overweight or obese high-fit individuals because of the impact of fitness. Cardiorespiratory fitness is a powerful risk modifier that explains, at least in part, the obesity paradox ([Lavie et al., 2014a, 2014b](#); [McAuley and Beavers, 2014](#); [McAuley and Blair, 2011](#)). More recent research continues to support this conclusion. That is, fitness may be more important

than obesity as a risk factor for cardiovascular disease outcomes, and all-cause mortality. Weight gain may be less deleterious than the loss of fitness throughout the life span. Fitness is more important than weight/fatness for predicting prognosis in morbidity and mortality (Barry et al., 2018; Elagizi et al., 2018; Kennedy et al., 2018; Lavie et al., 2019).

Of course, developing a high level of cardiorespiratory fitness and maintaining proportional muscle mass for an individual who is overweight or overfat is not easy. The Focus on Application Box: The Impact of Fatness on Fitness shows that more than just fatness has led to a decrease in children's aerobic fitness levels.

Summary

1. In the United States in the early 1970s, the prevalence of obesity was 4–6% for children/adolescents and 15% for adults. In 2017–2018, 42.8 of U.S. adults were obese as were 19.3% of U.S. children and adolescents and 13.4% of infants and toddlers.
2. The major reference techniques for determining body composition are cadaver analysis and magnetic resonance imaging (MRI). Although a multicomponent model typically measuring densitometry, bone mineral by dual-energy x-ray absorptiometry (DXA), and hydrometry is preferred, DXA by itself is frequently used in laboratory situations as the new criterion measure for the determination of body composition. Hydrostatic, or underwater, weighing is the historical criterion measure for body composition. Air displacement plethysmography (BOD POD®) is also an important laboratory technique.
3. Densitometry usually divides the body into two components, fat and fat-free weight (FFW). FFW is composed of water, protein, and bone mineral. The components are known and are relatively stable in adults but not in children and adolescents. Thus, no single equation, and especially not the adult equations of Brozek or Siri, can be used for children.
4. Important field tests for assessing body composition and/or

abdominal obesity include skinfolds (good to very good assessments), bioelectrical impedance (good to very good), height and weight indices of body mass index [BMI] (fair), lean mass indices [FFMI], and waist circumference (good). BMI is associated with but does not actually measure %BF and lean body mass.

5. Waist circumference is strongly related to visceral abdominal obesity, which in turn is predictive of morbidity and mortality.
6. The size and the number of adipocytes increase as a child grows to adulthood. Adult fat gain first involves hypertrophy of the adipocytes and then hyperplasia of precursor cells.
7. Fat is typically distributed in an android (abdominal), gynoid (hips and thighs), or intermediate pattern. The android pattern is more strongly linked with cardiovascular disease (CVD) risk. Visceral adipose tissue appears to have a stronger relationship with physiological and pathological process than total body fat does.
8. In addition to being a disease itself, the health risks of overweight and obesity include CVD, hypertension, gallbladder disease, hypercholesterolemia, diabetes mellitus, and cancer. Paradoxically, in some chronic disease states, overweight/obesity as measured by BMI has appeared to confer a survival advantage. However, the real factor may be muscularity and cardiovascular fitness. Cardiorespiratory fitness is a powerful modifier of cardiovascular risk in overweight and obese individuals and partially explains the obesity paradox.

Review Questions

1. Defend or refute: Obesity has reached epidemic proportions.
2. List the laboratory techniques for body composition assessment. Define densitometry. Relate densitometry to hydrostatic weighing.
3. Explain the assumptions that must be met in order for

hydrostatic weighing to be accurate. What variations in these basic assumptions occur with children, adolescents, and the older adults? State two practical applications of this information.

4. What is the most commonly used new criterion for body composition measurement in the laboratory? Why is it preferred over hydrostatic weighing?
5. List and identify the strengths and weaknesses of the different field techniques for estimating overweight and obesity. Which technique would you select to use in a field setting? Explain why.
6. Compare the accuracy of %BF determined by skinfolds and bioelectrical impedance with %BF determined by hydrostatic weighing.
7. Compare and contrast the %BF, %BF distribution, and typical patterns of fat distribution between males and females including the health implications of the fat distribution patterns (acknowledging that not all males or all females exhibit the stereotype fat distribution).
8. Differentiate between overweight and obesity. What are the advantages and disadvantages of determining overweight and obesity by BMI?
9. What is fat free mass index (FFMI)? Why might it be useful to know for disease states and athletic performance?
10. What happens to adipose cells as an individual becomes overweight and then obese?
11. List and briefly discuss the health risks of being overweight or obese.
12. Define the obesity paradox. Describe the interrelationships among BMI, muscle mass, cardiovascular fitness, overweight/obesity, and the obesity paradox. Describe the term normal weight obese.

Literature Search

In this chapter, we discussed criterion and field assessment methods for determining body composition, as well as the role of

obesity in relation to disease. To explore these topics further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- (a) Search body composition. This will yield a huge selection of articles.
- (b) Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Body composition assessment methods
 - ii. Obesity and cancer
 - iii. Obesity and diabetes
 - iv. Obesity and metabolic syndrome
 - v. Prevalence of obesity in the United States
 - vi. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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8 Body Composition and Weight Control



CHAPTER OUTLINE

Introduction

The Caloric Balance Equation

- Food Ingested

- Resting or Basal Metabolism

- Thermogenesis

- Exercise/Activity Energy Expenditure

The Effects of Diet, Exercise Training, and Diet Plus Exercise Training on Body Composition and Weight

- The Effects of Diet on Body Composition and Weight

- The Effects of Exercise Training on Body Composition and

Weight

The Effects of Diet Plus Exercise Training on Body Composition and Weight

The Effects of Diet, Exercise Training, and Diet Plus Exercise Training on Abdominal Obesity

Application of the Training Principles for Weight and Body Composition Loss and/or Control

Specificity

Overload

Rest/Recovery/Adaptation

Progression

Individualization

Retrogression/Plateau/Reversibility

Maintenance

Weight Cycling

Making Weight for Sport

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- State the caloric balance equation, and define and explain its components.
- Explain whether more calories are expended at rest or during exercise throughout a normal 24-hour period.
- Discuss the impact of diet, as caloric restriction, on the components of the caloric balance equation.
- Discuss the impact of an exercise session on the components of the caloric balance equation.
- Discuss the impact of exercise training on the components of the caloric balance equation.
- Compare and contrast the effects of diet alone, exercise training

alone, and combined diet plus exercise training on body weight and body composition control.

- Apply the training principles to body weight and body composition control.
- Describe possible mechanisms that make maintenance of weight loss difficult.
- Compose guidelines for making weight in a sport.

Introduction

How much an individual weighs and what makes up his or her body mass are the result of the interaction of unmodifiable factors such as age, sex, and heredity and the modifiable factors of energy intake and energy output. Given the concern over the increasing levels of overweight and obesity described in [Chapter 7](#), it is critical to understand these modifiable factors. Literally, hundreds of books and articles are written each year on these topics, often with exaggerated claims and untested suggestions. This chapter provides current scientific knowledge about body composition and weight control.

The Caloric Balance Equation

At its most basic level, weight control follows the first law of thermodynamics. The **first law of thermodynamics**, sometimes called the **law of conservation of energy**, states that energy can neither be created nor destroyed, but only changed in form. This law was described fully in [Chapter 2](#).

First law of thermodynamics or the law of conservation of energy Energy can neither be created nor destroyed, but only changed in form.

When the body converts the potential chemical energy of food into other chemical, mechanical, or heat energy, it follows the

Law of Conservation of Energy. Theoretically, if the amount of energy taken in equals the amount of energy expended, the body is in balance and the weight (mass) remains stable. If an excess of energy is ingested, that energy is neither destroyed nor lost; rather, weight (mass) is gained. If insufficient energy is ingested in relation to expenditure, the needed energy cannot be created but must be provided from storage sites, and weight (mass) is reduced.

Energy, in the forms involved in the human body, is usually measured in kilocalories (kcal) or kilojoules (kJ). One **kilocalorie** is the amount of heat needed to raise the temperature of 1 kg of water by 1°C at 1 atmosphere. One kilocalorie is equal to 4,186 kJ. A calorie is equal to 0.001 kcal. Calorie with a capital C is equivalent to 1 kcal. To avoid confusion, this chapter always uses the unit kilocalorie, not calorie. The term calorie is often used generically, however, as in the statement “Caloric intake should be equal to caloric output,” even though the units would be kilocalories. The caloric equivalent of 1 lb of fat is roughly 3,500 kcal; however, in a physiological system, this 3,500 kcal rule is likely an overestimation for longterm weight change (Thomas et al., 2013).

Kilocalorie The amount of heat needed to raise the temperature of 1 kg of water by 1°C at 1 atmosphere.

The **caloric balance equation**, the mathematical sum of the caloric intake (+), and energy expenditure (–) from all sources quantifies the Law of Conservation of Energy. It describes the source of potential energy as the food ingested and the various uses of that energy. As indicated in **Figure 8.1**, input and output can be divided into the following components:

Caloric Balance Equation The mathematical summation of the caloric intake (+) and energy expenditure (–) from all sources.

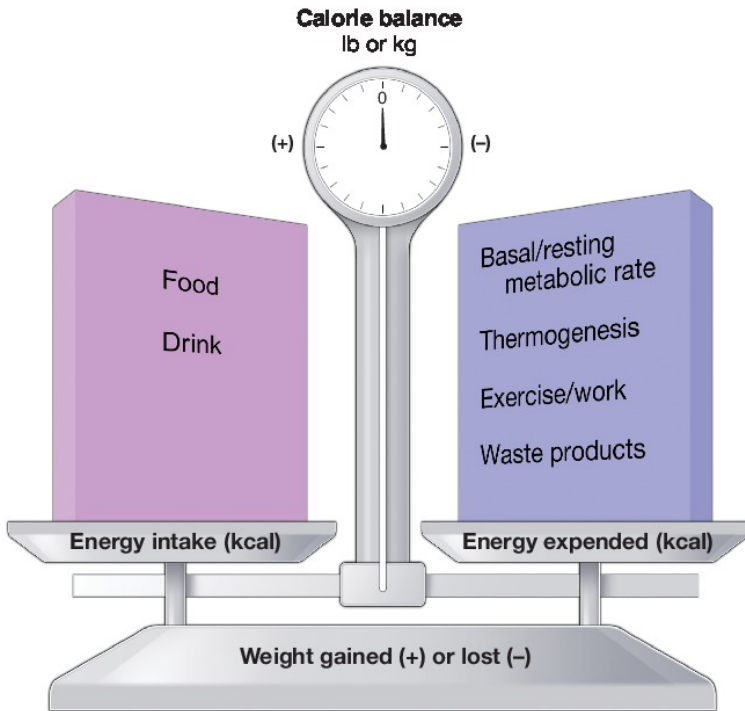


Figure 8.1 Caloric Balance.

Caloric balance is the mathematical sum of the caloric intake (+ food and drink) and energy expenditure (– BMR/RMR, – thermogenesis, – exercise/work, – waste products).

$$\begin{aligned}\text{caloric balance} = & + \text{food ingested (kcal)} \\ & - \text{basal or resting metabolic rate (kcal)} \\ & - \text{thermogenesis (kcal)} \\ & - \text{work or exercise metabolism (kcal)} \\ & - \text{energy excreted in waste products (kcal)}\end{aligned}$$

Food (and fluid) intake represents the only positive factor in the caloric balance equation. It is the only way that energy can be added to the system (the body). Energy is expended primarily in three ways: the basal or resting metabolic rate, thermogenesis, and work or exercise. These are the primary negative factors in the caloric balance equation. Although also negative, the amount

of energy excreted in waste products is insignificant, rarely measured, and need not be considered further here.

Figure 8.2 indicates that the basal or resting metabolic rate accounts for most of the *total energy expenditure* (TEE), varying from approximately 60% to approximately 75% in active and sedentary individuals, respectively. Thermogenesis (the production of heat) accounts for a relatively stable 10% of caloric expenditure in both sedentary and active individuals. The energy expenditure of exercise is obviously higher in active individuals than sedentary ones and depends on the intensity, duration, and frequency of exercise.

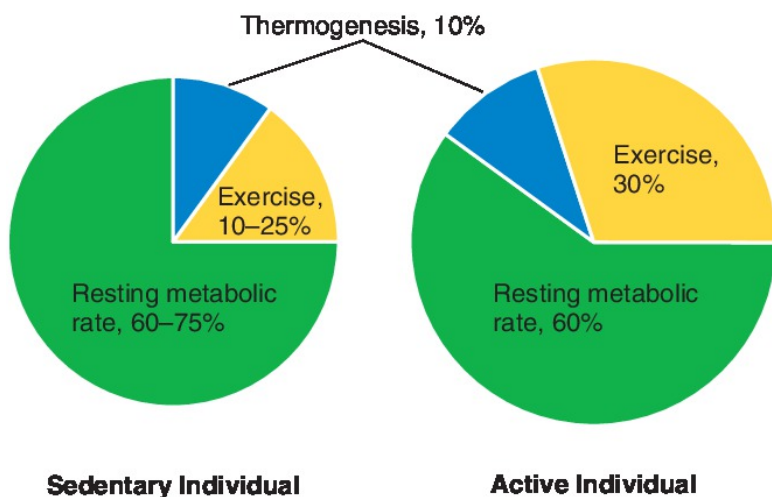


Figure 8.2 Energy Expenditure.

Resting metabolic rate accounts for the largest percentage of energy expended in both inactive and active individuals, although the exact percentage varies between the two groups. The percentage of energy expended during exercise is higher in active than in inactive individuals.

Thermogenesis accounts for about 10% of the energy expended in both active and inactive individuals.

If the amount of energy in the food ingested exceeds the energy expended, the body is in a positive balance and body weight will increase. If the amount of energy in the food ingested

is less than the energy expended, the body is in a negative balance and body weight will decrease. Many adults self-regulate an amazingly constant body weight throughout the years despite the ingestion of literally hundreds of thousands of calories; others struggle with self-regulation and either lose or gain weight. In the pages that follow, each of the positive (food intake including liquids containing calories) and negative (basal metabolic rate [BMR], thermogenesis, and physical activity) components of the caloric balance equation will be defined and discussed. (The impact of diet, exercise, and exercise training on each of the components is systematically addressed and summarized in **Table 8.2** at the end of the discussion.)

Food Ingested

The type and amount of food ingested are the result of many physiological, psychological, nutritional, behavioral, and sociological influences. It is not as simple as a physiological drive to balance energy demand and supply. People eat or do not eat for a variety of reasons. For example, when you are upset, do you cease eating, or do you consume everything in sight? Despite the diversity of influences, in this text, we will concentrate on the physiological controls.

Figure 8.3 presents a schematic of the basic physiological factors involved in appetite control. *Tonic appetite signals* arise from body composition and metabolic processes. Both fat-free mass and fat mass can impact resting metabolic rate (discussed later in detail). *Leptin*, an appetitesuppressing (anorexigenic) hormone primarily secreted by adipose tissue, is part of a negative feedback loop regulating the size of energy stores and energy balance over the long term (2–4 days). The release of leptin signals satiety, decreases food intake, and increases energy expenditure. *Insulin* produced by the pancreas (islets of Langerhans) is involved in glucose use, protein synthesis, and the formation and storage of lipids. Leptin and insulin are tonic or long-term energy-regulating hormones that operate over days or weeks. The reaction of leptin and insulin may possibly be described as an inverted U response. That is, low and very high levels of these stimulate appetite and food intake and suppress energy expenditure, whereas more typical moderate levels are

suppressive of appetite and food intake (Hagobian and Braun, 2010). Leptin's main role is to protect against weight loss in times of nutritional deprivation (Marieb and Hoehn, 2016).

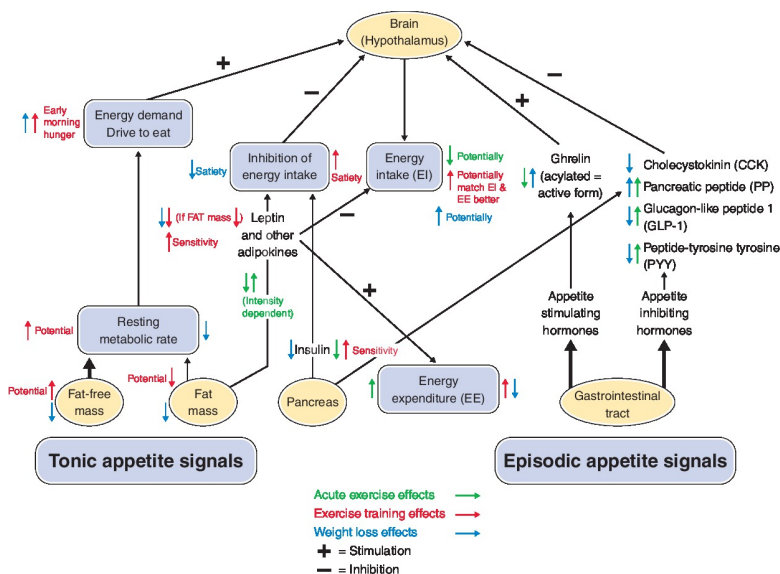


Figure 8.3 Appetite Control.

Appetite is controlled by both tonic (relatively stable over days) and episodic (varying during 1 day) signals originating in body tissues (fat-free mass and fat mass) and the gastrointestinal tract and operating through a variety of metabolic and hormonal signals that are coordinated in the brain. Acute exercise, chronic exercise training, and weight loss all impact the control of appetite. See text for details.

Sources: Blundell et al. (2015); Greenway (2015); Shubert et al. (2014); Sumithran and Proietto (2013).

Episodic appetite signals regulate meal initiation (thus the frequency of meals) and termination (thus meal size). Ghrelin, particularly the acylated (active) form, is the main episodic appetite-stimulating (orexigenic) hormone. It is secreted primarily by endocrine cells in the gastrointestinal tract. Ghrelin is fast acting such that its levels increase before meals and

decrease after meals and show additional daily variations (Kalra et al., 2005; Klok et al., 2007; Popovic and Duntas, 2005). In addition, a number of episodic peptides (amino acid compounds) such as cholecystokinin (CCK), pancreatic peptide (PP), glucagon-like peptide 1 (GLP-1), and peptide tyrosinetyrosine (PYY) function as appetite-inhibiting hormones. These hormones operate through specific areas in the hypothalamus (Blundell et al., 2015; Shubert et al., 2014).

The Impact of Diet on Food Intake

The term **diet** is used in many ways and often refers to the food regularly consumed during the course of normal living. It is also used to mean an intentional restriction of caloric intake. This second meaning is how the term is being used in this chapter. Therefore, “going on a diet,” by definition, means reducing food caloric intake.

Diet (1) The food regularly consumed during the course of normal living; (2) a restriction of caloric intake.

The Impact of Exercise on Appetite and Food Intake

The relationships among exercise, appetite, and energy intake are complex and often difficult to discern. Obviously, exercise involves the immediate expenditure of energy. **Figure 8.3** indicates what is currently known relative to the impact of an acute bout of exercise on tonic and episodic appetite signals. Insulin is the only tonic hormone impacted, and insulin decreases during exercise as part of the neurohormonal control of substrate utilization (**Chapter 2, Figure 2.17**). The major episodic appetite signals are modified in several ways: acylated ghrelin is decreased, while PP, GLP-1, and PYY are all increased. The result of these changes is a decrease in appetite stimulation by ghrelin and an increase in appetite suppression by the other three with the theoretical potential for a decrease in energy intake. To test if this theory occurs in practice requires the measurement of food

intake. Unfortunately, it is very difficult to accurately measure food intake in free living humans. The act of feeding individuals in a controlled setting where food can be measured may cause changes in eating behavior. Asking individuals to write down everything they eat requires faith that they are neither overreporting nor (more likely) underreporting their consumption. For these reasons, conducting studies on the effects of exercise on appetite in humans is difficult. Despite the difficulties, the following generalizations can be drawn from available studies:

1. There is little to no effect of exercise on either appetite or energy intake within a single day or in the short term. An immediate transient decrease in appetite for 20–30 minutes

after aerobic exercise at greater than 60% $\dot{V}O_2 \text{ max}$ (but not resistance exercise) has been seen relatively consistently in both males and females, but not always. This is often termed “exercise-induced anorexia” (Dorling et al., 2018; Hagobian et al., 2013; Laan et al., 2010). However, neither a reduction nor an increase in energy intake after the immediate transient decrease for up to 48 hours following a single bout of exercise has been clearly established (Balaguera-Cortes et al., 2011; Blundell and King, 1999; Dorling et al., 2018; Titchenal, 1988; Wilmore, 1983). In one study, the subjective rating of appetite was lowered in female subjects following 30 minutes of exercise at 50%

$\dot{V}O_2 \text{ max}$. Despite this decreased subjective rating, food intake at the postexercise meal and for 2 days after was not affected. Other studies (Maraki et al., 2005; Martins et al., 2007; Moore et al., 2004; Weigle et al., 2005) have shown a decreased appetite during exercise, an increased appetite after exercise, and an increased energy intake at the next meal, but either no change or a decrease in total energy balance for the day. The dietary composition of the meals may impact which of these occurs before the next meal, as can be seen in the Focus on Research box (Luscombe-Marsh et al., 2005). When increased energy intake follows an exercise bout, it typically occurs only with heavy exercise, and the compensation is often incomplete. For example,

when six young lean ($\text{BMI} = 21.4 \text{ kg}\cdot\text{m}^{-2}$) females exercised moderately (two 40-minute cycling sessions that burned 454 kcal), no significant increase occurred in daily energy intake above a no exercise condition. However, when the exercise was heavy (three 40-minute cycling sessions that burned 812 kcal), energy intake increased but only compensated for approximately 30% of what had been expended, resulting in a negative energy balance (Stubbs et al., 2002). Another exception to the lack of an effect of exercise on appetite seems to occur in swimming, depending on water temperature. Energy intake has been shown to be as much as 44% higher after exercising in cold water (20°C) than in thermoneutral (33°C) water and 41% higher than under nonexercise conditions (White et al., 2005). A 2014 review found no effect of exercise on energy intake in 69% of 40 acute studies and 50% of 10 short-term studies (Donnelly et al., 2014). Recently, another single study showed that exercise may not have a large effect on energy intake. Individuals who exercised six times per week lost significantly more weight compared to those who exercised just two times per week or not at all (control group). This shows that the larger energy expenditure from exercise was not compensated by increasing energy intake because there were no significant differences in energy intake between groups (Flack et al., 2020).

2. An activity-induced negative energy balance can be tolerated without compensation for approximately 1–2 weeks. However, subsequently, food intake spontaneously increases and compensates for approximately 30% of the energy expended in exercise. Thus, this compensation is partial and remarkably similar to that seen after heavy exercise on the same day. Furthermore, individuals tend to be either compensators (as high as 60%) or noncompensators (as low as 0%), for unknown reasons (Blundell et al., 2003, 2015). The difference may depend on the fitness and body composition status of the individual. Highly trained athletes and lean individuals usually increase their energy intake in response to increased training loads. Untrained and/or obese individuals often do not initially change energy intake in response to exercise training (Drenowatz, 2015; Jokisch et

al., 2012).

3. The response of appetite to exercise is not sex specific. The menstrual cycle does cause fluctuations in appetite, appetite-related peptides, and energy intake during the different phases for the female. It was demonstrated experimentally that when males and females expended similar amounts of energy (30% of total daily energy expenditure) in acute exercise, there was no difference between the sexes in subjective appetite, appetite hormones, or energy expenditure (Hagobian et al., 2013). Relative energy intake postexercise was suppressed equally. Furthermore, a meta-analysis revealed that the compensatory eating response to acute exercise was not different between the sexes (Schubert et al., 2013). Similarly, in another meta-analysis of data from 20 studies involving 241 participants (~78% male), Schubert et al. (2014) reported small to moderate decreases in acylated ghrelin and increases in three anorexigenic hormones (PYY, GLP-1, and PP) that should suppress hunger with no statistical difference between males and females in ghrelin and PYY.

The Impact of Exercise Training on Appetite and Food Intake

Exercise-trained males experience appetite suppression immediately postexercise but data are equivocal for exercise-trained females. Several studies have investigated the influence of exercise training on appetite-regulating hormones and measures of appetite to definitively identify the adaptations in both males and females, normal-weight sedentary, and/or overweight/obese sedentary individuals (Howe et al., 2014). There is some evidence of reductions in leptin after both aerobic and resistance exercise training but still much ambiguity about the adaptations of the peptides PYY, GLP-1, and PP (Dorling et al., 2018). A meta-analysis reported that chronic exercise significantly reduces leptin independent of age and sex. Additionally, the decreases in leptin concentrations are directly associated with decreases in adiposity (Fedewa et al., 2018). These findings are expected because leptin is primarily produced by adipocytes. However, the effect of exercise training will be mediated by changes in body

composition in addition to the short-term changes brought about by each acute exercise session (**Figure 8.3**). If training brings about an increase in fat-free mass, there will be an increase in early morning hunger and drive to eat. If training brings about a decrease in fat mass, there will be a decrease in leptin accompanied by increased satiety control. Thus, although overall energy intake may increase, there should be a better match of energy intake to energy expenditure ([Blundell et al., 2015](#); [Bouassida et al., 2010](#)). This is seen in physically active males, females, adults, and children—such as heavy manual laborers and athletes who consume more calories than sedentary individuals yet generally maintain their body weight and composition at or below normal levels. When chronic exercise training ceases, energy intake in humans is spontaneously reduced. Unfortunately, this reduction does not appear to be matched to the reduced energy expenditure. The result is often a positive energy balance, a regain of lost body weight, and a concomitant increase in body fat ([Blundell et al., 2003](#); [Stubbs et al., 2004](#)).

Thus, it appears that the physiological factors that modify appetite/food intake in response to hunger or a deficit energy intake are more finely tuned or are more efficient than those factors that respond to satiety or an increase in energy intake. It has been speculated that this response to food may have been a survival of the species mechanism in days before modern agriculture provided more than enough food for much of the world ([Prentice and Jebb, 2004](#)). In other words, humans may have evolved to eat more than needed in times of plenty, in expectation of coming times of less food availability. Thus, it truly may be easier to gain weight than to lose weight.

Resting or Basal Metabolism

Basal energy expenditure, more commonly called **basal metabolic rate (BMR)**, is the level of energy required to sustain the body's vital functions in the waking state. Technically, this definition means that the individual is resting quietly in a supine position, not having eaten for 8–18 hours, is at normal body temperature (37°C) in a neutral ambient temperature (27–29°C; 80–84°F), and is not experiencing any psychological stress. Because of the difficulty of achieving truly basal conditions in

laboratory settings, the term **resting metabolic rate (RMR)** is probably a more accurate descriptor. RMR is the energy expended while an individual is resting quietly in a supine position. These two terms are often used interchangeably, since the measured differences are small. That practice will be followed here, with the term RMR used primarily but not exclusively (Bursztein et al., 1989). As shown in **Figure 8.2**, RMR accounts for about 60–75% of energy expenditure at rest.

Basal Metabolic Rate (BMR) The level of energy required to sustain the body's vital functions in the waking state, when the individual is in a fasted condition, at normal body and room temperature, and without psychological stress.

Resting Metabolic Rate (RMR) The energy expended while an individual is resting quietly in a supine position.

Many organs and processes are responsible for energy consumption at rest. The liver is the largest consumer of energy at rest (29–32%), followed by the brain (19–21%), muscles (18%), heart (10%), lungs (9%), and kidneys (7%). Resting muscle energy is primarily for the maintenance of tonus or tension, necessary even in sleep. On the cellular level, the energy is used to fuel ion pumps (particularly the sodium-potassium pump), synthesize and degrade cellular constituents, conduct electrical impulses, and secrete various substances, including hormones (Bogert et al., 1973; Bursztein et al., 1989).

Basal or resting metabolism is usually determined in relation to body surface area (BSA) and expressed in $\text{kcal}\cdot\text{m}^{-2}$. It may

also be expressed in $\text{kcal}\cdot\text{d}^{-1}$ or $\text{VO}_2\text{mL}\cdot\text{min}^{-1}$. The choice of measurement unit depends on how the information will be used. To compare with submaximal or maximal oxygen values,

the choice is $\text{VO}_2\text{mL}\cdot\text{min}^{-1}$. To express caloric needs, the kilocalorie unit is used. The most common way to calculate BMR/RMR is the use of prediction equations such as those presented in

Table 8.1.

| TABLE 8.1 Estimation of Basal (Resting) Metabolic Rate | |
|--|--|
| Males | $\text{RMR} = 88.362 + (4.799 \times \text{HT}) + (13.397 \times \text{WT}) - (5.677 \times \text{AGE})$ |
| Females | $\text{RMR} = 447.593 + (3.098 \times \text{HT}) + (9.247 \times \text{WT}) - (4.330 \times \text{AGE})$ |

HT, height (in centimeters); WT, weight (in kilograms); AGE, age (in years).

Source: Based on [Roza and Shizgal \(1984\)](#).

Eighty-five percent of all normal subjects have estimated RMR values within 10% of their measured values. The other 15% of the population have either higher or lower values. The range of variation may exceed 20%. Thus, an individual with an RMR 20% higher than average can ingest more calories without gaining weight, but an individual with a 20% lower RMR must ingest less or gain weight ([Bursztein et al., 1989](#); [Guyton and Hall, 2020](#)).

Two criterion measures are available for RMR. The first is open-circuit indirect calorimetry, described in [Chapter 4](#). The second is a blood test for the determination of protein-bound iodine. The iodine comes from thyroxine (T4), the hormone secreted by the thyroid gland, which has the greatest impact on BMR. This test gives a relative indication of BMR, not a direct kcal·d⁻¹ value ([Bogert et al., 1973](#)).

The prediction equations in [Table 8.1](#) give an indication of the primary nonhormonal factors that influence RMR. What are they? If you said body size, age, and sex, you are correct. RMR is related strongly to BSA externally and to cell mass internally, so, of course, what that cell mass is composed of (fat or muscle) will have an influence. Obese individuals have a larger surface area and a larger cell mass (both fat and fat-free) than average-weight individuals. Therefore, not surprisingly, the RMR of obese individuals is higher than that of normal-weight individuals

(Jequier, 1987). The linear relationship between RMR and fat-free mass is identical in obese and lean people. However, although FFM and FM are good predictors of RMR, they account for only about 70% of RMR variability between individuals. Some of the residual RMR variation may be due to differences in organ mass (Hall, 2012).

The influence of age and sex can be seen in **Figure 8.4**. RMR is highest in infants and young children. The decline from age 6 to 18 is approximately 25%, or 2% per year. The decline then slows to about 2–3% per decade after that age. Part of this decline in RMR may be attributed to and be responsible for the increment in %BF that usually occurs as people age.

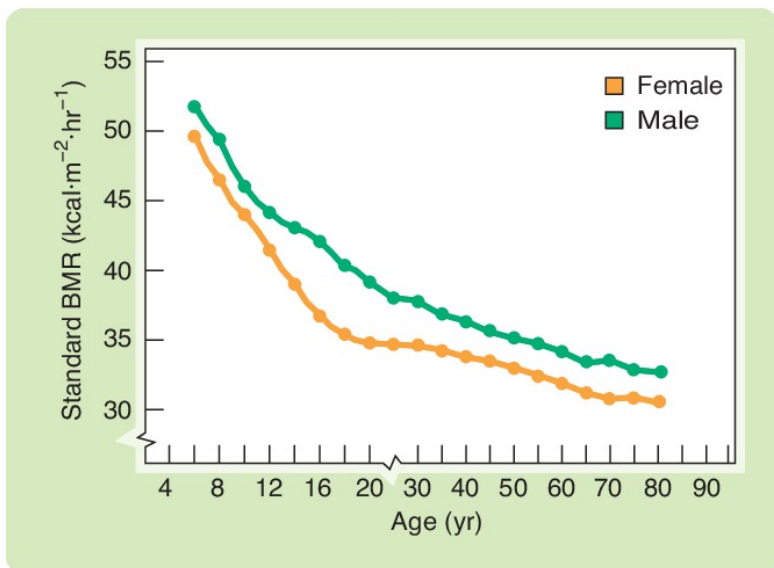


Figure 8.4 Standard Basal Metabolic Rate across the Age Span.

Standard basal metabolic rates decline steeply during childhood and adolescence and then more gradually during the adult years. At every age, male values are higher than female values, despite being prorated to body surface area.

Sources: Calculated from [Brownell et al. \(1987\)](#); [Bursztein et al. \(1989\)](#).

Figure 8.4 also clearly shows that, at all ages, average-weight females have lower RMR than average-weight males. The difference, which is least in young children, is accentuated at puberty and then tends to remain at about 5–6% through middle age and old age. In terms of calories, the RMR of adult males averages between 1,500 and 1,800 kcal·d⁻¹, and the RMR of adult females averages between 1,200 and 1,450 kcal·d⁻¹. Female values are lower partially because females have smaller internal organs than males and tend to have less total body mass, on average, than males. However, the most obvious explanation is the difference in body cell mass, particularly muscle tissue. Females on average have a higher %BF than do males. At any body weight, for each 1% increase in percent body fat, the RMR decreases 0.6 kcal·hr⁻¹ or 14.4 kcal·d⁻¹. If we assume a 10% difference in %BF between the average male and average female, the result can amount to 144 kcal·d⁻¹. When RMR is expressed relative to fat-free mass (FFM), the sex differences disappear (Bogert et al., 1973; Bursztein et al., 1989).

Another factor that influences RMR is core body temperature. For each degree increase in Celsius or Fahrenheit temperature, metabolic rate increases to 13% or 7.2%, respectively. This holds true even when the core temperature increases due to acute illness (pyrogenesis). Similarly, a decrease in body temperature, at least until the point where shivering is induced, reduces energy expenditure.

The Impact of Diet on Resting Metabolic Rate

Both the amount and the type of food ingested affect RMR. The effect of caloric restriction on RMR is well documented and clear-cut. Severe caloric restriction decreases RMR possibly as much as 10–20% after several weeks (Apfelbaum et al., 1971; Bray, 1969; Brownell et al., 1987; Grande et al., 1958a; Mole et al., 1989; Stiegler and Cunliffe, 2006). This is shown experimentally in the Focus on Research box where RMR decreased approximately 80 kcal·d⁻¹, regardless of the composition of the diet (Luscombe-Marsh et al., 2005). Because resting metabolism represents the greatest percentage of daily caloric expenditure in sedentary individuals, the discouraging result is a slowing of the weight loss that would be expected from the amount of dietary restriction

and negative balance if the $3,500\text{-kcal}\cdot\text{lb}^{-1}$ rule of thumb is used and the expectation is that the only change that occurs is a loss of body fat.

Example

1 lb (0.45 kg) of body fat contains the energy equivalent of approximately 3,500 kcal. If an individual expends $2,000\text{ kcal}\cdot\text{d}^{-1}$ and ingests $2,000\text{ kcal}\cdot\text{d}^{-1}$, weight should be maintained. If that same individual maintains this activity level but reduces his/her caloric intake to just $1,000\text{ kcal}\cdot\text{d}^{-1}$, a weight loss of $2\text{ lb}\cdot\text{wk}^{-1}$ would be anticipated ($2,000\text{ kcal}\cdot\text{d}^{-1} - 1,000\text{ kcal}\cdot\text{d}^{-1} = -1,000\text{ kcal}\cdot\text{d}^{-1}$; $-1,000\text{ kcal}\cdot\text{d}^{-1} \times 7\text{ d}\cdot\text{wk}^{-1} = -7,000\text{ kcal}$; $-7,000\text{ kcal} \div 3,500\text{ kcal} = -2\text{ lb}$), assuming that approximately 75% of the $2,000\text{ kcal}\cdot\text{d}^{-1}$ expenditure or $1,500\text{ kcal}\cdot\text{d}^{-1}$ is expended by RMR.

However, within 2–3 weeks of such a restricted diet, the RMR will have been reduced by approximately 15% (the range is typically 10–20%) to $1,275\text{ kcal}\cdot\text{d}^{-1}$. The difference is now $-1,775\text{ kcal}\cdot\text{d}^{-1}$ expended + $1,000\text{ kcal}\cdot\text{d}^{-1}$ ingested = $-775\text{ kcal}\cdot\text{d}^{-1}$; $-775\text{ kcal}\cdot\text{d}^{-1} \times 7\text{ d}\cdot\text{wk}^{-1} = -5,425\text{ kcal}$; $-5,425\text{ kcal} \div 3,500\text{ kcal}\cdot\text{lb}^{-1} = -1.5\text{ lb}$ instead of $-2\text{ lb}\cdot\text{wk}^{-1}$. As the drop in RMR continues with severe caloric restriction, the weight loss becomes progressively slower (Mole et al., 1989).

Researchers speculate that this decline in RMR is the body's protective response to energy restriction (Speakman and Selman, 2003). Conversely, a short-term excessive ingestion of food and calories results in an elevation of RMR (Apfelbaum et al., 1971; van Zant, 1992). This elevation has been seen as protection against an increase in the body's "natural" weight (Brownell et al., 1987).

Maintenance of a "natural" weight or body composition is often attributed to an individual's genetically determined set

point. According to the *set point theory*, changes in body mass or fat content are perceived in the periphery and appropriate signals sent to the hypothalamus. The hypothalamus integrates and interprets the incoming feedback and in turn sends out signals that modify food intake or energy expenditure (including but not limited to RMR) to correct any deviations in weight or fat from the set point. The existence of a set point for homeostatic control of human body weight/fat is uncertain and under debate. At the very least, a set point cannot be a fixed permanent value, as it is necessary to allow for such things as growth and development. It may be that the set point actually functions as a “settling point” with new levels set after continuous demands on the outer limits of the acceptable range. These demands may arise from a variety of influences including overfeeding or underfeeding without changes in activity level (Harris, 1990; Macias, 2004).

The Impact of Exercise on Resting Metabolic Rate

The energy cost of exercise (**Figure 8.5**), in oxygen or calorie units, includes a resting component. Metabolic equivalents (METs) ([Chapter 4](#)) express the energy cost of activity in multiples of the RMR. Therefore, although metabolism is definitely elevated by exercise, it is not the resting metabolism itself that is elevated. The resting metabolism is assumed to remain constant; the increase in energy consumption is attributed solely to the activity demands and responses.



Figure 8.5 Exercise Is an Important Component of Body Weight/Body Composition Control.

Immediately after exercise, the metabolic rate remains elevated. This rate is called the excess postexercise oxygen consumption (EPOC) and is discussed in [Chapter 3](#). This recovery oxygen utilization represents additional calories that are expended as a direct result of the response to exercise. These calories are typically not included in the measured caloric cost of the activity. Thus, if an individual expends 250–300 kcal walking or jogging 3 mi, an extra 20–30 kcal may be expended during the hour or two after exercise until complete recovery occurs. Additionally, a slow component that may last up to 48 hours expends even more calories ([Speakman and Selman, 2003](#)). The energy expenditure of EPOC for high-intensity interval training and sprint interval training may be larger than for continuous activity. The energy expenditure of EPOC for a traditional resistance training session is also minimal and is estimated to be approximately 35 kcal for a 60 min workout ([Arney et al., 2019](#)). These expenditures also do not mean that the RMR itself has been affected.

For it to be concluded that the resting metabolism is changed by exercise, the change would have to be evident 24 or 48 hours after exercise. Research evidence for such a change is mixed and

difficult to interpret. The inconsistency of the evidence can partially be attributed to the intensity and duration of the exercise involved. Mild to heavy exercise of moderate duration (35–86% $\dot{V}O_2 \text{ max}$ for 20–80 minutes) has generally been shown to cause no long-term metabolic elevation in RMR, whereas longer, moderate exercise (50–70% $\dot{V}O_2 \text{ max}$; 80–180 minutes) has.

There is a problem, however, in interpreting metabolic changes greater than 48 hours after exercise as being caused by the exercise. Studies showing an increase in RMR over the long term, even after heavy exercise, have not controlled for the ingestion of food during the intervening 48 hours. The elevated metabolism observed was probably due, at least in part, to the thermic effect of the meals taken. (The TEM will be fully explained later in this chapter.) Consequently, it is unlikely that exercise causes any permanent change in RMR per se—at least not light or moderate aerobic endurance exercise or dynamic resistance activity (Bingham et al., 1989; Horton, 1985; Melby et al., 1993; Stiegler and Cunliffe, 2006).

The Impact of Exercise Training on Resting Metabolic Rate

The effect of exercise training on RMR is complex and remains somewhat controversial. Results of cross-sectional studies involving RMR and training/fitness status have reported higher, lower, or the same values between trained-untrained and high fit-low fit groups. Similarly, training studies have produced inconsistent results ranging from an increase, to no change, to a decrease. However, a meta-analysis (MacKenzie-Shalders et al., 2020) clearly showed that resistance exercise programs generated increases in RMR, but neither aerobic programs alone nor aerobic plus resistance training induced any change in RMR. Perhaps surprisingly, the effect of resistance exercise was most evident when total body mass remained stable during the intervention period. But, this may be because fat was lost and muscle was gained in these programs. However, one study reported that every 1 lb of muscle only contributes an increase of approximately 6 kcals in total daily energy expenditure (Wang et

al., 2011). Perhaps, the effects of resistance exercise on RMR go beyond just those of additional FFM.

FOCUS ON RESEARCH

Macronutrient Impact on Appetite and Thermic Effect of a Meal during Caloric Restriction

Fifty-seven individuals, aged 20–65 years, with BMI values between 27 and 40 kg·m⁻², completed 12 weeks of caloric restriction and 4 weeks of maintenance. The purpose was to determine the effects of two calorically equal diets that differed in protein and fat content on weight loss, appetite regulation, and the thermic effect of test meals. The low-fat, high-protein (LF-HP) diet consisted of 29% fat, 34% protein, and 37% carbohydrate. The high-fat, standard-protein (HF-SP) diet consisted of 45% fat (~29% monounsaturated), 18% protein, and 37% carbohydrate. Caloric intake was set at approximately 1,400 kcal·d⁻¹ for both groups during weight loss and at energy balance during maintenance.

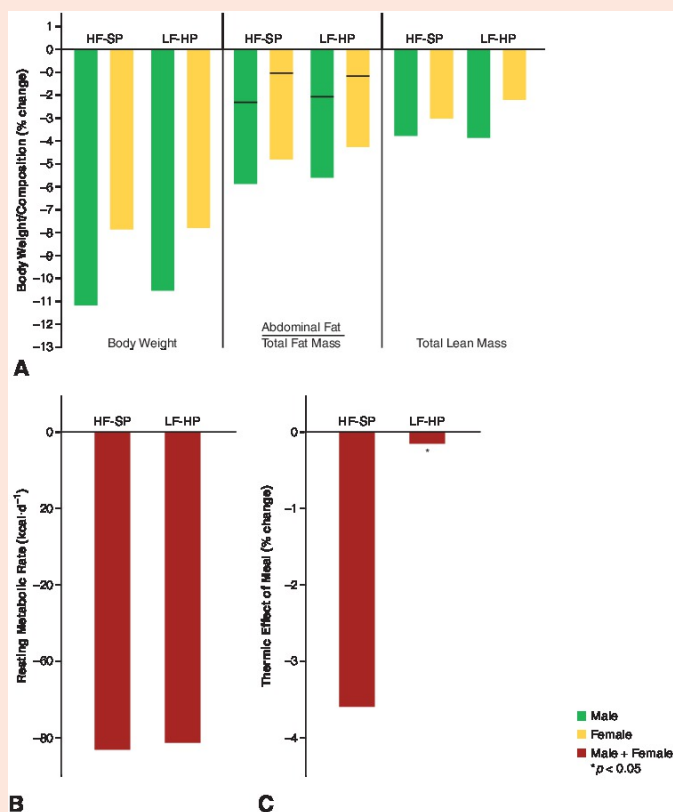
Hunger, fullness, satiety, desire to eat, and the amount of food desired were measured before and at 30, 60, 120, and 180 minutes after the test meal. Participants desired less to eat after the LF-HP meal than after the HF-SP meal both before and after the 16 weeks of dieting and maintenance. This indicates a greater satiety with a high-protein content despite equal total calories.

Total body weight loss, total fat mass loss, and abdominal fat mass loss are shown in the accompanying graph (A). The males lost more in each category than the females; however, there was no significant difference between the diet groups. This is consistent with the conclusion of most caloric restriction studies. That is, it is the energy intake and not macronutrient composition that primarily determines total weight and fat loss.

The differences in energy expenditure are presented in

graphs B and C. The decrease in RMR (B) was not significantly different between diet groups. However, the decrease in the thermic effect of a meal (TEM) with weight loss (C) was smaller in the LF-HP groups than in the HF-SP group. The authors concluded that although over the short term of the study this difference did not appear to enhance weight loss, it might have some small effect on weight maintenance later.

Thus, a high-protein diet, still within the recommended level of 10–35%, had the advantage over a high monounsaturated fat diet of blunting both the appetite and the dietary-induced decrease in the TEM.



Source: Luscombe-Marsh, N. D., M. Noakes, G. A. Wittert, J. B. Keogh, P. Foster, & P. M. Clifton: Carbohydrate-restricted

diets high in either monounsaturated fat or protein are equally effective at promoting fat loss and improving blood lipids. *American Journal of Clinical Nutrition*. 81:762–772 (2005).

Longitudinal studies investigating the impact of exercise training on RMR often add the exercise component after several weeks of severe caloric restriction in an attempt to reverse the decline in RMR. Others simply examine the impact of training on RMR during a calorically balanced state. High-intensity programs performed daily have not only brought about a return to baseline in RMR but also, in some instances, caused an elevation of 7–10%. In those studies showing a reversal of the decline in RMR, the change began within just a few days, which is well before any change would be expected in body composition. Thus, the conclusion again is that the effect of training on RMR is over and above any change in muscle mass. The increases in RMR above pretraining levels in nondieting individuals could also reflect increased norepinephrine levels ([Brownell et al., 1987](#); [Mole et al., 1989](#); [Nieman et al., 1988](#)).

In those studies showing an additional decline in RMR when exercise was added to caloric restriction, the decline is often explained as part of the body's protective mechanism. If RMR declines when calories are restricted, burning additional calories in activity simply makes matters worse. Therefore, it is logical that the RMR would decrease even further. It has been suggested that this decline occurs in individuals (such as some athletes) who are attempting to maintain body weights below their natural level ([Brownell et al., 1987](#)).

If diet and/or exercise results in a loss of body weight (mass), then a proportional long-term reduction in RMR should be expected. Despite the claims of many overweight individuals to the contrary, overweight or obese people typically have a higher RMR than normalweight individuals of similar age, sex, and height to begin with. With weight loss, this higher RMR should not be expected to be maintained ([Garrow, 1987](#); [Stiegler and Cunliffe, 2006](#); [Wadden et al., 1990](#)).

[Byrne et al. \(2012\)](#) attempted to determine to what extent

changes in RMR and body composition explained the less-than-expected weight loss in obese men and women participating in concurrent (16-week) severe dietary restriction (energy intake = 564–650 kcal·d⁻¹) and exercise training (aerobic plus resistance). The relative energy deficit from baseline was 74–87%. Measured weight loss averaged 67% of the predicted value but ranged from 39% to 94%. Changes in RMR explained on average 67% of the less-than-expected weight loss and variability in the proportion of weight lost as fat mass accounted for 5% more. Only when adjusted for these changes in RMR and fat mass did the actual weight loss attain 90% of predicted values.

Because RMR is highly correlated with or related to FFM, but not entirely dependent on it, it might be expected that resistance training would bring about an increase in RMR. The results of the aforementioned meta-analysis ([MacKenzie-Shalders et al., 2020](#)) seem to support this expectation. An impressive single study for this idea comes from research with males 50–65 years old. After 16 weeks of heavy resistance strength training, these subjects had no change in total body weight but showed a decrease in %BF, an increase in FFM, and an increase in RMR. This increase in RMR remained significant even when expressed per kilogram of FFM. Resting norepinephrine levels also increased. The researchers concluded that the increase in RMR with strength training was only partially due to the increased FFM and may also have been linked to an increase in basal sympathetic nervous system activity ([Pratley et al., 1994](#)). This conclusion reinforces the idea that RMR does not totally depend on muscle mass.

Thermogenesis

Think about this. The temperature is 38°C (100°F) with a relative humidity of 80%. Your apartment air conditioner is not working. You are hungry but cannot afford to eat out. In your food stock are ground beef, red beans and rice, tuna, and the ingredients for a fruit salad and a tossed salad. Which do you select? In all probability, you will pick one or both of the salad options, intuitively choosing a meal you assume will not add to your heat load. Actually, though, following ingestion of any meal, metabolism is elevated.

The production of heat is called **thermogenesis**. Heat production is associated with physical activity/exercise (EAT = exercise-associated thermogenesis) and nonexercise activity thermogenesis (NEAT). Thermogenesis also occurs in response to cold (*cold-induced thermogenesis*). When a mammal is exposed to cold, it first attempts to preserve body heat by vasoconstriction, piloerection, and/or postural changes to decrease exposed body surface area. If that is insufficient, endogenous heat production is increased by *shivering thermogenesis* (nonproductive muscular twitches). After a prolonged period in the cold, shivering ceases but a high metabolic rate continues. This is termed *nonshivering thermogenesis*. Finally, thermogenesis is associated with the ingestion of food (Cannon and Nedergaard, 2011).

Thermogenesis The production of heat.

The increased heat production as a result of ingesting a meal, such as seen in **Figure 8.6**, is called the **thermic effect of a meal (TEM)** or *dietary-induced thermogenesis (DIT)*. TEM has two components: obligatory and facultative. *Obligatory* thermogenesis is due to the energy-requiring processes of digestion, absorption, assimilation, and synthesis of protein, fat, and carbohydrate. However, more energy is expended than can be accounted for by these processes. The extra energy expenditure is the *facultative* portion of TEM. Facultative thermogenesis usually peaks in 30–90 minutes but, depending on the size and content of the meal, may last as long as 4–6 hours. This energy all appears in the form of heat and has been attributed to sympathetic nervous system activity (Acheson et al., 1984; Blanchard, 1982; Dulloo et al., 2004). Convincing evidence is available for sympathetic activation after carbohydrate-rich meals, but similar evidence is currently lacking after protein and fat ingestion (van Baak, 2008). Cumulatively, the energy expenditure associated with the ingestion of all food during a day is called the *thermic effect of feeding (TEF)*. TEF is what is depicted as thermogenesis in **Figure 8.2** as constituting approximately 10% of daily energy expenditure (Blanchard, 1982; van Baak, 2008).

Thermic Effect of a Meal (TEM) The increased heat production as a result of food ingestion.



Figure 8.6 The Thermic Effect of a Meal.

The thermic effect of a meal is dependent upon the macronutrient content and total kilocalories ingested.

Most studies comparing the thermic response of lean and obese individuals to a test meal show a blunted TEM in the obese (Blanchard, 1982; de Jonge and Bray, 2002; Jequier, 1987; Segal et al., 1984, 1985, 1987).

Dietary-induced and nonshivering thermogenesis may occur as a result of the uncoupling of oxidative phosphorylation. That is, energy substrates are oxidized but ATP is not produced. Instead, heat is produced. This process may occur at specific steps in the metabolic pathways (known as substrate or futile cycling) or in brown adipose tissue (BAT) mitochondria (Himms-Hagen, 1984; van Baak, 2008) (see Figure 7.11). The high number of iron-containing mitochondria in these adipose cells is the origin of their brown designation. The energy substrates may be the lipid droplets stored within the BAT cells or from circulating glucose and lipid. Brown adipose cells originate from precursor cells that

can also differentiate to skeletal muscle cells (Sidossis and Kajimura, 2015; Wu et al., 2013). They contain a protein called uncoupling protein 1 (UCP1). In uncoupling, the influx of H^+ ions into the matrix of the mitochondrion from the intermembrane space (Chapter 2, Figure 2.10) bypasses the ATP synthase ball and stalk apparatus in electron transport and instead moves through the UCP1, releasing heat but not producing any ATP (Figure 8.7) (Cohen and Spiegelman, 2015). BAT appears to be under adrenergic sympathetic nervous control (specifically norepinephrine) and leptin may play a role. Increasing insulin levels after eating may be responsible for dietary-induced thermogenesis. Conditions such as cold exposure or overfeeding increase norepinephrine activity in BAT. Adaptation can occur in cold-induced thermogenesis and diet-induced thermogenesis (Lee et al., 2014).

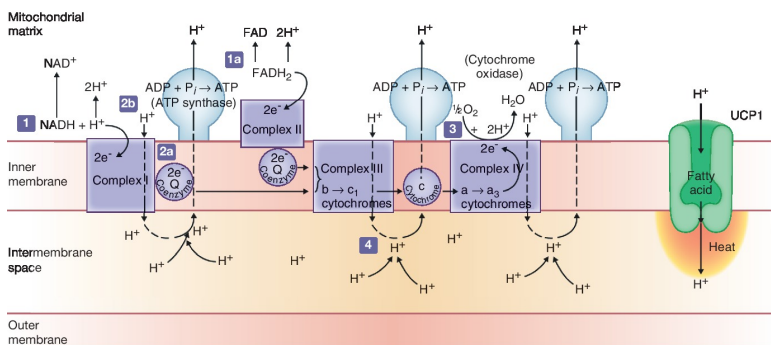


Figure 8.7 Mitochondrial Uncoupling and Thermogenesis.

Uncoupling protein 1 (UCP1) provides an alternative way for hydrogen ions (H^+) to leak across the inner mitochondrial membrane in both brown and beige adipose tissues. Under the influence of norepinephrine fatty acids activate the UCP1, which increases H^+ conductance. This increased leak through the UCP1 re-directs some H^+ away from ATP synthase and the production of ATP. Instead, cellular energy is dissipated as heat. **Source:** Modified from Fuller-Jackson and Henry (2018).

The term **adaptive thermogenesis** is not unequivocally defined but has come to mean any change in heat production or energy expenditure in response to a changing internal or external environment indicating either an increase or decrease in the efficiency of energy utilization. In the context of dietary-induced thermogenesis, an *adaptive increase in thermogenesis* indicates a change in the efficiency of energy utilization resulting in increased heat production preventing the storage of energy. Reduction of adaptive thermogenesis can be interpreted as a defensive, body mass-saving mechanism after underfeeding (Erlanson-Albertsson, 2003; Mozo et al., 2005; Müller and Bosy-Westphal, 2013; Wijers et al., 2008).

Adaptive Thermogenesis Any change in heat production or energy expenditure in response to a changing internal or external environment indicating either an increase or decrease in the efficiency of energy utilization.

Until very recently, it had been thought that only human newborns had any BAT. It has now been demonstrated that substantial amounts of metabolically active BAT are present in healthy adult humans. The main BAT depots are in the neck and shoulder region of the upper torso, along the vertebrae, in the thoracic cavity, and around the renal glands. All individuals do not exhibit defined regions of functionally active BAT. BAT prevalence and activity are higher in young, lean individuals than older, obese individuals (Betz and Enerbäck, 2015; Lee et al., 2013).

To complicate things even more, in 2012, an additional variation of adipocyte cell was identified. It appears that white adipose tissue may be “browned” becoming “*brite*” (brown-in-white) or *beige* adipose tissue. Beige adipose cells (see **Figure 7.11**) arise from the same precursor cells as white adipose cells and reside sporadically with white subcutaneous adipose cells emerging in response to certain environmental cues such as temperature (cold), exercise, endocrine hormones (including GLP-1, **Figure 8.3**), metabolites (including lactate and the ketone beta-hydroxybutyrate), and nutrition. However, they are, like

BAT, thermogenic and utilize substrates to produce heat not ATP. Humans have both classic BAT and beige cells. That is, a large portion of adult human brown fat is not classic BAT but actually beige fat. In adults, the depth of fat in the neck seems to be a determinant of relative amounts of brown versus beige fat; that is, classic BAT is more abundant in deeper neck locations. Because brown fat cells of all types can dissipate stored chemical energy as heat, there is optimism that strategies targeting human BAT/beige fat induction and activation will eventually help to prevent weight gains, assist weight loss, maintain weight, prevent against the complications of obesity such as fatty liver and cardiovascular disease, improve glucose homeostasis in diabetes, and enhance overall metabolic, bone, and skeletal muscle health. Whether these potential therapeutic benefits can be achieved at all and, if so, be achieved by cold exposure or pharmaceuticals remains to be seen (Betz and Enerbäck, 2015; Cohen and Spiegelman, 2015; Carrière et al., 2014; Cypess et al., 2013, 2014; Lee et al., 2013; Loyd and Obici, 2014; Nedergaard and Cannon, 2010, 2014; Richard et al., 2010; Sharp et al., 2012; Sidossis and Kajimura, 2015; Wu et al., 2013; Virtanen et al., 2009).

The relationship between BAT and/or beige fat and dietary-induced thermogenesis in humans has not been firmly established. However, a study involving 19 healthy males participants (11 BAT positive and 8 BAT negative) revealed that after the ingestion of a 500-kcal test meal, postprandial energy expenditure increased more in the BAT+ than the BAT- group (Saito et al., 2011). A rigorously controlled study (Lee et al., 2014) provided evidence linking BAT activity to overnight temperature exposure (1 month each at 24°C vs. 19°C) and its metabolic consequences. In 5 young normal-weight males, after cold acclimation, BAT volume and overall fat metabolic activity increased by 42% and 10%, respectively. Dietary-induced thermogenesis was increased by 32%. These changes were accompanied by enhanced postprandial insulin sensitivity, an increase in thyroid hormone T3-to-T4 ratio, an elevation in adiponectin (a strong insulin sensitizer), and a reciprocal reduction in leptin.

The Impact of Diet on the Thermic Effect of a Meal

Both the total caloric content and the percent composition of a meal impact the thermic effect of the meal. The thermic effect of the macronutrients, as a percentage of their energy content, is 2–3% for fat, 6–8% for carbohydrate, and 25–30% for protein (Jequier, 2002). These differences seem to suggest that a high-protein diet would be valuable for individuals wishing to expend extra calories. Although the thermic effect was not measured directly, when a standard protein diet ($0.8 \text{ g PRO} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$) was compared to a higher protein diet ($1.34 \text{ g PRO} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$) in a hypocaloric state (500 kcal below maintenance calories), data suggested that those who had high dietary adherence (75% adherence) and consumed higher protein lost more weight than those who consumed standard protein (-9.5% vs. -5.8% , respectively) (Campos-Nonato et al., 2017).

If the percentages of protein, fat, and carbohydrate are kept constant and close to those values recommended for a healthy diet, a direct relationship is found between the caloric content of a meal and TEM; that is, higher caloric meals cause a higher thermic effect. As a result, an individual on a restricted caloric diet will burn fewer calories through dietary-induced thermogenesis than when eating larger meals (Belko et al., 1986; Bursztein et al., 1989).

The Impact of Exercise on the Thermic Effect of a Meal

Both meal ingestion and exercise stimulate the sympathetic nervous system and thermogenesis. Because many people wish to maximize energy expenditure, researchers have tried to determine whether a combination of exercise plus a meal in close temporal proximity would potentiate (or increase) the singular effect of either exercise or food. A number of studies have been completed following two basic sequences. After a period of rest, the subjects eat a meal and then exercise. Or conversely, after a period of rest, the subjects exercise and then eat a meal. In both sequences, some studies have shown that TEM was enhanced due to exercise (Belko et al., 1986; Binns et al., 2015; Denzer and

Young, 2003; Segal et al., 1984, 1985, 1987; Zahorska-Markiewicz, 1980), while others have shown that TEM was not enhanced due to exercise (Dallasso and James, 1984; Pacy et al., 1985; Warwick, 2007; Welle, 1984; Willms and Plowman, 1991). The studies have been so diverse in terms of subjects, meal composition and energy value, exercise mode, intensity, and duration, however, that it is impossible to determine a pattern for when the effect is additive and when it is not.

The Impact of Exercise Training on the Thermic Effect of a Meal

Both cross-sectional and longitudinal studies investigating the effects of training have shown that aerobictrained individuals have a larger (Davis et al., 1983; Hill et al., 1984; Lundholm et al., 1986), smaller (LeBlanc et al., 1984a, 1984b; Tremblay et al., 1983, 1985), or similar (Owen et al., 1986; Ratcliff et al., 2011) TEM response. Similarly, studies with resistance trained individuals are inconsistent (Gilbert et al., 1991; Ratcliff et al., 2011; Thyfault et al., 2004). As in the studies dealing with the acute effects of exercise, these studies involve a wide diversity of subjects (especially in training level), meal composition, and meal energy value. The reasons for these inconsistent findings are presently unknown (Sullo et al., 2004).

A different approach has yielded interesting results. It is known that the facultative component of the TEM is brought about, in part, by energy intake sympathetic nervous system activation and β -adrenergic receptor (β -AR) stimulation of cellular energy metabolism. Stob and colleagues (2007) investigated how active and sedentary individuals would respond to the direct excitation of β -AR by a known drug stimulator (isoproterenol). Their results showed that habitually exercising adults demonstrated both increased peripheral β -AR thermogenic responsiveness and enhance TEM. Thus, more studies that combine cellular and whole body testing may be needed to fully clarify the impact of exercise training on the TEM.

Exercise/Activity Energy Expenditure

The last element in the caloric balance equation is the amount of energy expended in manual work or exercise. Restricting calories does not change the energy expended in any activity except as it influences body weight/body composition (Ghiani et al., 2015). However, individuals on calorically restrictive diets may have insufficient energy and may not be able to do as much physical work or exercise (Westerterp, 2003).

By definition, exercise increases energy expenditure and the effects of training are cumulative. Unless efficiency and/or body weight changes, the caloric expenditure of any activity does not change with exercise training.

Table 8.2 summarizes the impact of diet, exercise, and exercise training on all of the elements of the caloric balance equation.

| TABLE 8.2 Impact of Diet, Exercise, and Exercise Training on the Components of the Caloric Balance Equation | | | |
|---|--|--|---|
| Caloric Balance Component | Diet | Exercise Response | Training Adaptation |
| Food ingested (+) | By definition, a reduction occurs | No clearly established effect; appetite may transiently decrease immediately, but not over an entire day | Energy intake may increase to more closely match energy expenditure; when training ceases, food intake spontaneously decreases but does not match the decrease in expenditure |
| BMR and/or RMR (–) | Severe caloric restriction causes a 10–20% decrease; weight cycling does not decrease | Unchanged per se, but metabolic rate postexercise remains elevated | No consistent effect is evident |
| Thermogenesis (TEM) (–) | Decreases because fewer calories are being ingested; dependent on composition of meals | No consistent additive effect in a sequence of either food-exercise or exercise-food | No consistent effect in humans. However, direct cellular stimulation of the TEM mechanism preliminarily indicates increased TEM in habitually exercising individuals |
| Work or exercise expenditure (–) | If calories are insufficient, may voluntarily do less exercise; but no direct effect on caloric cost | By definition, an increase occurs | A cumulative increase occurs; relatively constant per kg BW |

The Effects of Diet, Exercise Training, and Diet Plus Exercise Training on Body Composition and Weight

Many people wish to lose weight strictly for aesthetic reasons (to look better) without caring where this weight comes from. From

a physiological standpoint, however, there are four goals for weight loss:

1. To lose body fat with special consideration to visceral abdominal fat
2. To preserve fat-free mass (FFM)
3. To maintain or improve health
4. To maintain or improve performance in athletes

Many factors influence whether these goals can be and are met, including the following:

1. The initial status of the individual (Is he or she a few pounds overweight or obese?)
2. The type of diet selected (Is the caloric restriction minimal, moderate, severe, or maximal? What percentages of the basic nutrients are included in the diet?)
3. The duration of the weight-reducing program (24–48 hours, 5–20 weeks, or longer)
4. Whether or not exercise training is included as part of the program, and if so, the amount and type of exercise (dynamic aerobic endurance, weight-bearing or non-weight-bearing, or dynamic resistance training)

The [American College of Sports Medicine \(2009\)](#) recommends that adults with a BMI ≥ 25 kg·m⁻² consider reducing their body weight, especially if their waist circumference indicates an excess of visceral abdominal fat. Adults with a BMI of ≥ 30 kg·m⁻² are encouraged to seek weight loss assistance. The Obesity Evaluation and Treatment Expert Committee ([Barlow and Dietz, 2007](#)) recommends that children aged 2–7 years with a BMI in the 85th–94th percentile range according to the CDC growth charts ([Chapter 7](#); [Figure 7.8A and B](#)) and those greater than the 95th percentile without health complications maintain their weight. Weight loss is recommended for children above the 95th percentile with health complications. Children and adolescents over the age of 7 years with a BMI in the 85th–94th percentile range and no health complications are advised to maintain their weight. Weight loss is recommended for children between the

85th and 94th percentile with health complications and for all individuals with a BMI above the 95th percentile. The following discussion is primarily intended for these individuals for whom weight loss is recommended.

Exact responses to all possible combinations of the above factors (initial status of the individual, type of diet selected, duration of the weight loss program, inclusion or exclusion of exercise, type of exercise program) are not available. But some generalizations can be made. Chief among them is that weight reduction requires that energy expenditure exceed dietary energy intake. Excluding surgical and pharmacological manipulations, this means diet restriction, exercise, or a combination of both. Separately and collectively, diet and exercise affect body composition and body weight.

Underlying recommendations for weight loss by diet alone, exercise alone, or a combination of the two is the general guideline that states that a cumulative energy deficit of 3,500 kcal is required to lose 1 lb of body weight. The origin of this guideline can be traced back to a calculation that assumes exclusive loss of adipose tissue consisting of 86–87% fat. One pound of fat (or anything else) is equal to 454 g. Each gram of fat contains 9 kcal of energy, thus $454 \text{ g} \times 9 \text{ kcal} \cdot \text{g}^{-1} = 4,219 \text{ kcal} \times 0.86 = 3,514 \text{ kcal}$ (rounded to 3,500 kcal) per pound. The rest of the adipose cell is fluid and protein. However, when energy intake does not meet energy expenditure requirements, the deficit is made up for by the metabolism not only of body fat but also protein and glycogen, and in the process, large amounts of water are also lost. The metabolizable energy density of glycogen is 1,912 kcal \cdot kcal \cdot lb $^{-1}$ and of protein 2,140 kcal \cdot lb $^{-1}$. Water has no metabolizable energy content. Thus, the 3,500 kcal \cdot lb $^{-1}$ is an oversimplification as an energy deficiency prescription (Hall, 2008).

Furthermore, the 3,500 kcal \cdot lb $^{-1}$ rule implies (1) that loss of weight will be at a constant rate depending only on the magnitude of the energy intake change, (2) that the energy expended by the body remains unchanged during this time, and (3) that the rate of change is the same regardless of an individual's starting point in terms of body adiposity. However, a recent analysis demonstrates that the use of the 3,500 rule

significantly overestimates the size of weight loss in practice. In an analysis of seven studies with 103 participants (sex, 71 M, 32 F; baseline body weight, 82.1 ± 22 kg; baseline caloric intake, $2,876 \pm 484$ kcal·d⁻¹), an average deficit of $1,439 \pm 784$ kcal·d⁻¹, and an average treatment duration of approximately 65 days (range 31–93 days), the majority of participants recorded substantially less weight loss than the amount predicted by the 3,500-kcal rule. The mathematically predicted weight loss was 27.6 ± 16 lb; participants lost 20.1 ± 11.3 lb, that is, 7.4 lb less than expected (Thomas et al., 2013).

In point of fact, the energy expended by the body does not stay constant as an individual loses weight even if the physical activity level remains relatively constant (as was discussed in the section on resting metabolic rate) and weight change varies depending on initial body fat content. People with more initial fat involuntarily allocate a greater proportion of a net energy deficiency toward a loss of body fat than do people with lower initial fat. Lean tissue requires more energy to maintain and thus contributes more to the body's overall energy expenditure (Hall, 2008, 2012; Hall et al., 2011). However, everyone will lose some lean tissue (FFM). The fact that the 3,500-kcal·lb⁻¹ rule does not function in an unwavering assumed linear fashion does not invalidate the caloric balance equation. The caloric balance equation simply functions dynamically as the body adapts to the induced changes of a caloric deficit (or excess). Despite these problems with the 3,500-kcal rule, much of the literature and many of the recommendations for weight control still rely on it and it cannot be totally avoided in the discussions to follow.

Complete the [Check Your Comprehension 1](#) box.

CHECK YOUR COMPREHENSION 1—CASE STUDY 1

Pamela wants to lose 12 lb. To help her, you will calculate the number of calories she should be ingesting each day over a 3-month period and compare this with the theoretical 3,500-kcal·lb⁻¹ “rule.” Check your answer in [Appendix C](#).

Go to <http://bwsimulator.niddk.nih.gov>. This is the web site for the National Institute of Diabetes and Digestive and Kidney Disease

Select Body Weight Planner. Watch the 3-min video. Launch the Body Weight Planner. Enter the following information: WT, 152 lb; sex, female; age, 21 y; height, 5 ft. 6 in.; physical activity level, moderate at work and moderate at leisure (it should come up as 1.8). Select no change in activity. Subtract the calories (kcal) per day to reach your goal from the Calories per day to maintain your current weight. Multiply that by 180 days for the total caloric deficit over the 180 days. Divide the goal weight loss (12 lb) into the total caloric deficit to get $\text{kcal}\cdot\text{lb}^{-1}$ of weight loss. How does this compare with the $3,500\text{-kcal}\cdot\text{lb}^{-1}$ “rule”? If the $3,500\text{-kcal}$ rule were accurate, how many pounds would Pamela expect to lose for this caloric deficit? How does the number of calories ingested after the weight loss needed to maintain the reduced weight compare with the number of calories needed to maintain the before weight?

Repeat the body weight planner calculations, but this time, select and increase in physical activity of medium running $3\times$ ’s per week, $30\text{ min}\cdot\text{d}^{-1}$. What, if anything, does this change in the calculations?

The Effects of Diet on Body Composition and Weight

The vast majority of individuals wanting to lose weight choose to diet. Caloric restriction may be minimal (a deficit of about $250\text{--}500\text{ kcal}\cdot\text{d}^{-1}$), moderate (a total intake of $1,200\text{--}1,500\text{ kcal}\cdot\text{d}^{-1}$), severe (a total intake of $400\text{--}800\text{ kcal}\cdot\text{d}^{-1}$), or maximal (fasting or zero caloric intake). Assuming adherence, all of these will result in a weight loss.

With dietary restriction, the percentages of body fat and fat-free mass (FFM) will vary based on the caloric content of the diet. Thus, body weight loss from total fasting is split equally between body fat (50%) and FFM (50%); from very-low- ($400\text{--}800\text{ kcal}\cdot\text{d}^{-1}$) or low- ($800\text{--}1,200\text{ kcal}\cdot\text{d}^{-1}$) calorie diets, the split is approximately 75% body fat and 25% FFM and from $1,200$ to $1,500\text{ kcal}\cdot\text{d}^{-1}$ diets 90% body fat and only 10% FFM (Nietman, 1990). Fasting, which is obviously incompatible with life over the long haul, is not recommended as a dietary technique. However,

time-restricted eating (a dieting method that falls under the umbrella of intermittent fasting) is a strategy where an individual chooses to eat only during certain time constraints to control caloric intake. Common time-restricted windows per day include 16 hours of fasting with an 8-hour eating period (16:8) or 20 hours of fasting with a 4-hour eating window (20:4). Time-restricted feeding has been shown to reduce overall caloric intake without additional dietary intervention (Moro et al., 2016; Tinsley et al., 2017). Other strategies such as alternate day fasting may have unwanted effects on muscle mass (Templeman et al., 2018). However, it appears that when combined with resistance training, there is no loss in muscle mass over time (Tinsley et al., 2017). Regardless, the data show that these possible benefits to the various fasting protocols are due to the inherent caloric restriction rather than the fasting itself (Moro et al., 2016; Templeman et al., 2018; Tinsley et al., 2017).

Severe low-calorie and very-low-calorie diets (VLCDs) should be undertaken only in extreme situations under the direct supervision of a physician. This technique is typically reserved for extremely obese individuals and is often conducted in a live-in metabolic ward of a hospital. VLCDs result in larger, more rapid reductions in weight, and weight maintenance may be greater with this technique. However, neither the long-term health effects nor the effects of the rate of weight loss on body composition or regional fat loss have been determined (Volek et al., 2005). The 1,200–1,500 kcal·d⁻¹ regimen is often recommended, but even that does not totally preserve lean body mass (ACSM, 1983). Furthermore, it is better to individualize this approach to a reduction in energy intake of 500–1,000 kcal·d⁻¹. With this level of energy deficit, a weight loss of approximately 1–2 lb (0.5–0.9 kg) per week is reasonable but not guaranteed given the discussion on the 3,500-kcal rule above. A faster rate of weight loss or more initial weight loss has not been shown to improve long-term weight loss compared with this conservative approach (Ashtary-Larky et al., 2017). The importance of the FFM loss may depend on how much excess weight the individual started with. Obese individuals have an excess of both body fat and FFM; their excess body weight is composed of 62–78% fat and 22–38% FFM. The excess FFM is necessary to support and move their larger mass. Therefore, as one loses total body weight, less FFM is

needed, and its loss therefore is neither unexpected nor particularly harmful. The loss of some FFM in such an individual may not be critical, but its preservation would also not be detrimental from a health standpoint. Individuals with less weight to lose need to protect their FFM more (Brown et al., 1983; Donnelly et al., 1991; Jequier, 1987; Pacy et al., 1986).

Unfortunately, body weight/composition changes by dietary restriction alone also bring about decreases in resting and nonresting energy expenditure, fat oxidation, thyroid hormones (all of which are associated with increased energy storage), total energy expenditure, and exercise energy expenditure. Factors that increase energy intake, namely, increased ghrelin, and decreased leptin, PYY, insulin, and sympathetic nervous activity also occur (Figure 8.3). All of these may persist for up to a year post weight loss (Washburn et al., 2014).

Recall that FFM is composed of water, protein, and bone mineral (see Figure 7.2 in Chapter 7). Bone loss often accompanies weight loss brought about by caloric restriction alone (McTigue et al., 2006; Villareal et al., 2006). The key to maintaining bone density may be the intake of adequate or higher calcium (Riedt et al., 2007) or exercise to achieve the negative energy balance (Villareal et al., 2006).

The relative proportions of water and protein loss when dieting vary with the duration of the diet. During the first several days of a diet, most weight loss (55–70%) is water loss. But by the end of the first month, this percentage is considerably reduced (to ~40%), and after 7–8 months, it may be as low as 5%. The protein (primarily the component of muscle tissue) loss is a consistent 5% in a nonexercising dieter (Grande et al., 1958a; Heymsfield et al., 1989). What does this say about the proportion of fat loss? Simply that the percentage of fat loss is a mirror image of the percentage of water loss: little fat is lost at first and then progressively more fat is lost as caloric restriction continues. In terms of the 3,500 kcal/lb–1, because body water has no metabolizable energy content, early weight changes have an energy density substantially lower than this value (Hall, 2012).

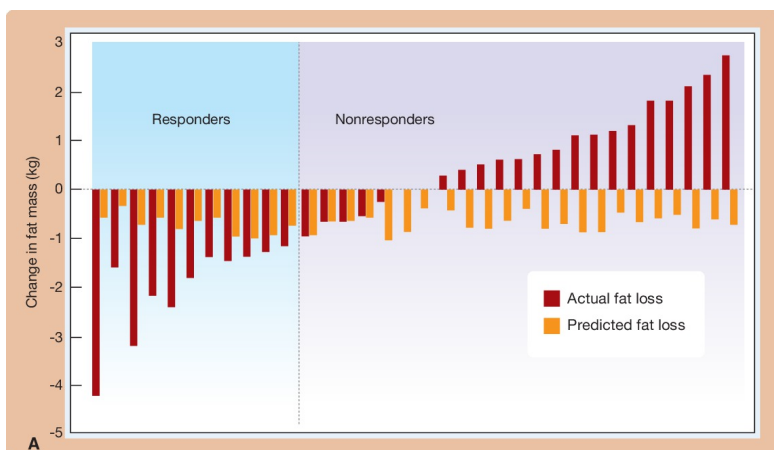
Strangely enough, restricting water intake while dieting causes a higher proportion of water to be lost, not less, which could lead to dehydration. More total weight is lost if water is

restricted, but not more fat. Water intake should not be restricted when dieting ([Grande et al., 1958b](#); [Heymsfield et al., 1989](#)).

The Effects of Exercise Training on Body Composition and Weight

Little evidence suggests that exercise alone produces weight losses as great as what can be achieved with dietary modification alone. Results from short-term studies lasting ≤ 16 weeks have shown that increased physical activity is positively associated with a reduction in total fat in a dose-response relationship; that is, the more calories expended in exercise, the greater the fat loss. In long-term studies (≥ 26 weeks), however, no dose-response is seen. On average, the weight loss attained in short-term studies is approximately 85% of that expected (based on the 3,500 rule of caloric expenditure) and is composed almost entirely of fat. Research evidence does not support resistance exercise as effective for weight loss with or without dietary restriction ([ACSM, 2009](#)). A 2014 metaanalysis ([Washburn et al., 2014](#)) found greater long-term weight losses with diet compared to exercise (only $\sim 3\%$) but attributed this to insufficient energy expenditure in the exercise trials. They concluded that "... clinically significant weight loss can be achieved with aerobic exercise alone when exercise energy deficits are similar in magnitude to those induced by energy restriction" (p. 30). The failure of exercise in some studies to produce weight losses similar to those induced by dietary control may also result from individuals compensating for the increased energy expenditure during exercise by decreasing energy expenditure the rest of the day (sometimes called nonexercise activity thermogenesis or NEAT), which again results in a lower-energy deficit than intended. The study presented in [Figure 8.8](#) reveals that NEAT changes actually do occur. Thirty-four overweight or obese females (age, 32 years; BMI, $29.3 \text{ kg}\cdot\text{m}^{-2}$) exercised for $150 \text{ min}\cdot\text{wk}^{-1}$ for 8 weeks expending a *net* total of $30.2 \pm 12.6 \text{ MJ}$ ($7,724.7 \pm 3,014.3 \text{ kcal}$) for a predicted fat loss of $0.8 \pm 0.2 \text{ kg}$ ($1.76 \pm 0.44 \text{ lb}$). Food intake was not controlled. For the group as a whole, no change in body fat mass occurred. However, there was a large individual variation. The range of fat loss can be seen

in **Figure 8.8A**. The 11 individuals who achieved more than the predicted fat loss were classified as “responders,” and the 23 individuals who achieved less than the predicted fat loss were classified as “nonresponders.” There were no significant differences in the energy intake or macronutrient percentages between responders and nonresponders. Responders increased their daily caloric intake by 0.86 ± 0.75 MJ (206 ± 179.4 kcal) and nonresponders by 1.03 ± 0.53 MJ (246 ± 126.8 kcal) per day. **Figure 8.8B** shows the changes in daily energy expended by responders and nonresponders categorized as total energy expenditure (TEE), activity energy expenditure (AEE, calculated as the energy expenditure of all active activities except the structured exercise sessions), sedentary energy expenditure (SEDEE), and sleeping energy expenditure (SEE). The only significant change was in the AEE with responders showing an increase of 0.79 ± 0.5 MJ (+189 kcal) and the nonresponders a decrease of 0.62 ± 0.39 MJ (−148.3 kcal). Given that both groups expended approximately the same amount of energy during exercise [28.55 MJ \pm 2.14 ($6,830 \pm 523$ kcal) responders; 30.29 ± 1.76 ($7,246.3 \pm 421$ kcal) nonresponders] and increased their energy intake approximately equally, these data indicate that the lower-than-predicted fat mass loss in the “nonresponders” can be attributed, to some extent, to a compensatory reduction in physical activity outside the supervised exercise sessions.



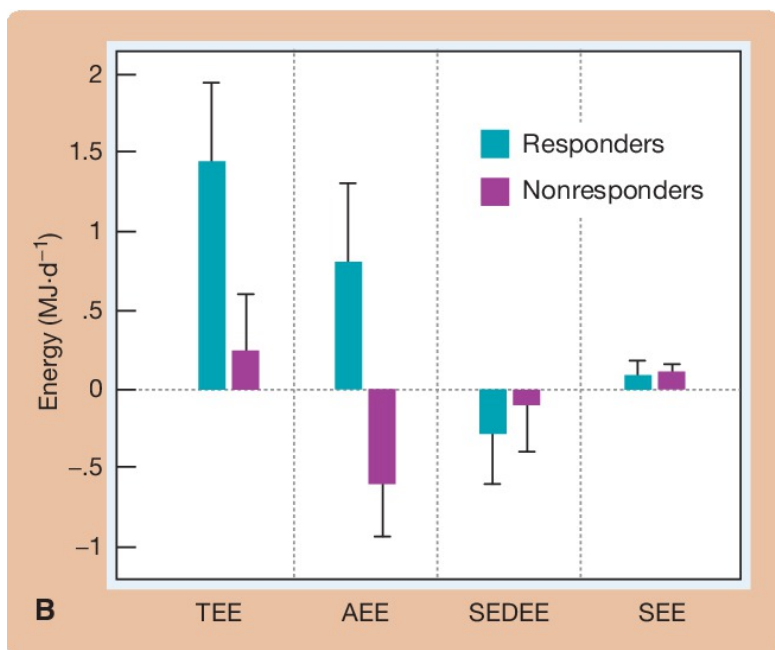


Figure 8.8 Behavioral Compensation during Exercise Training and Individual Fat Loss.

Graph A compares the predicted and actual changes in body fat mass in individual overweight females who participated in an 8-week exercise training program. Few lost the calculated amount and as a group were divided in “responders” (those that actually lost fat) and “nonresponders” (those who did not lose fat). **Graph B** shows that the nonresponders compensated for the energy expended during the exercise training by significantly decreasing all other active energy expenditure (AEE) during the day. TEE, total energy expenditure; AEE, active energy expenditure excluding exercise expenditure; SEDEE, sedentary energy expenditure; SEE, sleeping energy expenditure. **Source:** Reprinted with permission from Manthou, E., J. M. R. Gill, A. Wright, & D. Malkova: Behavioral compensatory adjustments to exercise training in overweight women. *Medicine & Science in Sports & Exercise*. 42(6): 1221–1228 (2010). Copyright ©2010 The American

When the energy deficit is held constant and other factors affecting energy balance are controlled, dynamic aerobic exercise can induce meaningful weight loss. Increases of less than 150 min·wk⁻¹ of exercise result in minimal weight loss. Exercise increases between 150 and 225 min·wk⁻¹ result in modest weight loss (~2–3 kg/4–7 lb), whereas 225–420 min·wk⁻¹ result in 5–7.5 kg (~11–17 lb) weight loss over months to years of activity. Obviously, there is a dose-response relationship operating in that higher doses can potentially provide larger losses from initial weight (ACSM, 2009; Hansen et al., 2007; Ross and Janssen, 2001).

The Influence of Sex on Body Composition and Weight Changes

When energy expenditure is equal (in supervised studies where exercise energy expenditure is substantial), weight and fat loss are likely to be equal regardless of sex. There is some evidence (although not overwhelming) that suggests that lean females do not lose as much weight in response to exercise as lean males. However, in overweight/obese individuals, any differences in weight loss can be attributed to greater exercise-induced energy expenditure in the males rather than an increased compensatory energy intake in females regardless of the time span of the exercise training. When the exercise intervention is based on intensity and duration, larger individuals (typically males) will expend more energy. When the energy expenditure is controlled, measured, and the same for males and females, then similar changes in body weight and body fat are observed. There is, of course, a large individual variability in response to the same dose of exercise within the sexes so any given male or any given female may lose more or less weight or %BF than any other male or female (Caudwell et al., 2014). **Figure 8.9A** shows the changes in anthropometric variable in 107 overweight and obese males and premenopausal females as the result of a 12-week aerobic exercise program in which the intensity was set at 70% HR_{max} and the duration individualized so that each participant expended

500 kcal each exercise session, 5 days·wk⁻¹. The intervention resulted in significant reductions in BMI, total body mass (BM), fat mass (FM), and waist circumference (WC). The mean reduction in %FM (−2.45%) was identical between males and females. A significant increase in absolute fat-free mass (FFM) was observed in the females but not males. However, when FFM was expressed as a percentage of BM, a significant and similar percentage of increase occurred (2.47% M; 2.50% F). **Panel 8.9B** shows the extreme interindividual variation in FM and FFM (Caudwell et al., 2013).

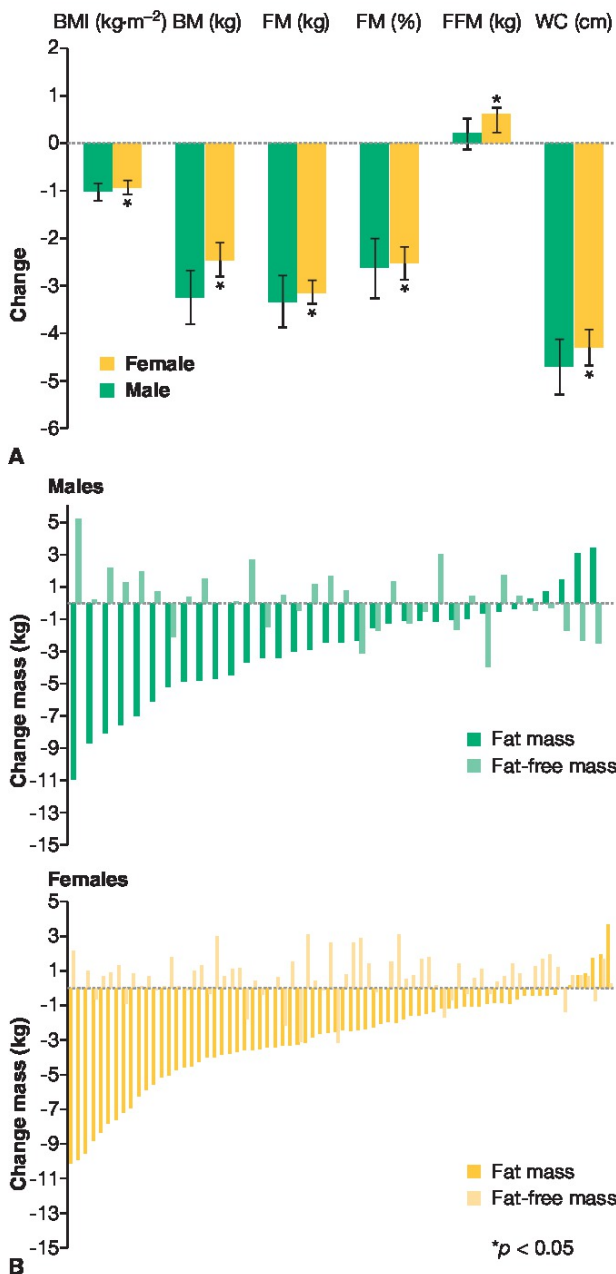


Figure 8.9 Body Composition and Anthropometric Changes in Response to Aerobic Exercise Training in Overweight/Obese Males and Females.

Panel A indicates that significant changes occurred in both male and female overweight/obese individuals as a result of 12 weeks of aerobic training set at an intensity of 70% HRmax and an individualized duration to burn 500 kcal per session, 5 sessions per week. **Panel B** shows the individual results for males (*upper panel*) and females (*lower panel*). There is a wide variation between individual in both sexes despite no significant mean difference in either fat mass or percent fat-free mass changes. **Source:** Reprinted with permission from Caudwell, P., C. Gibbons, M. Hopkins, N. King, G. Finlayson, & J. Blundell: No sex difference in body fat in response to supervised and measured exercise. *Medicine & Science in Sports & Exercise*. 45(2):351–358 (2013). Copyright ©2013 The American College of Sports Medicine.

Realistically, few people wishing to lose body weight set out to do it entirely by exercising. You may encounter some of these people in health clubs, but they are a minority. On the other hand, there are many individuals who consciously or unconsciously do control their body weight through exercise training. These are, of course, athletes. If we ignore the fact that some athletes also closely watch their caloric intake, indirect evidence of the effect of exercise on body composition can be inferred from the %BF values measured in various groups of athletes. **Figure 8.10** presents a sampling of sports.

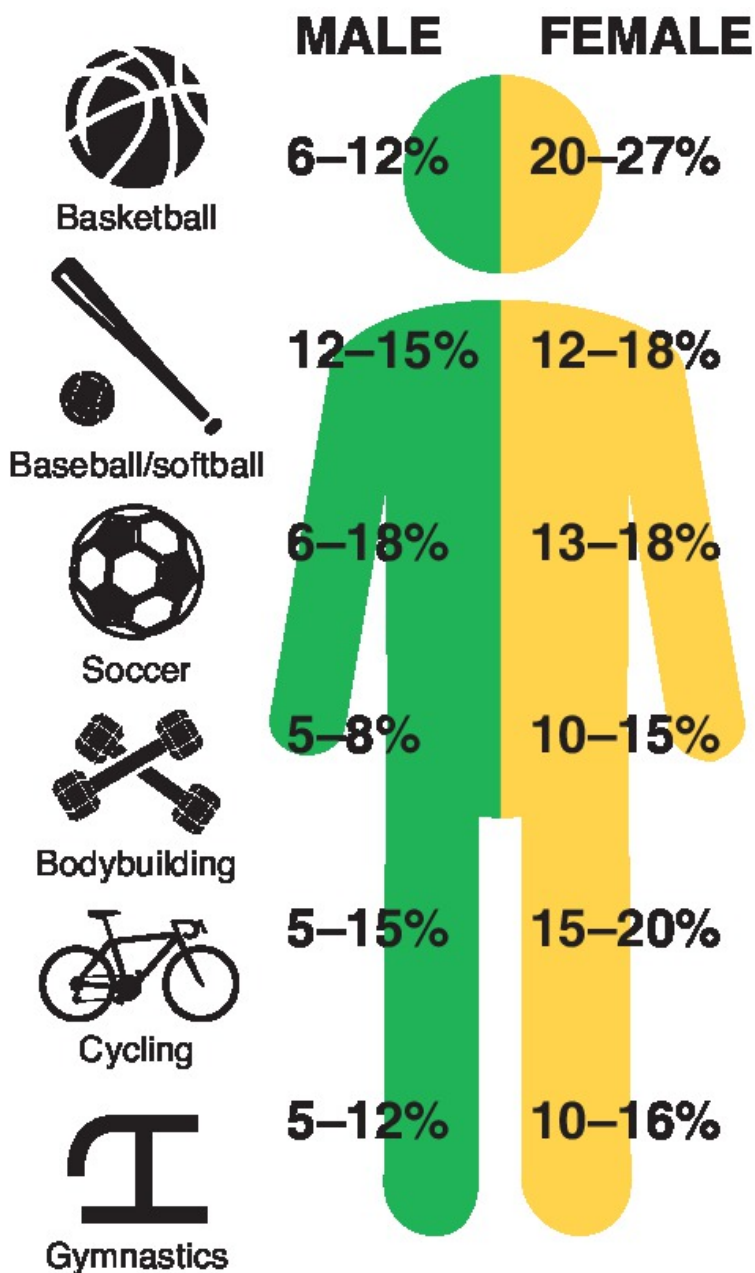


Figure 8.10 Average Percent Body Fat of Male and Female Athletes (17–35 Years) in Selected Sports.

Source: Wilmore and Costill (1988).

Keeping in mind that the %BF for an average young adult male should be 12–15% and that for a young adult female it is 22–25%, several conclusions can be made from this graph. First, even among athletes, the malefemale difference in %BF is maintained. Second, male and female athletes in different sports vary considerably in their %BF, from much leaner than average to slightly above average. Third, the %BF values appear to have a direct relationship to the demands of the sport. Athletes in sports where body aesthetics play a part in success (bodybuilding and gymnastics) tend to have low %BF values. Endurance athletes (cyclists) also tend to have low %BF values. Athletes in predominantly motor skill sports (baseball) tend to be about average.

These data tend to support the value of exercise training in maintaining a low %BF, because athletes in activities known to have high caloric costs and engaged in for long periods of time have the lowest %BF. However, the problem of self-selection is present in studies of this type; that is, we do not know whether individuals who are genetically programmed for leanness and success in these sports naturally gravitate to them or whether the training demands of the sport determine the body composition of the performer. Both factors are probably operating.

Although the mean values show a definite pattern, a wide range of variability exists among successful athletes in any given event. For example, successful female distance runners have an average-measured %BF value of 16.5% (Wilmore and Costill, 1988). Included in that group, however, was an athlete who won six consecutive international cross-country championships and another who held the 1972 world best time for the marathon; both of these runners had only 6% BF. At the other end of the range, the mid-1970s world record holder for the 50-mi run tested at 35.8% BF (Brownell et al., 1987). These extremes point out the importance of not establishing a specific %BF value for all athletes in any sport. The average values should not be interpreted as optimal values for all athletes competing in a particular sport. If %BF values are suggested to athletes, they should be in the form of a range of values. The health and the performance of the athlete need to be monitored within that

range, and individual adjustments are made based on this monitoring. Eating disorders, which are sometimes associated with attempts to control body composition for sport, are discussed fully in [Chapter 6](#).

It must be emphasized that the key to weight and fat loss is not exercise per se but the achievement of a sufficient caloric deficit through exercise ([ACSM, 2009](#); [Kim, 2021](#)). Diet alone can also achieve a caloric deficit, but when weight is lost through dieting alone, it includes a greater percentage of FFM. Dynamic aerobic endurance activity is most helpful for increasing caloric expenditure; dynamic resistance activity (which is low in caloric cost) acts more directly to increase muscle mass. There seems to be no direct influence of training intensity on fat mass loss; however, there is a volume effect in short-term studies, as more exercise equates to greater caloric expenditure ([Hansen et al., 2007](#)).

A 2011 meta-analysis ([Thorogood et al., 2011](#)) of 28 studies found that aerobic exercise alone resulted in a moderate reduction of weight and waist circumference but was not an effective strategy for reversing obesity in the absence of dietary intervention. Conversely, resistance exercise had little effect on weight loss but increased FFM by approximately 2–5 lb (1–2 kg). More importantly, recent evidence has shown that overweight/obese individuals benefit from exercise even if they remain overweight/obese. Indeed, it has even been suggested that dietary interventions in the treatment of obesity in adults must be supplemented by exercise training not in anticipation of additional weight loss but to induce clinically relevant changes in cardiovascular and metabolic risk factors, visceral adipose tissue mass, physical fitness including muscle mass, and quality of life ([Verboten and Hanses, 2021](#)). The Focus on Application box in [Chapter 7](#) discusses some of this evidence from dynamic aerobic exercise. Active overweight/obese persons have better risk profiles for the diseases identified earlier and lower rates of morbidity and mortality than their sedentary counterparts. These health gains are related to the beneficial changes in glucose-insulin responses, lipoprotein values, and cardiovascular function. There is also some evidence that resistance training improves high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), insulin, and blood pressure risk

factors. Thus, the value of exercise training for overweight individuals goes far beyond the sometimes minor contribution to weight loss (ACSM, 2009).

The Effects of Exercise Training on Body Composition and Weight change in Children and Adolescents

Children and adolescents appear similar to adults in relation to the influence of exercise training on body composition. Comparisons of young athletes with their sedentary counterparts typically show that both boy and girl athletes have a lower percent body fat and higher FFM values. The variation among sports is similar to that for adults. Training studies also show that a decrement in percent body fat results from systematically applied exercise programs (Boileau et al., 1985; Epstein and Goldfield, 1999; Plowman, 1989). Two separate systematic reviews (Atlantis et al., 2006; Watts et al., 2005) both concluded that exercise training does not consistently decrease body weight in children and adolescents but that exercise is associated with beneficial changes in fat (decreases) and fat-free mass (increases). The National Heart, Lung, and Blood Institute's Growth and Health Study reported that at ages 18–19 years, BMI was 2.10 kg·m⁻² less for active white girls and 2.98 kg·m⁻² less for active black girls than their sedentary counterparts. Similarly, the sum of skinfolds was 15.04 and 13.54 mm less for the active white and black girls, respectively, than their inactive counterparts. Active girls were doing only the equivalent of 30 minutes of brisk walking 5 d·wk⁻¹. The change in reported energy intake ranged from 17 to 121 kcal·d⁻¹ (Kimm et al., 2005).

The Effects of Exercise Training on Body Composition and Weight Change in Older Adults

It is often questioned whether older adults benefit from exercise training as much as younger individuals for body composition and weight change. Several studies reported that exercise can help reduce bodyweight and improve body composition in older

adults. One study, using individuals that were 65–70 years old, demonstrated that a 12-week resistance training program reduced body fat percentage and increased muscle mass compared to the control group (Marcos-Pardo et al., 2018). Data suggest that both aerobic and resistance training are equally effective for weight loss in older adults; however, resistance training or a combination of resistance training and aerobic training seem to be more beneficial than aerobic training alone to maintain muscle mass while undergoing weight loss (Villareal et al., 2017). It is clear that exercise provides significant health benefits, weight loss and/or body composition improvements at any age.

FOCUS ON RESEARCH | *Clinically Relevant*

The Effects of Resistance Training on Weight Loss, Body Composition, and Energy Expenditure during Caloric Restriction

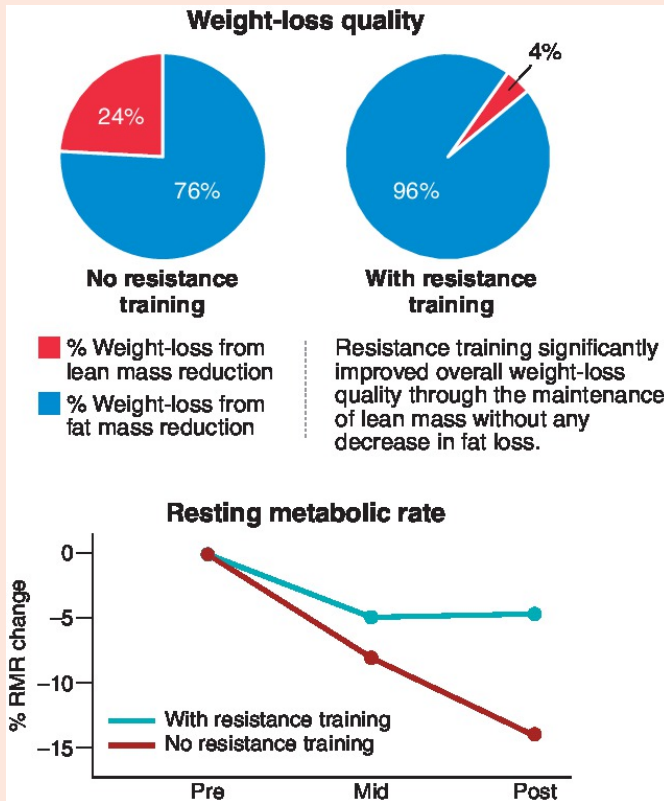
The manipulation of an individual's caloric intake is a common approach used for weight loss. Given the need to create a consistent caloric deficit to successfully lose weight, decreasing caloric intake is a valid approach for weight loss. However, when individuals attempt to lose weight, the main goal is to lose fat mass and not lean mass. Using dietary interventions without the addition of exercise can result in weight loss; however, a larger percentage of the total weight loss may be from lean mass rather than fat mass compared to those who also include exercise as part of their weight loss approach.

A clinical study evaluated the effects of resistance training on enhancing body composition and functional outcomes in obese individuals seeking bariatric surgery who were undergoing a 12-week very low-calorie diet (VLCD). Participants in this study were morbidly obese, meaning they had a BMI greater than $35 \text{ kg}\cdot\text{m}^{-2}$. The medically prescribed dietary intervention consisted of a caloric intake of 1,200

kcal·d⁻¹. All participants consumed the same diet and participated in a walking-based exercise program. However, half the participants performed resistance training (RT; n = 6) three times weekly for a total of 12 weeks, while the other half did not (control; n = 5).

The results indicated that both groups lost significant amounts of total body mass (control: 19.4 ± 2.3 kg, vs. RT: 15.8 ± 1.5 kg) and fat mass (control: 14.7 ± 1.8 kg, vs. RT: 15.1 ± 2.1 kg) with no differences between groups. Thus far, the results indicate that both treatments were effective for weight loss with no benefits of one over the other. However, resistance training mitigated any losses in lean mass associated with caloric restriction (control: -4.6 ± 0.8 kg). The proportion of weight loss from fat mass (CON: $75.6 \pm 3.4\%$ vs. RT: $96.0 \pm 6.0\%$) and lean mass (CON: $24.4 \pm 3.2\%$ vs. RT: $4.0 \pm 6.5\%$) were also significantly different between the groups. When looking at differences in fat and lean mass, the study demonstrates that resistance training provides favorable outcomes for body composition during caloric restriction in terms of retaining muscle despite losing large amounts of body weight.

These changes in body composition also resulted in differences in resting energy expenditure (REE). The control group decrease REE by 328.6 ± 72.7 kcal·d⁻¹ ($14.3 \pm 2.4\%$), while there were no significant decreases in the RT group. RT also improved measures of strength and function compared to control, which is an expected result since RT is well-known to improve these measures. Overall, these findings suggest that including RT during caloric restriction can help improve body composition and may contribute to longterm weight loss success by mitigating decreases in daily energy expenditure. A higher energy expenditure will allow individuals to consume more calories throughout the day while maintaining caloric balance.



Source: Jo, E., P. R. Worts, M. L. Elam, A. F. Brown, A. V. Khamoui, D. H. Kim, M. C. Yeh, M. J. Ormsbee, C. M. Prado, A. Cain, K. Snyder, & J. S. Kim: Resistance training during a 12-week protein supplemented VLCD treatment enhances weight-loss outcomes in obese patients. *Clinical Nutrition*. 38(1):372–382 (2019).

FOCUS ON APPLICATION

Guidelines for Evaluating a Weight Loss Diet

Throughout this chapter, guidelines are presented for developing and evaluating exercise programs through the use of the training principles. Because the most effective technique for weight loss and control is a combination of diet and exercise, the following guidelines can assist in the development and evaluation of diets intended for weight loss. An acceptable diet should meet the following standards (ACSM, 2000, 2007; [Dietary Guidelines for Americans, 2020](#); [van Horn et al., 1998](#)). The diet should:

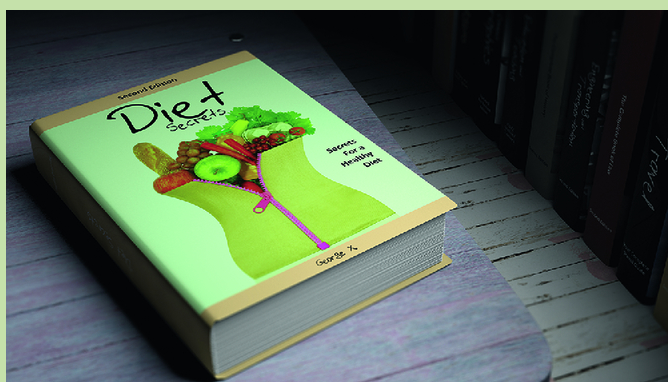
1. Provide a daily energy intake of approximately 500–1,000 kcal below normal prediet intake, but not lower than RMR ($\sim 1,200 \text{ kcal} \cdot \text{d}^{-1}$ for females and $1,500 \text{ kcal} \cdot \text{d}^{-1}$ for males), or the total calories halfway between RMR and 30% above RMR.
2. Meet the nutritional requirements of 45–65% carbohydrate, 10–35% protein, and 20–35% total fat ($<10\%$ saturated, trans fats as low as possible) and the dietary reference intakes/recommended daily allowance for vitamins and minerals.
3. Emphasize a variety of food choices and allow for individual and cultural preferences in terms of acquisition, taste, preparation, and cost.
4. Not be based on some “secret” ingredient or “magic” combination of foods or require the simultaneous ingestion of “fat burning” or other supplements. Taking a daily multivitamin may be acceptable.
5. Be backed by credible scientific or medical organizations (e.g., the American Dietetic Association or the American Heart Association) and/or well-designed research published in peer-reviewed journals that supports both the effectiveness and safety of the diet. Anyone may write a diet book; the publication of diet books is not regulated by any governmental agency or professional society. If someone intends to make money with the diet, “let the buyer beware.”
6. Include at least three meals per day (more frequent smaller meals are also acceptable) and 74–100 fluid oz of drinking water and other beverages per day. Alcohol

intake should be controlled.

What about the manipulation of macronutrients in diets? Claims that the ingestion of a low-glycemic index diet (as opposed to a high-glycemic index diet) will increase the weight loss have generally not been supported. Calorie-restricted diets differing substantially in glycemic load can result in comparable long-term weight loss. If anything, a reduced calorie intake may be harder to sustain on a low-glycemic diet over time (Das et al., 2007). Low-carbohydrate/high-fat diets and high-protein diets can be successful for weight loss but are probably based on water loss, ketosis-induced appetite suppression, and high satiety. Any beneficial impact appears to result from reducing caloric intake, rather than from reduced carbohydrate intake (Strasser et al., 2007). Similarly, high-carbohydrate diets have been shown to have unfavorable effects on blood lipid levels (Volek et al., 2005). Moderate levels of fat (20% to <30%) as recommended by American College of Sports Medicine (2001) often represent a decrease in fat intake in overweight/obese individuals. However, it is probably not fat restriction per se that results in weight loss but the decrease in total caloric intake (Volek et al., 2005). Fat restriction avoids the increase in ghrelin caused by dietary energy restriction (Weigle et al., 2003). In addition, a fat intake less than 30% could have beneficial health effects as well that result from the fat restriction per se. Some theoretical and experimental evidence suggests that diets high in protein are beneficial for weight loss and weight maintenance (ACSM, 2001; Arciero et al., 2014; Kim et al., 2016; Layman, 2004; Volek et al., 2005; Westerterp-Plantenga et al., 2012). A high-protein diet can be defined as 18–30% or a ratio of 1 g PRO/1.5 g CHO. A high-protein, moderate-fat, moderate-carbohydrate diet has been shown to be better than a low-protein, moderate-fat, high-carbohydrate diet of equal caloric restriction either alone or when combined with an aerobic plus resistance exercise program for promoting weight loss (Meckling and Sherfey, 2007). Similarly, a high-protein diet has been shown to maintain weight after weight loss with or without controlling for the percentage of carbohydrate in the diet (Moon and Koh, 2020;

Weigle et al., 2005; Westerterp-Plantenga et al., 2004). Finally, a high-protein diet was shown to maintain FFM, even when weight was regained (Leidy et al., 2007a; Westerterp-Plantenga et al., 2004). The benefits of high-protein diets have been attributed in these experimental studies to decreased food efficiency, decreased ghrelin rise, increased leptin sensitivity, increased thermogenic effects of feeding, and increased satiety. A study (Dansinger et al., 2005) of four popular diets (Atkins, carbohydrate restriction; Zone, 40–30–30 macronutrient balance; Weight Watchers [caloric restriction]; Ornish, fat restriction) found that after a year, each diet modestly reduced body weight by approximately 2–3 kg (~4–7 lb) and improved several cardiac risk factors. The amount of weight lost was related to adherence to the diet but not to the diet type.

Above all, it must be remembered that the macronutrient content of any diet will affect body weight only when there is a reduction in caloric intake that results in a negative caloric balance. The health costs or benefits of any diet must also be considered. The optimal levels of each macronutrient beyond those established in the current dietary guidelines, if optimal levels actually exist, remain to be determined.



Sources: American College of Sports Medicine (2000, 2001, 2007); Arciero et al. (2014); Astrup et al. (2004); Dansinger et al. (2005); Das et al. (2007); Dietary Guidelines for Americans (2020); Kim et al. (2016); Layman (2004); Leidy

et al. (2007a); Meckling and Sherfey (2007); Strasser et al., (2007); Moon and Koh (2020); van Horn et al. (1998); Volek et al. (2005); Weigle et al. (2003); Westerterp-Plantenga et al. (2004, 2012).

The Effects of Diet Plus Exercise Training on Body Composition and Weight

Studies that have combined diet and exercise seem to suggest that this approach is best for positive, long-term changes (Washburn et al., 2014). Body weight is not lost faster in combined exercise plus diet programs than in diet alone programs because, as has been said several times, weight loss is all about achieving a negative caloric balance and changes in fat mass exceeding changes in FFM (Hansen et al., 2007; Stiegler and Cunliffe, 2006; Wing, 1999).

Exercise training, whether alone or in combination with dietary restriction, achieves the desirable maintenance of FFM (Stiegler and Cunliffe, 2006). In some cases, muscle mass can actually be gained, despite very similar total weight loss (See “Clinically Relevant” Box) (Caudwell et al., 2013; Zuti and Golding, 1976). Furthermore, the addition of an exercise component to a weight control program carries several other benefits. These include improved dietary compliance, the maintenance of resting metabolic rate, the numerous fitness benefits mentioned throughout this chapter, and the possibility of more effective maintenance of weight loss (discussed later in this chapter) (Hansen et al., 2007).

The key to weight loss while maintaining FFM is probably the total caloric deficit, the inclusion of resistance training, and the nutrient content (PRO and CHO) of the calories ingested (ACSM, 2009; Walberg, 1989). One advantage of adding exercise to a weight loss regimen may simply be that it allows an individual to ingest more kilocalories and still be in a negative caloric balance. Eating a moderately restricted diet allows for adequate nutrition and lifestyle changes that can be tolerated for a lifetime; by contrast, a VLCD can seldom be sustained for a prolonged time. Furthermore, physical activity will increase weight loss if dietary

restriction is moderate, but not if severe, that is, less than the number of calories equal to RMR ([ACSM, 2009](#)).

The Effects of Diet, Exercise Training, and Diet Plus Exercise Training on Abdominal Obesity

Of special concern is the impact of diet, exercise training, and diet plus exercise training on abdominal fat, both subcutaneous abdominal adipose tissue (SAAT) and visceral adipose tissue (VAT), also known as visceral abdominal adipose tissue, as illustrated in **Figure 8.11**. The concern is that abdominal fat is known to be an independent predictor of the metabolic risk factors that are the antecedents for type II (non-insulin-dependent) diabetes, metabolic syndrome, and various cardiovascular diseases.



Figure 8.11 Abdominal Obesity.

While many individuals are concerned with scale weight, the amount of visceral abdominal fat is more important for

health.

A review of studies investigating the impact of diet alone on VAT found that for every kilogram (2.2 lb) of weight loss, VAT was reduced 3–4 cm², or approximately 2–3% of the total VAT. Furthermore, there appeared to be a preferential reduction in VAT (which is desirable) over subcutaneous fat loss (Ross, 1997). The degree of caloric restriction (low-calorie or very-low-calorie diet) does not appear to have any influence on the ratio of the % change in VAT to the % change in total fat. Individuals with the most VAT lose the greatest amount of VAT (Smith and Zachwieja, 1999). The macronutrient content of the diet does not appear to influence the amount of visceral fat lost (de Souza et al., 2012).

Physical activity with or without weight loss is associated with reductions in visceral and abdominal subcutaneous fat loss when measured by techniques such as MRI or computerized tomography (CT) scan (Ross and Janssen, 1999) in both male and female adults and children/adolescents (Jung et al., 2019; Kim, 2018; Maillard et al., 2018). The fact that changes in SAAT and VAT can occur without changes in body weight is important because it means that individuals using exercise to lose weight need to be informed of this possibility and appropriate measures taken so that they can see that positive changes are taking place despite what the scale may indicate. On the other hand, both total and abdominal fat are generally much more reduced when weight is lost. Limited evidence from randomized trials shows that reductions occur in abdominal (SAAT and VAT) fat when moderate- to high-intensity aerobic exercise interventions of at least 8 weeks' duration are undertaken by middle- to older-aged, overweight or obese males and females (Kay and Fiatarone Singh, 2006; Ross and Janssen, 1999).

A systematic review of clinical trials (Ohkawara et al., 2007) reinforced the conclusions that in obese adults with no metabolic disorders, there is (1) a significant relationship between aerobic exercise training and visceral fat reduction and (2) that although visceral fat reduction is significantly related to weight reduction during the aerobic exercise training, a significant reduction in visceral fat can occur without significant weight loss. Moreover, there is a dose-response relationship between the amount of

aerobic exercise and the amount of visceral fat reduction. At least 10 MET-hr⁻¹wk⁻¹ of aerobic activity appears to be the threshold to achieve visceral fat reduction. Brisk walking at 3.0 mi·hr⁻¹ represents 3.3 METs. Thus, it would take roughly 182 minutes of walking at this pace to achieve the threshold—very comparable to current exercise recommendations ([ACSM, 2009](#)).

Although some evidence shows that the average % change in VAT to the % change in fat ratio is higher as a result of exercise training than caloric restriction (CR) alone, it is insufficient to regard exercise training as a more specific therapy for visceral fat loss than dietary restriction ([Smith and Zachwieja, 1999](#)). This is shown by a randomized controlled trial conducted on 48 healthy males and females with an initial BMI of 27.3 kg·m⁻². Participants were assigned either to a 20% CR diet, an exercise program (EX) designed to produce a similar energy deficit, or healthy lifestyle control group (HL). After 1 year, weight changes corresponding to -10.7%, -8.4%, and -1.7% occurred in the CR, EX, and HL groups, respectively. Whole-body fat mass as well as visceral and SAAT decreased significantly and comparably in the CR and EX group but did not change in the HL group ([Racette et al., 2006](#)).

The benefits of strength training on VAT reduction are unclear. A 2013 meta-analysis that included only 1 strength training study and 2 studies that combined strength and aerobic training versus 12 strictly aerobic training studies found that aerobic exercise of moderate to vigorous intensity had a greater effect on VAT than low-intensity aerobic exercise or resistance strength training in adults. The authors concluded that combining aerobic training with resistance strength training did not appear to result in a greater decrease of visceral adipose tissue than aerobic training alone ([Visseers et al., 2013](#)). However, the Health Professionals Follow-up Study ([Mekary et al., 2015](#)) that has tracked 10,500 healthy males since 1996 found that less age-associated waist circumference increase was seen in those who had a 20 min·d⁻¹ increase in weight training than a similar amount of moderate to vigorous aerobic activity. Finally, two additional studies, these involving obese adolescents, support the importance of weight training. In the first study, it was determined that strength training alone was as effective in reducing VAT as aerobic training or aerobic plus resistance

training (Alberga et al., 2015); in the second, it was found that aerobic plus resistance training was more effective than aerobic training alone (Damaso et al., 2014). Obviously, more research is needed here to determine the precise role of resistance training in VAT reduction. Despite an overall paucity of research, interactions between exercise training/physical fitness and abdominal adiposity seem to extend to children and adolescents as exemplified by the two studies reported above. Additionally, a cross-sectional study of 9- and 15-year-old students has shown that cardiovascular fitness is inversely associated with abdominal adiposity independently of the time spent at different intensities of physical activity and total physical activity. However, in the low-fit group, time spent in vigorous physical activity was related to lower abdominal adiposity. Suggested cutoff values for the inclusion of vigorous physical activity are ≥ 40 and ≥ 25 minutes for boys and girls and ≥ 20 and ≥ 15 minutes for adolescent males and females (Ortega et al., 2007, 2010). The Focus on Application: Clinically Relevant box shows that exercise training can reduce subcutaneous adipose tissue and retard visceral adipose tissue increase in children (Owens et al., 1999).

The few studies that have investigated the impact of diet plus exercise training on VAT have shown that the relative reduction in VAT is similar to the response induced by diet alone; that is, the addition of exercise training did not provide any preferential benefit for the reduction of VAT (Fayh et al., 2013; Ross, 1997). This may or may not be true for subcutaneous adipose tissue, as seen by the results from a 20-week intervention study (You et al., 2006). Forty-five obese (BMI, $33 \text{ kg}\cdot\text{m}^{-2}$) middle-aged females were assigned to one of three groups, each intended to achieve a $2,800\text{-kcal}\cdot\text{wk}^{-1}$ deficit: diet only, diet plus low-intensity exercise, and diet plus high-intensity exercise. All three interventions reduced body weight, fat mass, % BF, waist circumference, and hip girth similarly. However, only the diet plus exercise interventions significantly reduced subcutaneous abdominal adipose cell size. Whether this is another case where the use of waist circumference to reflect changes in VAT masked results that imaging might have shown was unknown.

A study by Redman et al. (2007) provides some insight. This study directly compared the effects of equal amounts of exercise increase and caloric reduction on weight loss, total fat mass

(measured by dual-energy x-ray absorptiometry), and visceral fat mass (measured by computed tomography). Participants were randomized into a control group ([C] who ingested a healthy weight maintenance diet), a caloric restriction group ([CR] who had a 25% reduction in energy intake), or a caloric restriction plus exercise group ([CR + EX] who had a 12.5% caloric restriction and 12.5% increase in energy expenditure). No significant changes occurred in the control group. The CR group lost 8.3% body weight and the CR + EX group lost 8.1% body weight. These losses were not different between groups. Fat losses are presented in **Figure 8.12**. They represented losses of 27 and 22% for total fat mass, 31 and 24% for VAT mass, and approximately 30 and 25% for subcutaneous abdominal adipose tissue in males and females, respectively. Males showed a significant preferential reduction in VAT. These data suggest that exercise training plays the same role as caloric restriction in terms of changes in total fat mass and abdominal visceral fat mass. However, the addition of exercise did improve the aerobic fitness of the participants more, and this has important additional health implications. The pattern of fat distribution throughout the whole body and within the abdominal compartment was not altered by caloric restriction, reinforcing the idea that individuals are genetically programmed for fat storage in a particular pattern.

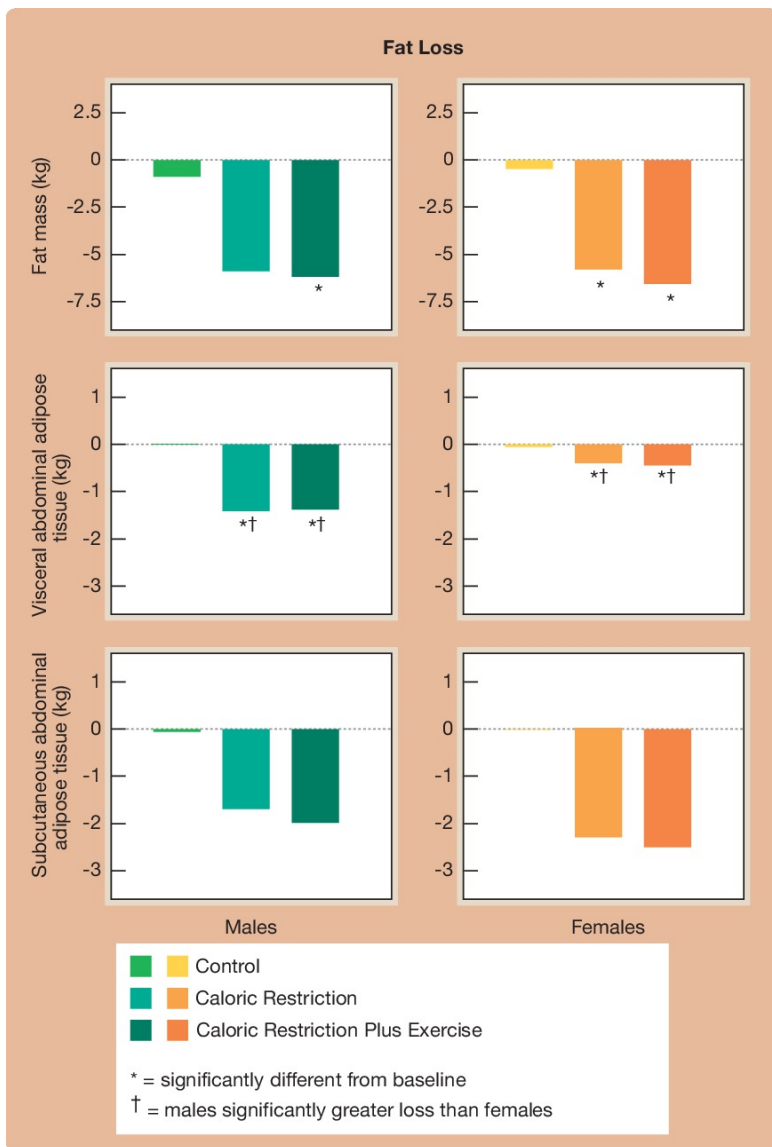


Figure 8.12 The Effect of Caloric Restriction with or without Exercise on Fat Loss.

Individuals were randomly assigned to a control group, a 25% caloric restriction group or a 12.5% caloric restriction and 12.5% exercise training group. After 6 months, both groups lost significant and similar amounts of fat mass and

visceral abdominal adipose tissue and nonsignificant amounts of subcutaneous abdominal adipose tissue. Males showed a significant preferential loss of VAT compared with females. **Source:** Based on data from [Redman et al. \(2007\)](#).

FOCUS ON APPLICATION

Exercise Training and Visceral Fat in Obese Children

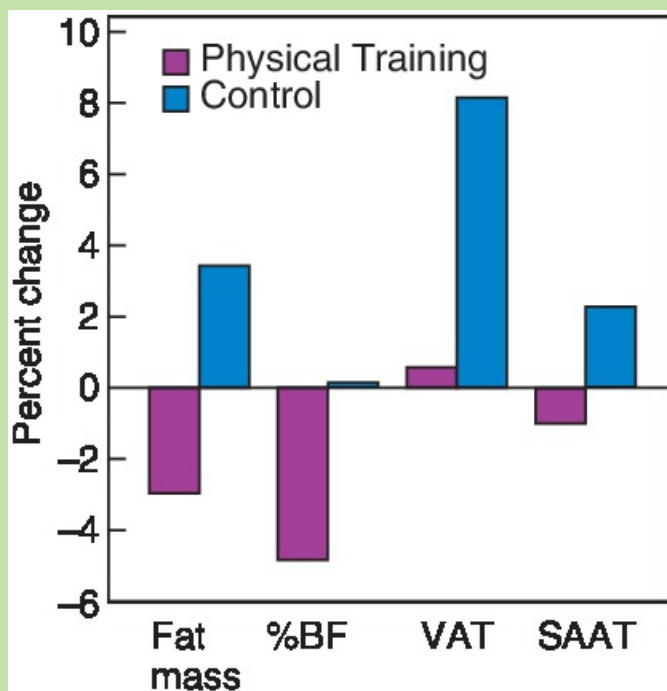
High levels of total body fat mass and visceral adipose tissue (VAT) increase the likelihood that children will develop other coronary artery disease risk factors and non-insulin-dependent diabetes mellitus. [Owens et al. \(1999\)](#) conducted this study to determine if controlled physical training would have a favorable impact on VAT and % body fat in obese children. These authors assigned volunteers to a control group or a physical training group. The physical training group exercised for 40 minutes, at an average heart rate of $157 \text{ b} \cdot \text{min}^{-1}$, 5 days a week, for 4 months. The accompanying graph presents the percent change in several variables in the training group and in the control group after 4 months.

These data indicate that obese children

1. Were capable of participating in a high-intensity exercise training program
2. Experienced a loss in fat mass, % body fat, and SAAT, whereas the control group experienced a gain in fat mass and SAAT over the same period
3. Experienced a smaller increase in VAT than the control group

This study indicates that increasing physical activity of obese children, even without dietary intervention, can

improve the aspects of body composition related to cardiovascular risk factors.



Source: Owens et al. (1999).

Application of the Training Principles for Weight and Body Composition Loss and/or Control

Based on the previously described impact of diet, exercise training, and diet plus exercise training on body weight and composition, the training principles can be applied to achieve weight and body composition loss, change, and/or control using the following guidelines. These guidelines are intended for the mildly or moderately overweight and overfat individuals whom physical educators and fitness leaders are likely to encounter;

they are not meant for more severely obese individuals, who require more drastic reductions and medical supervision.

Specificity

In reality, weight/body composition control is important in three situations:

1. When the goal is to lose weight
2. When the goal is to prevent weight gain, particularly that which tends to occur as individuals age
3. When the goal is to maintain weight loss

Table 8.3 presents physical activity recommendations for weight loss and maintenance from several organizations. The principles for weight loss are discussed first; those for weight maintenance will be discussed under the maintenance training principle.

TABLE 8.3 Physical Activity Recommendations for Weight Loss and Maintenance

| Source | Focus | Recommendation |
|---------------------------------|---|---|
| ACSM (2001, 2009) | Weight loss | 1. A caloric deficit of 500–1,000 kcal·d ⁻¹ achieved by a combination of decreased energy intake and increased energy expenditure 2. A minimum of 150 min·wk ⁻¹ of moderate-intensity physical activity progressing to 250 min·wk ⁻¹ 3. Resistance exercise should supplement aerobic endurance exercise |
| ACSM (2009) | Maintenance of normal weight | 150–250 min·wk ⁻¹ with an energy expenditure of 1,200–2,000 kcal·wk ⁻¹ |
| IOM (2002) | Maintenance of BMI 18.5–25 | ≥60 min·d ⁻¹ of moderately intense physical activity for a PAL of 1.6–1.7 |
| IOTF (Erichman et al., 2002) | Minimize weight gain/maintain normal weight | 60–90 min·d ⁻¹ to achieve a PAL ≥ 1.8 |
| IASO (Saris et al., 2003) | Maintenance of normal weight Prevention of weight regain | 45–60 min·d ⁻¹ or PAL of 1.7 60–90 min·d ⁻¹ of moderate-intensity physical activity or fewer of vigorous-intensity |
| ACSM/AHA (Haskell et al., 2007) | Maintenance of normal weight Prevention of weight regain | ~60 min·d ⁻¹ of moderate to vigorous activity most days of week while not exceeding caloric intake requirements 60–90 min·d ⁻¹ of moderate activity while not exceeding caloric intake requirements |
| ACSM (2009) | Prevention of weight regain | ~200–300 min·wk ⁻¹ ; “more is better” |

ACSM, American College of Sports Medicine; IOM, Institute of Medicine; IOTF, International Obesity Task Force, 2002; IASO, International Association for Study of Obesity; AHA, American Heart Association; PAL, physical activity level.

Weight Loss

Only the 2001 and 2009 recommendations by the American College of Sports Medicine are directed specifically at weight loss. The general goals of weight and body composition control should be (as stated previously) to maximize the decrease in body weight or body fat while minimizing FFM loss and supplying adequate nutrition. To accomplish this, [American College of Sports Medicine \(2001\)](#) recommends a caloric deficit of 500–1,000 kcal·d⁻¹ achieved by a combination of decreased energy intake (dietary restriction) and increased energy expenditure (exercise and/or physical activity). Both dynamic aerobic endurance exercise and dynamic resistance training exercise are recommended. The endurance exercise modality selected (walking, jogging, cycling, swimming, aerobic dancing, stair stepping, or stair climbing) does not matter. The aerobic activity produces most of the caloric deficit, while the resistance activity helps in the maintenance of fat-free mass. A minimum of 150 min·wk⁻¹ of moderate-intensity physical activity is encouraged. A dose-response relationship exists with more physical activity/caloric deficit resulting in more weight loss at least initially. High-intensity interval training (HIIT), where short periods (30-60s) of maximal effort aerobic exercise are interrupted by short periods (30-60s) of low-intensity exercise, can also be used. A 2017 meta-analysis found that this modality of exercise is as effective as traditional aerobic exercise for improving body composition and reducing body weight ([Wewege et al., 2017](#)).

Spot Reduction

Another way to interpret specificity in relation to weight control might be a desire to reduce body fat from a specific body location called spot reduction. The idea behind spot reduction is that fat will be selectively mobilized and thus reduced from the area exercised. For example, as seen in **Figure 8.13**, individuals wishing to reduce his or her abdominal region would concentrate on pelvic tilts, curls, and sit-ups.



Figure 8.13 Anatomically Specific Exercises Do Tone Muscles But Do Not Reduce Fat in That Area.

Consistent training of specific muscles can increase muscle tone, which may give a slimming appearance (or if hypertrophy occurs, give a more defined appearance). However, no experimental evidence shows that fat (in the form of fatty acids) is mobilized preferentially from adipose cells located near active muscles; that is, spot reducing may be an attractive idea but in reality does not work for either males or females. In one study, when 13 men underwent a 27-day training program during which 5,004 sit-ups were performed, no significant changes occurred in %BF, skinfold, or girth measurements either as a result of training or in comparison with six nonexercising controls. Adipose biopsy measures did show significant decreases in fat cell diameters, but there was no significant difference in these changes between the heavily exercised abdominal site and nonexercised gluteal and subscapular locations ([Katch et al., 1984](#)). A newer study involving 24 males and females 18–40 years old divided into either a control group or an abdominal exercise group confirmed these results. The exercise group performed seven abdominal exercises, for 2 sets of 10 reps, 5 days per week for 6 weeks. There was no significant effect on body weight, %BF, android fat, abdominal circumference, and abdominal or suprailiac skinfold

measurements although the exercise group did significantly increase the number of curl-ups performed (Vispute et al., 2011). Similarly, when 7 males and 4 females participated in 12 weeks of localized muscle endurance resistance training, the training was effective in reducing total fat mass, but this reduction was not achieved in the trained body segment (Ramirez-Campillo et al., 2013).

Fat is mobilized to be used as a fuel by hormonal action. Hormones circulate to all parts of the body via the bloodstream. The distribution of body fat in the android (abdominal), gynoid (gluteofemoral), or intermediate pattern does not appear to affect the amount of weight lost by caloric deficit, nor is the relative distribution of fat altered by the weight loss. That is, the general shape of the individual is preserved despite a reduction in the total amount of fat and despite any attempt at spot reduction. Because spot reduction does not work, any activity that burns enough calories to cause a negative caloric balance can be used.

Weight Gain Prevention

Five of the other recommendations from national and international organizations (ACSM, 2009; Erlichman et al., 2002; Haskell et al., 2007; Institute of Medicine, 2002; Saris et al., 2003; **Table 8.3**) as well as a recent ACSM pronouncement (Jakicic et al., 2019) deal with strategies to prevent weight gain [defined by American College of Sports Medicine (2009) as an increase of greater than 3%] in an individual initially of normal weight. All of these have concluded that a minimum of 45–60 min·d⁻¹ or 150 min·wk⁻¹ of moderate to vigorous physical activity are needed for adults, and probably more for children. There is now sufficient prospective epidemiological evidence that an increase of approximately 350 kcal·d⁻¹ (0.25 PAL units) or 50–60 minutes of moderate-intensity activity is needed to prevent weight gain over the years (Haskell et al., 2007; Saris et al., 2003).

The reports cited above introduced another way of quantifying physical activity—**physical activity level (PAL)**. The PAL is defined as the ratio of total energy expenditure (TEE) to 24-hour resting or basal/resting energy expenditure (BMR/RMR), that is, TEE/RMR. Four categories of PAL are frequently used:

sedentary (1.0–1.39), low active (1.4–1.59), active (1.6–1.89), and very active (1.9–2.5) (Ross and Janssen, 2007). By definition, RMR alone is equal to a PAL of 1.0. The sedentary category includes the energy expenditure required for BMR/RMR, TEF, and maintenance activities of daily independent living (ADL). Because everyone has some values for TEF and ADL, a baseline of approximately 1.4 is typically the starting point for calculating additional needed energy expenditure. Thus, if an individual had an RMR of 1,400 kcal·d⁻¹, the difference between PALs of 1.4 and 1.7 is an additional 420 kcal of activity ($1.7 - 1.4 = 0.30$; $0.30 \times 1,400 \text{ kcal} = 420 \text{ kcal}$) (Saris et al., 2003). This can be achieved by a combination of exercise and other physical activities, such as mowing the grass, cleaning house, occupational demands, etc., during the day.

Physical Activity Level (PAL) The ratio of total energy expenditure (TEE) to 24-hour resting or basal/resting energy expenditure (RMR), that is, TEE/RMR .

Complete the [Check Your Comprehension 2](#) box. Check your answer in [Appendix C](#).

CHECK YOUR COMPREHENSION 2—CASE STUDY 2

Danladi is a 48-year-old male professor. He weighs 191 lb and is 6'2" tall. How many calories does he need to expend in activity to achieve a PAL of 1.6? Use the RMR equation in **Table 8.1** to calculate the baseline PAL of 1.4, ignoring the minor variation from BMR caused by TEM and ADL. He likes to play recreational basketball at noon (6 METs) for 3 days a week and walk with his family (3.3 METs) on Saturdays and Sundays. What is the minimum number of minutes he should do each of these activities on any given day to achieve his goal? How many miles should he walk? Why should most of his additional caloric expenditure come from his planned activity and sport? Check your answer in [Appendix C](#). Then, use the MET levels in **Table 4.8** to find the values for your favorite activity, and do

the calculation for yourself.

Overload

When weight loss is the goal, overload really means the attainment of a net deficit. However, the general guidelines for the application of the training principles for dynamic aerobic endurance training outlined in [Chapters 5](#) and [13](#) also apply here. The one exception is a shift in the importance of intensity and duration. The bottom line is that burning large numbers of calories is what is most important.

Previously sedentary, unfit, and overweight or overfat individuals can work at high-intensity levels for short periods of time. Data show that high-intensity interval training can help reduce body fat and increase muscle mass and likely, albeit indirectly, improve the capacity for weight loss in overweight adults ([Gillen et al., 2013](#)). For those who cannot or chose not to participate in high-intensity exercise, low-intensity activities continued for a long duration are recommended. The American College of Sports Medicine (ACSM) recommended duration of exercise for weight loss is presented in [Table 8.3](#). Note that this recommendation is for a minimum of 150 min·wk⁻¹ of moderate-intensity exercise progressing to greater than 250 min·wk⁻¹ of endurance and resistance training. This can be broken up into sessions in any number of different ways. The bottom line is that any combination of frequency, duration, and intensity of exercise that burns sufficient calories to obtain a deficit is the goal for weight loss.

This recommendation is contrary to a common misconception that the best way to lose fat is to burn fat doing long-duration, low-intensity exercise. Although it is true that fat is the dominant fuel in long-duration, low-intensity exercise, the important factor in weight loss is to establish a caloric deficit, regardless of the fuel being used during exercise. This principle is exemplified in a study by [Ballor et al. \(1990\)](#) of two groups of obese women on equally restricted caloric intakes (1,200 kcal·d⁻¹). One group exercised on a cycle ergometer at 51% of their peak $\dot{V}O_{2\max}$ for 50 minutes and the other at 85% of their peak $\dot{V}O_{2\max}$ for 25 minutes. The low-intensity group expended an average of 283

kcal per session at an RER of 0.80, and the high-intensity group expended an average of 260 kcal per session at an RER of 0.92. The estimated fat utilization was 26% for the high-intensity group and 66.6% for the low-intensity group. Although the high-intensity group did improve their cardiovascular fitness more, both groups lost equal amounts of body weight, fat-free mass, fat mass, and percent body fat and had the same decrease in the sum of five skinfold thicknesses.

The advantage of the low-intensity exercise is not an increased loss of fat but a better initial tolerance to the exercise sessions. Individuals who are overweight or obese are also frequently out of shape, and low-intensity work at the initiation of an exercise program is more appropriate and less likely to bring about muscle and joint problems than a high-intensity program. On the other hand, more active or higher fit individuals need not think about decreasing exercise intensity in order to reap the weight control benefits of exercise. The intensity and duration can be manipulated to best suit each individual interested in body weight or composition control.

Increasing caloric expenditure per session and increasing the frequency of the exercise both enhance fat and weight loss. An individual combining dynamic aerobic endurance and resistance weight training can alternate days (three each) and still have a rest day or can combine sessions on each of 3 or 4 days. The caloric cost of dynamic resistance exercise is less than that of dynamic endurance exercise, and this difference needs to be taken into account when designing the exercise program for weight loss or maintenance.

In calculations of the energy cost of an activity session, the *net* value rather than the *gross* value should be used. That is, the calories that would have been burned anyway, had the individual been sedentary, should be subtracted from the cost of the exercise (ACSM, 2022; Pacy et al., 1986). For example, if the energy cost of playing tennis is $7.1 \text{ kcal} \cdot \text{min}^{-1}$ but the individual would have expended $1.3 \text{ kcal} \cdot \text{min}^{-1}$ at rest, the net cost is $5.8 \text{ kcal} \cdot \text{min}^{-1}$. Thus, it would take almost 52 minutes of tennis to burn 300 excess calories, not 42 minutes.

Rest/Recovery/Adaptation

The importance of adaptation in caloric deficit lies in the fact that the composition of the weight loss varies the longer the deficit is maintained. One consistent, moderate-deficit diet is therefore better than a series of short, very calorically restricted diets. The individual must get past the early water loss stages and into the stage where fat loss is proportionally the greatest. This adaptation will occur in addition to the specific adaptations to the exercise training. There is no rest or recovery specific for weight loss beyond that built into any exercise program.

Progression

The greatest progression is made in the number of calories that can be expended in exercise as the individual adapts to the training. When the calories expended in exercise increase, the amount of food ingested can be kept at the same level, thereby increasing the deficit and/or possibly offsetting any decrease in RMR that may occur. Another possibility is to proportionally increase the amount of food ingested to maintain the same relative deficit. The fact that a lower body weight may also mean a lower-energy expenditure for any given physical activity or exercise ([Ghiani et al., 2015](#)) should be kept in mind. Although it may take a greater caloric deficit to continue to lose weight as the weight loss program continues ([Thomas et al., 2013](#)), progression should not be interpreted as an attempt to exercise more and more while eating less and less beyond a reasonable point.

Individualization

To tailor a weight or fat loss program for an individual, several evaluations and calculations are helpful. The first is the direct measurement or estimation of the person's RMR. Formulas such as those presented earlier in this chapter (**Table 8.1**) can be used to estimate RMR fairly easily. Second, the nutrient and caloric intake of the individual should be analyzed. Computer programs are available for this analysis. Third, the number of calories normally expended in daily activity needs to be calculated. Lists of the caloric cost per kilogram of activities, such as that in **Table**

4.8 in [Chapter 4](#), can be used for this calculation.

Once these numbers are known, the relative proportions of the caloric deficit to come from food intake and from energy expenditure can be determined. How much comes from which category should depend on the current caloric intake and fitness level of the individual.

Example

If a sedentary, unfit female ingests $2,000 \text{ kcal}\cdot\text{d}^{-1}$ and expends $1,800 \text{ kcal}\cdot\text{d}^{-1}$, $1,200 \text{ kcal}$ of which is RMR, then an initial 650-kcal decrement makes sense ($2,000 - 1,800 = 200 \text{ kcal}$ to get to a caloric balance, and a 450-kcal deficit for a weight loss). If this individual is really unfit, $100 \text{ kcal}\cdot\text{d}^{-1}$ of exercise may be all she can do; the other 550 kcal needs to come from decreasing the calories ingested. This diet would still allow for daily caloric ingestion of $1,350 \text{ kcal}$. If, however, the individual is relatively fit, more calories can be expended in exercise and either a greater intake of food allowed or a greater deficit achieved.

The caloric ingestion should not fall below the RMR. Although the flat values of $1,200$ for females and $1,500 \text{ kcal}\cdot\text{d}^{-1}$ for males are often suggested as a lower limit of caloric intake ([ACSM, 1983](#)), a more individualized system is to calculate a value halfway between RMR and 30% above RMR as used in the above example ([Schelkun, 1991](#)).

Retrogression/Plateau/Reversibility

Weight loss tends to be uneven, even when caloric intake and output remain the same. Weight loss is fastest in the early stages of caloric deficit and then tends to level off. This pattern occurs, at least in part, because of the early loss of large amounts of glycogen and water. Later, RMR may decrease, and food

efficiency and adaptive decreases in exercise energy expenditure may become a factor (Astrup et al., 1999; Bray, 1969; Major et al., 2007). Although few who are attempting to lose weight want to hear about these patterns, they need to be informed so that they are mentally prepared to deal with them. Reverting to old habits of food intake and a sedentary lifestyle (in a sense, detraining) will result in a rapid retrogression or complete reversal, exemplified by regaining lost weight.

Maintenance

As stated previously, many more people manage to lose weight than to maintain that weight loss. The key to the maintenance of a weight or fat loss may be exercise training (ACSM, 2009; Jakicic and Rogers, 2013; Saris et al., 2003; Tate et al., 2007; Wing, 1999). As can be seen in **Table 8.3**, the major public policy organizations in this area universally recommend exercise/physical activity to maintain weight loss. Evidence is compelling that prevention of weight regain in formerly obese individuals requires at least 60–90 minutes daily of moderate-intensity activity (or lesser amounts of vigorous-intensity activity) or approximately 200–300 min·wk⁻¹ (Jakicic and Rogers, 2013; Saris et al., 2003), which is more than a never-obese individual at a similar absolute body weight. This International Association for the Study of Obesity (IASO) recommendation and the remarkably consistent recommendations from the other reports all assume that caloric intake will not exceed that required to maintain weight. More activity does seem to be better than minimal activity (ACSM, 2009). The level of food intake and exercise output at the weight the individual wishes to maintain must be continued for life.

Weight Cycling

Individuals who fail to maintain a weight loss often find themselves weight cycling. **Weight cycling** is repeated bouts of weight loss and regain. Sometimes, this cycling is called the “rhythm method of girth control” or the yo-yo effect.

Weight Cycling Repeated bouts of weight loss and regain.

In addition to nonmaintaining dieters, some athletes (wrestlers, jockeys, ski jumpers, and the like) (**Figure 8.14**) lose and gain weight purposely as they make weight for competition and then make up for missed meals by eating large quantities. Dieters may take months or years to complete each cycle; weight class athletes will do the same thing in 2 or 3 days. Furthermore, dieters may lose and gain 20–100 lb or more in each cycle, but athletes generally lose or gain less than 20 lb.

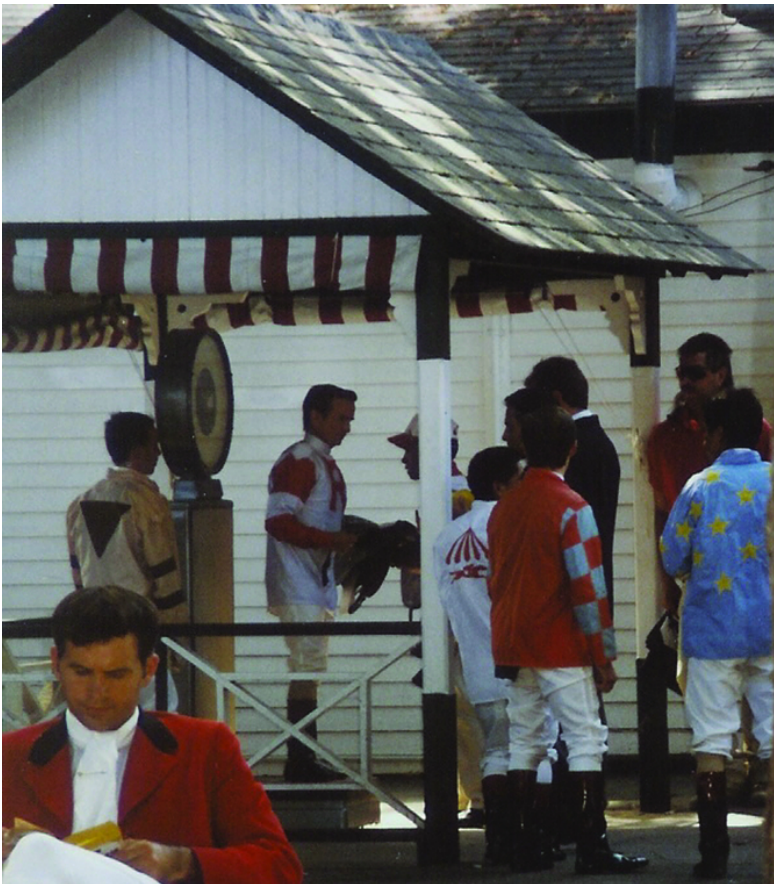


Figure 8.14 Some Athletes, such as Jockeys, Undergo Repeated Bouts of Weight Cycling to Make Weight in

Their Sport.

Jockeys must “weigh out” before races and “weigh in” after races and be within 1 kg of their prerace weight.

While weight cycling is undoubtedly frustrating to dieters and possibly inconvenient for weight class athletes, there have also been concerns about how it affects the health of the individuals involved. It has been theorized that weight cycling slows down the RMR, increases the difficulty of subsequent weight loss, and enhances abdominal fat ([Blackburn et al., 1989](#); [Nash, 1987](#)). Despite the theory, experimental evidence from studies testing the influence of weight cycling on RMR has had inconclusive results. High school wrestlers who weight cycled exhibited a 15% lower RMR than those who did not ([Steen et al., 1988](#)). Conversely, college wrestlers who weight cycled had RMRs similar to those of non-weight-cycling wrestlers, and both wrestling groups had higher RMR values than nonwrestling controls ([Schmidt et al., 1993](#)).

Higher android (upper body) fat deposition, increased adiposity in normal weight, and greater weight gain have been found in several studies of individuals who weight cycled ([Anastasiou et al., 2010](#); [Field et al., 2004](#); [Rodin et al., 1990](#); [Saarni et al., 2006](#); [Strychar et al., 2009](#); [Wallner et al., 2004](#)). Conversely, similar studies have found no difference in fat distribution, waist-to-hip ratio, or increased long-term weight gain as a function of weight-cycling history ([Graci et al., 2004](#); [Jeffery et al., 1992](#); [Mason et al., 2010](#); [van Wye et al., 2007](#)). It may matter what the initial body weight/body composition is. Indeed, it is dieting to lose weight in people who are in the healthy normal range of body weight, rather than those who are overweight or obese, that most strongly and consistently predicts future weight gain ([Dulloo et al., 2015](#)). There is no direct magnetic resonance imaging (MRI) evidence that weight cycling leads to increased abdominal visceral fat deposition ([Montani et al., 2006](#)). A 2017 systematic review that included 31 studies of nonathletes concluded that there is insufficient evidence to suggest that weight cycling promotes obesity and only three of these studies reported a reduced RMR. Thus, weight loss attempts should be encouraged, despite cyclical behaviors ([Mackie et al.,](#)

2017). Thus, neither body fat distribution nor body composition in humans is conclusively affected by a history of weight cycling (Mackie et al., 2017; National Task Force on the Prevention and Treatment of Obesity, 1994).

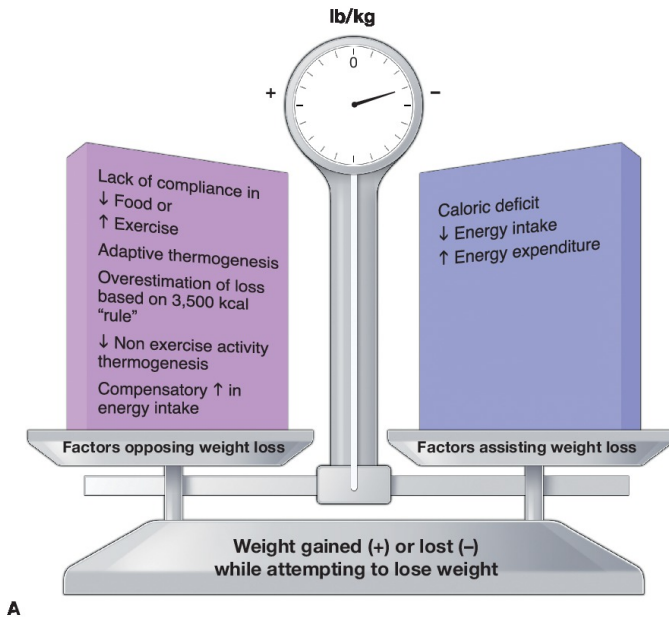
Another area of controversy involves evidence of increased cardiovascular risk factors, cardiovascular disease mortality, and all-cause mortality associated with weight cycling (Diaz et al., 2005; Montani et al., 2006; Olson et al., 2000; Rzehak et al., 2007). It has yet to be determined if this relationship is directly or indirectly linked to fat accumulation or other factors such as the effects of preexisting disease and smoking. There is neither an established mechanism nor firm evidence that weight loss or weight fluctuation in otherwise healthy previously overweight/obese individuals is hazardous (Graci et al., 2004; Strychar et al., 2009; Wannamethee et al., 2002). More importantly, the previously cited 2017 meta-analysis concluded that there is no evidence suggesting that weight cycling negatively impacts cardiometabolic risk factors (Mackie et al., 2017).

However, counterintuitively, there is emerging evidence that fluctuations of cardiovascular risk factors such as blood pressure, blood glucose, lipids, and insulin put more stress on the cardiovascular system of relatively lean people who initially did not really need to lose weight but chose to diet and ended up rapidly regaining more weight than they initially started with during each cycle than those who were initially overweight or obese (as a result of a progressive weight gain) and ended up weight cycling. More and more individuals are beginning to diet at early ages and could thus be setting themselves up for major future difficulties (Dulloo et al., 2015; Montani et al., 2015). It is, of course, preferable to lose weight into a healthy zone and/or maintain a healthy weight rather than to weight cycle. This is unfortunately easier said than done.

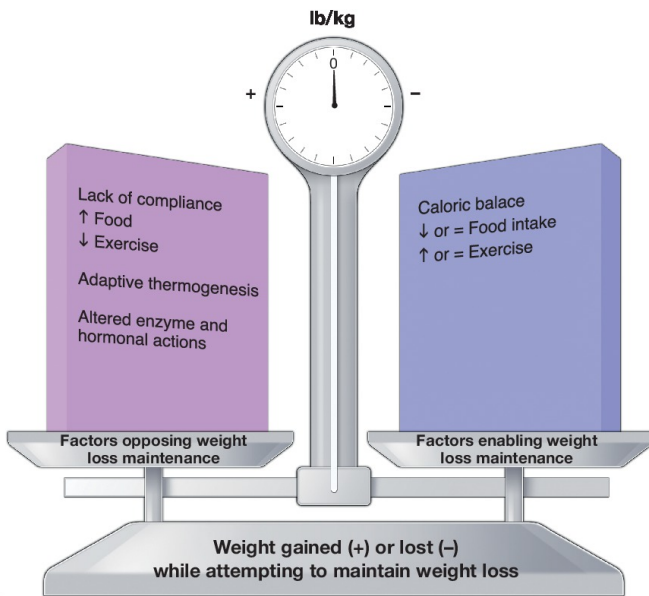
Why Is It So Hard for Some People to Lose Body Weight/Fat?

Under the same energy deficit conditions, whether imposed by diet, exercise, or diet + exercise, there is a wide variation in body weight/fat loss. Why is it so hard for some individuals to

lose weight? Why do almost all individuals lose less weight than theoretically expected? And why do virtually all individuals seeking to lose weight ultimately plateau ([Melanson et al., 2013](#))? How many times have you heard someone say: “I just can’t seem to lose that last 5 lb”? There are a number of possibilities (**Figure 8.15A**):



A



B

Figure 8.15 **A.** Factors that oppose or assist in weight loss while attempting to lose weight. **B.** Factors that oppose or enable weight loss maintenance while attempting to

maintain weight loss.

1. Lack of compliance
2. Overestimation of weight/fat loss based on the 3,500-kcal “rule”
3. A reduction in NEAT (nonexercise activity thermogenesis)
4. A compensatory increase in energy intake
5. Adaptive thermogenesis

LACK OF COMPLIANCE It is possible that any given individual is simply not exercising as much as he or she thinks or as much as is needed and/or is actually eating more than is being reported. Self-reporting of both of these variables is notoriously unreliable with underreporting of energy intake in the 20–30% range (Melanson et al., 2013; Thomas et al., 2012). If the energy imbalance is lower than prescribed, the weight/fat loss will also be lower. However, there are any number of well-controlled studies in which equally compliant individuals lose vastly different amounts of weight/fat (see **Figure 8.8**).

OVERESTIMATION OF WEIGHT/FAT LOSS BASED ON THE 3,500-KCAL “RULE” The deficiencies of this “rule” have been previously discussed. Part of the difficulty with this guideline is the uncertainty of the baseline energy needed to maintain the starting weight. If this value is inaccurate (and in many cases, it is only a calculated estimate or generic 1,200–1,500 kcal·d⁻¹), then the estimated changes based on this will also be inaccurate (Hall, 2012).

A REDUCTION IN NEAT (NONEXERCISE ACTIVITY THERMOGENESIS) The failure of some individuals to lose weight/fat may also result from those individuals compensating for the increased energy expenditure during exercise by decreasing energy expenditure the rest of the day (sometimes called nonexercise activity thermogenesis or NEAT). Some, but not all, studies show a clear trend for a decrement in NEAT with an energy deficit and that decrement may be more likely when diet is used alone to achieve the deficit. Older individuals, in particular, are more likely to decrease nonexercise physical

activity. Some of this may be on purpose (the individual may feel tired and decided to do less) or it may be unconscious (Melanson et al., 2013).

A COMPENSATORY INCREASE IN ENERGY INTAKE When exercise is used as the sole means of attempting to achieve an energy deficit and there is no specific energy intake restriction, a compensatory increase may occur in energy intake. The evidence is not compelling; however, whether this occurs or not may depend upon the degree of overweight/obesity at the initiation of the weight/fat loss program. In general, lean individuals (who nonetheless want to lose weight) tend to spontaneously increase their energy intake to unconsciously maintain energy balance, whereas obese individuals are more likely not to do this (Melanson et al., 2013; Thomas et al., 2012). Nonetheless, any given individual can be susceptible to enhanced hunger and hyperresponsive to the pleasures of eating and increase intake (Melanson et al., 2013; Tremblay et al., 2013). It has been shown that there is a large variation among individuals in the amount of compensation in caloric intake with an average being around 50% of the energy expended in exercise without regard to the total energy expenditure (Flack et al., 2020).

ADAPTIVE THERMOGENESIS As stated earlier in this chapter, the term adaptive thermogenesis is not unequivocally defined but has come to mean any change in heat production or energy expenditure in response to a changing internal or external environment, indicating either an increase or decrease in the efficiency of energy utilization. In the context of energy expenditure relative to weight/fat loss difficulty, this means a reduction in energy expenditure (i.e., an increase in energy efficiency). Decreasing adaptive thermogenesis is independent of both cold-induced and diet-induced thermogenesis. While cold- and diet-induced thermogenesis probably reside in brown adipose tissue and increase energy expenditure in the form of heat, decreasing adaptive thermogenesis is primarily located in fat-free mass (most body organs including skeletal muscle) (Müller and Bosy-Westphal, 2013). It may represent a defense mechanism to protect energy stores from depletion. This adaptation occurs in both resting metabolic rate (RMR) as previously discussed and

the energy expenditure for any given physical activity/exercise. Part of the decrease cost of activity/exercise is because of any weight/fat loss that has occurred, but the remainder is due to the increase in energy efficiency. This change has been shown to average $-15.4 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ or 7% beyond weight-related changes. Paradoxically, the greater the decrease in thermogenesis, the higher the fasting hunger sensations. A decrease in plasma thyroid hormone (T3), leptin, and sympathetic nervous system activity has been found in weight-reduced obese individuals to be among the possible causes of adaptive thermogenesis (Thomas et al., 2012; Tremblay and Chaput, 2009; Tremblay et al., 2013).

Why Is It So Hard for Many People to Keep Body Weight/Fat Off Once It Has Been Lost?

There is no easy answer to why maintenance of a body weight/fat loss is so difficult. The only sure thing is that the problem is pervasive. The recidivism rate is approximately 75–80% in both youngsters and adults. That is, after an otherwise successful weight loss, the vast majority of obese individuals who have successfully lost weight regress to pre-weight loss levels of body weight/fat. This appears to result from the coordinated action of behavioral, neuroendocrine, and metabolic responses designed to maintain body energy stores (fat) at a central nervous system-defined “ideal” weight (Rosenbaum and Leibel, 2010). The following factors (**Figure 8.15B**) appear to operate for weight/fat regain:

1. Lack of compliance
2. Altered enzymatic and hormonal functions
3. Adaptive thermogenesis

LACK OF COMPLIANCE The most obvious behavioral reason for the difficulty in maintaining weight loss, at least in part, is failure of compliance—the failure to maintain a regular exercise program of sufficient energy expenditure and/or a healthy calorically controlled dietary intake. Sooner or later, many individuals revert back to the same lifestyle that made their

weight loss necessary initially. We live in an obesogenic environment marked by fast food promotion, high availability of calorically dense foods and beverages, a social milieu based around partying, large serving sizes, and decreased necessity and opportunity for active employment, transport, and recreation. Such environmental pressures may overshadow biological control of body weight. It is important in the maintenance phase of a weight loss program to adhere to obesity-reducing behaviors. Those who successfully maintain weight losses exhibit frequent self-monitoring of food and calorie intake, select lower-calorie foods with little variation, plan meals in advance, eat breakfast, weight themselves regularly, limit television, and participate regularly in an exercise program (Greenway, 2015; Strohacker et al., 2014; Sumithran and Proietto, 2013). For example, Tate et al. (2007) randomly assigned 202 overweight adults into either 18 months of standard behavioral treatment (SBT) with an exercise goal of 1,000 kcal·wk⁻¹ or a high physical activity (HPA) group with an exercise goal of 2,500 kcal·wk⁻¹ in addition to the SBT. The HPA group achieved significantly greater exercise levels and weight loss than the SBT group at 12 and 18 months of follow-up. At 30 months, the average exercise levels were no longer different between the groups, nor did weight loss differ. Participants who reported continuing to engage in high levels of exercise maintained a significantly larger weight loss.

Similarly, when 14 participants in the Biggest Loser competition were measured 6 years later, those who maintained the greater weight loss (approximately -25%) had increased their physical activity on average by 160% versus 34% in the weight regainers (~1% heavier than their precompetition baseline). Energy intake changes were similar between the weight loss maintainers (approximately -9%) versus the weight regainers (approximately -7%). The weight regain was inversely associated with the absolute changes in physical activity but not energy intake (Kerns et al., 2017). Large persistent increases in physical activity (not just the amount of exercise used to lose the weight) may be required for long-term maintenance of lost weight.

There seems, however, to be more to the problem than a lack of compliance. Will power may be counteracted by powerful internal signals that sense deviations in body weight and trigger

compensatory mechanisms such as those discussed below (Dulloo, 2007; Major et al., 2007).

ALTERED ENZYMATIC AND HORMONAL FUNCTIONS When individuals gain weight, body composition also changes including significant alterations in adipose cellularity. Initially, adipose cell size increases (hypertrophy), but eventually, adipose cell number will also increase (hyperplasia). With body weight loss, adipocyte hypertrophy decreases, but adipocyte hyperplasia does not. One possible internal signal is lipoprotein lipase. Lipoprotein lipase is the enzyme responsible for fat synthesis and storage in adipose tissue. It has been theorized that lipoprotein lipase notifies the brain when fat cells have shrunk, as they would when the body is losing weight. In response, appetite increases, fat metabolism slows, and fat cells refill. In other words, “starving leads to stuffing.” The appetite not only increases but also seems selectively predisposed to disproportionately increase the amount of fat ingested. Fat, of course, is calorically dense. Furthermore, after restrictive dieting, the body can more easily convert other excess food nutrients to fatty acids. Previously or currently obese individuals seem to be more susceptible to the stimulus and response of lipoprotein lipase. In these individuals, lipoprotein lipase activity may be twice as high as in nondieting individuals. That is the bad news. The good news is that when people who are not obese are put on a mildly or moderately restricted diet, the changes in lipoprotein lipase activity are minimal. It may be that a certain amount of weight (~15% of total body weight) must be lost before lipoprotein lipase activity becomes a counterproductive factor (Gershoff, 1991, 1992; Nash, 1987; Schelkun, 1991).

In addition to insulin, several other appetite-related hormones have a key role in weight regain after weight loss. With the exception of increases in PP (pancreatic peptide), changes in hormones following weight loss tend to favor weight regain by increasing hunger and promoting energy storage. Following diet-induced weight loss, there are increases in circulating levels of ghrelin and decreases in circulating levels of leptin, PYY (peptide tyrosine-tyrosine), CCK (cholecystokinin), insulin, and GLP-1 (glucagon-like peptide) (Greenway, 2015) (Figure 8.3). These changes can persist at least up to 1 year.

Recall that leptin is an appetite-suppressing (anorexigenic) hormone primarily secreted by adipose tissue. Conversely, ghrelin is an appetite-stimulating (orexigenic) hormone secreted primarily by endocrine cells in the gastrointestinal tract. Given the respective roles of leptin and ghrelin, it would be expected that in obese individuals, pre-weight loss leptin levels would be decreased and ghrelin levels increased. Precisely, the opposite is true. Obese individuals exhibit high levels of leptin and low levels of ghrelin. Obese individuals seem to be leptin resistant—exhibiting high levels of leptin whose function is blocked. At the same time, obese individuals seem to be overly sensitive to ghrelin, and the decreased levels may represent a physiological adaptation to the positive energy balance of obesity (Huda et al., 2006; Klok et al., 2007). Leptin levels are reduced within 24 hours of energy restriction. These reductions appear to trigger a “starvation defense response” despite the availability of abundant fat stores. Exercise training protocols that result in reduced fat mass are also generally accompanied by lower leptin concentrations (Bouassida et al., 2010). The result is a reduction in metabolic rate and physical activity (hence lower-energy expenditure), increased hunger, reduced thyroid metabolism, and diminished sympathetic activity (Ochner et al., 2013; Rosenbaum et al., 2005).

When an individual loses weight, ghrelin levels increase. Conversely, ghrelin levels decrease when anorexia nervosa patients gain weight. These opposing responses suggest that ghrelin levels change in response to energy intake variations to maintain the current body weight. Increasing ghrelin, therefore, might be a compensation for weight loss, making maintenance of that loss difficult (Hansen et al., 2002; Klok et al., 2007; Leidy et al., 2007b; Romon et al., 2006). The good news is that the rise in ghrelin with weight loss may be transient and may eventually decrease with weight maintenance if that can be achieved. In a randomized clinical trial using diet, exercise, and the weight loss drug orlistat, a group of obese females lost 8.5% of their body weight in 6 months. They had the expected rise in ghrelin. However, when they maintained their weight loss for 6 months, their ghrelin levels returned to baseline (Garcia et al., 2006). Thus, ghrelin may be a counterregulatory mechanism in the short but not long term. In addition, a low-fat diet seems to have an

inhibitory effect on ghrelin levels (Klok et al., 2007). Protein consumption results in ghrelin suppression over a long period of time. Carbohydrate consumption results in an initial suppression of ghrelin more than protein, but then, ghrelin rebounds to a higher than baseline level within hours (Foster-Schubert et al., 2008). Despite the importance of leptin, ghrelin, and insulin, the changes that have been reported in studies have not been found in and of themselves to predict weight regain following weight loss (Strohacker et al., 2014).

Both PYY and CCK promote satiety. Thus, their reduction potentially encourages formerly obese individuals to overeat (Ochner et al., 2013). Reduced thyroid hormone (T3) results in decreased thermogenesis (discussed below) and overall metabolic rate (Trexler et al., 2014).

ADAPTIVE THERMOGENESIS An adaptive decrease in (or suppression of) thermogenesis indicates a change in the efficiency of energy utilization that results in a reduction of energy expenditure in any or all components of TEE including TEF, resting, nonexercise, and exercise expenditure. The composition of the diet used for weight loss may influence subsequent weight gain. Low-fat diets tend to decrease TEE and RMR more than low-carbohydrate diets (Greenway, 2015).

Maintenance of $\geq 10\%$ reduction in body weight in either lean or obese individuals is accompanied by approximately 20–25% decline in a 24-hour TEE. This decline in weight maintenance calories is 10–15% below what is predicted based solely in the accompanying alterations in FFM and FM. Thus, a formerly obese individual would require approximately 300–400 fewer calories per day to maintain the same body weight/composition and physical activity level as a never-obese individual of the same body weight/composition (Rosenbaum and Leibel, 2010; Trexler et al., 2014).

As described earlier, the TEF depends on macronutrient and caloric content of the food ingested. When caloric input goes down in dieting, so does the TEF. Further, as was seen in the Focus on Research box, the TEM decreases with a reduction in body weight. The adaptive decrease in TEF indicates a change in food efficiency. **Food efficiency** is an index of the number of

calories an individual needs to ingest to maintain a given weight or percent body fat. Food efficiency *increases* when the calories needed to sustain a certain weight (or percent fat) decrease. This response could be protective if food were scarce. Food efficiency *decreases* when the number of calories needed to sustain a certain weight (or percent fat) increases. Being able to eat more to maintain a given weight is what every dieter would like to have happen, but which does not happen ([Brodie, 1988](#)).

Food Efficiency An index of the number of calories an individual needs to ingest to maintain a given weight or percent body fat.

A residual reduction (adaptive decrease) in RMR is well documented but not universally shown, in part because of wide differences among individuals. The impact on RMR is particularly important because it accounts for the highest percentage of daily energy expenditure. When 12 studies were combined in a traditional meta-analysis, relative RMR was 5.1% lower in the formerly obese group than the control group. The authors concluded that the cause of the lower RMR remained unknown; however, the implication was clear. A low RMR is likely to contribute to the formerly obese individual's difficulty in maintaining a weight loss ([Astrup et al., 1999](#); [Fothergill et al., 2016](#)).

As stated in the last section, many, but not all, studies show a clear trend for a decrement in NEAT (nonexercise activity such as fidgeting or normal daily activities) during a period of energy deficit. This decrement persists after individuals return to a free choice diet and are simply trying to maintain the weight loss unfortunately making this goal more difficult to achieve ([Trexler et al., 2014](#)).

Finally, an adaptive decrease in exercise energy expenditure has also been documented ([Greenway, 2015](#); [Major et al., 2007](#); [Trexler et al., 2014](#)). Several studies involving dietary and activity analysis indicate that some female distance runners ingest only between 1,400 and 1,990 kcal·d⁻¹ while training as much as 65 mi·wk⁻¹. These caloric intake values are much lower than

those for comparably sized, inactive females of the same age (Brownell et al., 1987). Similar discrepancies have been shown in obese adult males walking on the treadmill (Doucet et al., 2003).

Part of this decrease in energy expenditure may be related to changes in uncoupling protein-3 (UCP3). UCP3 is similar to UCP1 in that it separates oxidative phosphorylation from ATP synthesis with energy dissipated as heat. UCP1 is found in BAT and beige tissue; UCP3 is located primarily in skeletal muscle mitochondria. Decreases in UCP3 have been documented in humans in response to energy restriction and as such could potentially play a role in decreasing energy expenditure. Individuals identified as “diet resistant” have shown decreased UCP3 activity compared to “diet-responsive” subjects during maintenance of a reduced bodyweight (Trexler et al., 2014).

Making Weight for Sport

Although athletes in many sports are concerned with maintaining a low body weight and/or a low-percent body fat, only a few sports organize the competition around weight classes. The original intention was to make the competitions as fair as possible by matching individuals of approximately the same size. However, in practice, many participants manipulate their body weight to drop down to a lower-weight class on the assumption that they will then have a physical and psychological advantage over their opponents. One wonders, however, what possible advantage there can be when both competitors are following the same strategy (although each athlete will always think he or she can drop down more than his or her opponent) and, more importantly, the health and performance implications are ignored. Despite these concerns, dropping into a lower-weight class is routinely done, often by individuals in their growth years. Competitors in boxing, judo, karate, mixed martial arts, taekwondo, wrestling (free style and Greco Roman), weight and power lifting, rowing, American “Sprint” football, and horse racing all routinely engage in making weight, but most research attention has involved wrestlers (**Figure 8.16**).

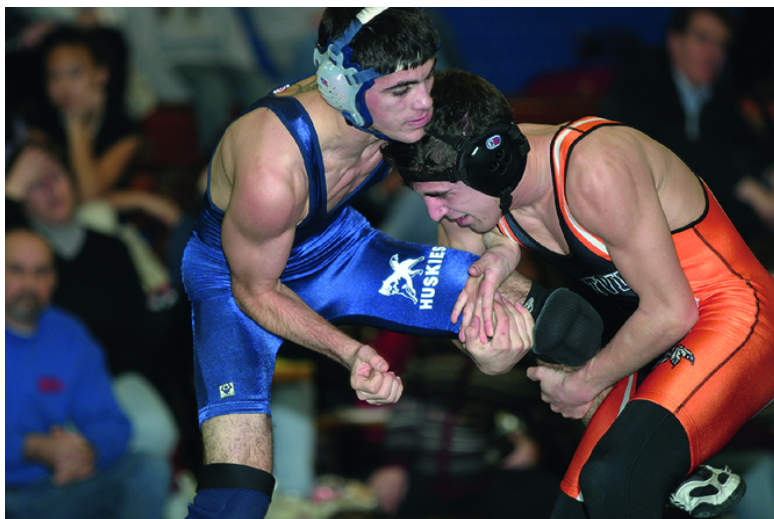


Figure 8.16 Making Weight by Following Sound Scientific Guidelines Should Be an Important Goal for Wrestlers at All Levels of Competition.

The American College of Sports Medicine has published several versions of the “Position Stand on Weight Loss in Wrestlers,” the latest titled “ACSM Expert Consensus Statement on Weight Loss in Weight-Category Sports ([Burke, et al., 2021](#)). The techniques typically used by wrestlers and the physiological consequences were detailed in the 1976 version, as well as suggestions for “making weight” in a healthier manner. Health concerns include cardiovascular complications and/or heat stroke secondary to severe fluid restriction, passive heat exposure, and/or extreme exercise to induce large sweat losses that in turn can also lead to electrolyte imbalances and acute renal stress. Chronic exposure to negative energy balance and low energy availability both contribute to the Relative Energy Deficiency in Sport (RED-S) syndrome discussed in [Chapter 6](#). The most serious consequence is death ([Burke et al., 2021](#)). In 1997, in one 5-week period, three collegiate wrestlers died from complications caused by rapid weight loss ([Centers for Disease Control and Prevention, 1998](#)). These tragedies brought about rule changes from the NCAA (and adopted by many high school associations). These changes and regulations included adding 7 lb to each weight

class, moving weighins to no less than 2 hours before the competition; requiring preseason assessment of body composition, hydration, and weight by a member of the school's athletic medical staff; a minimal weight for each wrestler; and regulation of weight loss/gain. The raw data from the preseason assessments are entered into the online National Wrestling Coaches Association Optimal Performance Calculator. An ideal minimal competition weight and a safe weight loss/gain plan are established, and athletes are assigned to daily nutrient goals based on their weight loss/gain plan. This plan mandates that a wrestler may not lose more than 1.5% of body weight per week while descending to the lowest certified weight class. The ACSM guidelines of not less than 7% BF for male competitors younger than 16 years and not less than 5% BF for males 16 years or older are part of this mandated plan. Female wrestlers are required to have a minimum of 12–14% BF. Total body weight loss cannot exceed 7%. The use of laxatives, emetics, excessive fluid and food restriction, self-induced vomiting, hot environments (including practice rooms of $>80^{\circ}\text{F}$), saunas, steam rooms, diuretics, vapor-impermeable suits, and intravenous rehydration are prohibited (Khodae et al., 2015; National Collegiate Athletic Association, 2007; Rosenfeld, 2014). Hydrostatic (underwater) weighing, displacement plethysmography (Bod Pod), and skinfolds using the Lohman (1981) equation are approved by the NCAA to determine body composition. Several high school associations allow bioelectrical impedance analysis (BIA) as well, although BIA has been shown to have a high prediction error in wrestlers and research indicates that skinfolds and BIA body composition values cannot be used interchangeably (Clark et al., 2002, 2005).

The Lohman skinfold equation is as follows:

$$\text{body density (g} \cdot \text{cc}^{-1}) = 1.0982 - \{[0.000815 \times \text{sum of triceps + subscapular + abdominal skin folds (mm)}] + [0.0000084 \times \text{sum of triceps + subscapular + abdominal skinfolds squared (mm)}]\}$$

$$D_b = 1.0982 - (0.000815 \text{ sum of skinfolds} + 0.0000084 \text{ sum of skinfolds}^2) \text{ (ACSM, 1983)}$$

Example

If a 17-year-old wrestler weighs 165 lb and his sum of skinfolds for the selected sites is 46 mm, the calculation would be

$$\begin{aligned}D_B &= 1.0982 - [0.000815 (46) + 0.0000084 (2116)] \\&= 1.0429 \text{ g} \cdot \text{cc}^{-1}\end{aligned}$$

The D_B value is then substituted into the age-appropriate formula presented in **Table 7.2** in **Chapter 7** for a male adolescent to determine %BF. For a 17-year-old, this is

$$\%BF = \left[\frac{5.03}{D_B} - 4.59 \right] \times 100 = 23.31$$

Equations 7.3, 7.4, and 7.5 are then used to determine the wrestler's most appropriate competitive weight. Using **Equation 7.3**,

$$FFW = 165 \text{ lb} \times \left[\frac{100\% - 23.3\%}{100} \right] = 126.6 \text{ lb}$$

Using **Equation 7.3**,

$$WT_2 = \left[\frac{100 \times 126.6}{100\% - 16.3\%} \right] = 151.3 \text{ lb}$$

Note: 16% is used here as the desirable %BF, not 5%, which is the lowest recommended %BF for a wrestler of this age. The 16.3% complies with the recommendation that weight loss not exceed 7% of body weight.

To get down to 5% BF, this wrestler would need to lose 18.3% of his body weight and that is too much. Using **Equation 7.5**,

$$151.3 \text{ lb} - 165 \text{ lb} = -13.7 \text{ lb}$$

To achieve his recommended body weight, this wrestler needs to lose 13.7 lb.

Despite several decades of work, harmful weight-making practices still are followed and the standards themselves are not without problem—one of which is uneven enforcement. It is important that expert guidance by a sports nutrition professional is available to each weight-category sport athlete to establish a workable long-term approach to body mass and body fat management. Such advice needs to take into account the nuances of the particular sport while attempting to achieve both positive health and competitive performance outcomes for the athlete (Burke et al., 2021). Complete the case study in the [Check Your Comprehension 3](#) box.

CHECK YOUR COMPREHENSION 3—CASE STUDY 3

Calculate the weight at which the following 14-year-old wrestler should compete.

| | |
|----------------|----------------------------|
| Name: Zachary | Triceps skinfold: 8 mm |
| Weight: 138 lb | Subscapular skinfold: 9 mm |
| | Abdominal skinfold: 12 mm |

How much weight does Zachary need to gain or lose to achieve this weight?

Check your answer in [Appendix C](#).

Several studies have investigated compliance with the 1998 regulations. A 1999 survey of 43 collegiate teams (Oppliger et al., 2003) found that the most weight lost during the season was 6.9% of body weight, but average weekly weight loss was 4.3%. Although 40.2% indicated that the then-new NCAA rules deterred extreme weight loss behaviors, approximately 55% fasted, approximately 28% used saunas, and approximately 27% used

vapor barrier suits at least once a month. Overall, however, compared to college wrestlers in the 1980s, weight behavior was less extreme. A follow-up study (Oppliger et al., 2006) of 811 competitors in Division I, II, and III national championship tournaments from 1999 to 2004 showed that weight and %BF decreased from preseason to postseason competition. However, the preseason certified minimum weight remained unchanged (68.0 ± 9.2 kg vs. 67.9 ± 9.1 kg), thus showing good agreement between the preseason recommendations and the actual end-of-season weights. Rapid weight loss before weigh-in was found to be statistically significant but small ($\sim 1.7\%$ of body weight). The average wrestlers at the tournaments were competing at 9.5% BF, down from the preseason average of $12.3 \pm 3.4\%$, but well above the minimum of 5% . The investigators concluded that the NCAA weight management program appears to be effective in reducing unhealthy weight-cutting behaviors (although the wrestlers were not asked how they achieved weight goals) and promoting competitive equity.

Summary

1. Weight gain, loss, and stabilization follow the first law of thermodynamics as expressed in the caloric balance equation. The components of this equation are food ingestion (+), resting or basal metabolic rate (–), thermogenesis (–), and exercise (–).
2. Diet, acute exercise, and exercise training can have an impact on the components of the caloric balance equation (see **Table 8.2**).
3. The goal of weight or fat control should be to lose body fat (especially visceral abdominal fat), to preserve fat-free mass (FFM), to maintain or improve health, and for athletes to maintain or improve performance.
4. The theoretical $3,500\text{-kcal}\cdot\text{lb}^{-1}$ rule significantly overestimates the size of weight loss in practice.
5. Body weight loss from very low (ingesting $400\text{--}800$ kcal $\cdot\text{d}^{-1}$) and low (ingesting $800\text{--}1,200$ kcal $\cdot\text{d}^{-1}$) dietary

restriction is approximately 75% BF and 25% FFM; from a moderate intake diet ($1,200\text{--}1,500 \text{ kcal}\cdot\text{d}^{-1}$), the split is approximately 90% BF and 10% FFM.

6. Body weight loss from exercise alone rarely is as much as from diet alone, probably because of insufficient energy expenditure, compensatory decreases in nonexercise energy expenditure, or an increase in unrestricted energy intake. When these factors are controlled, weight loss from exercise alone will not differ from diet alone. More FFM is preserved when weight is lost using exercise alone than diet alone.
7. A combination of diet plus exercise is the preferred technique for body composition and body weight control both to accomplish an initial loss and to maintain a weight loss.
8. The exercise training component of weight and fat control should include both an aerobic endurance portion (to expend $1,200\text{--}2,000 \text{ kcal}\cdot\text{wk}^{-1}$) and a resistance weight training portion (to maintain and/or build FFM).
9. Under the same energy deficit, there are wide individual differences in body weight/fat loss. Possible reasons include (1) lack of compliance, (2) overestimation of weight/fat loss based on the theoretical $3,500\text{-kcal}\cdot\text{lb}^{-1}$ rule, (3) a reduction in NEAT, (4) a compensatory increase in energy intake, and/or (5) adaptive thermogenesis.
10. Difficulty in maintaining weight/fat loss may be linked to (1) lack of compliance, (2) altered enzymatic and hormonal functions, and/or (3) adaptive thermogenesis.
11. Proper weight for all sports should be determined based on %BF and safe weight loss procedures.

Review Questions

1. State the caloric balance equation, and relate it to the first law of thermodynamics. Define and explain the components of the caloric balance equation.
2. Discuss the impact of dietary restriction on the components

of the caloric balance equation.

3. Discuss the impact of exercise on the components of the caloric balance equation.
 4. Discuss the impact of exercise training on the components of the caloric balance equation.
 5. Compare and contrast the effects of diet alone, exercise training alone, and diet and exercise training combined on body weight and composition control.
 6. List and explain how each of the training principles should be specifically applied for body weight or body composition control or maintenance.
 7. Defend or refute the following statements, using evidence provided in this chapter.
 - a. Weight cycling makes subsequent weight loss physiologically more difficult.
 - b. The most important reason to add exercise or exercise training to a weight loss or maintenance program is that exercise decreases appetite.
 - c. If food is eaten near the time of exercise (either directly before or after), the thermic response is potentiated (made more effective) so that more calories are burned and weight is lost faster.
 - d. The maintenance of, increase in, or decrease in resting metabolic rate depends on the maintenance or change in lean body mass.
 - e. To lose fat, burn fat by doing long-duration, low-intensity exercise.
 8. Why is it so hard for some people to lose body weight/fat?
 9. Why is it so hard for many people to keep body weight/fat off once it has been lost?
 10. Prepare a set of weight control guidelines for a jockey keeping in mind that jockeys pre- and postrace weight must be within 1 kg of each other and they can ride several days a week.
-

Literature Search

In this chapter, we discussed body composition and weight control. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search exercise and body composition, and this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. High-intensity interval training and body composition
 - ii. Resistance exercise and weight control
 - iii. Nutrition and body composition
 - iv. Athletes and body composition
 - v. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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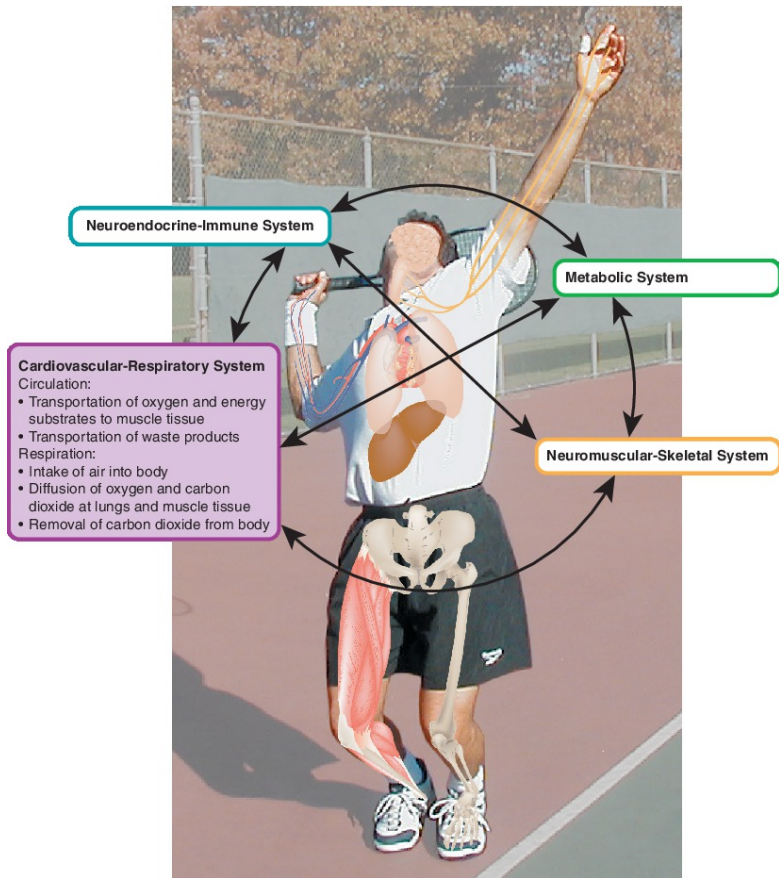
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Cardiovascular- Respiratory System Unit



The cardiorespiratory system brings oxygen into the body (respiratory system) and transports it to the cells (cardiovascular system), which use the oxygen in the production of energy through the process of cellular respiration. Thus, the respiratory and cardiovascular systems are functionally linked and often referred to collectively as the cardiovascular-respiratory, or cardiorespiratory, system. The cardiorespiratory system directly supports metabolism by delivering oxygen and nutrients to the cells of the body. The metabolic production of ATP from oxygen and foodstuffs then directly supports the neuromuscular system by providing the energy for muscle contraction. The neuroendocrine-immune system plays an important role in regulating both the respiratory and the cardiovascular systems.

9 Respiration



Chapter Outline

Introduction

Structure of the Pulmonary System

- The Conductive Zone

- The Respiratory Zone

Mechanics of Breathing

Respiratory Circulation

Minute Ventilation/Alveolar Ventilation

- Major Pulmonary Ventilation Variables

Measurement of Lung Volumes

Static Lung Volumes
Dynamic Lung Volumes
Spirometry
Gas Dilution
Standardization

Partial Pressure of a Gas: Dalton's Law

Regulation of Pulmonary Ventilation

The Respiratory Centers
Anatomical Sensors and Factors Affecting Control of
Pulmonary Ventilation

Gas Exchange and Transport

Gas Exchange: Henry's Law
External Respiration
Major External Respiration Variables
Internal Respiration
Major Internal Respiration Variables
Oxygen Transport
Carbon Dioxide Transport
The Respiratory System and Acid-Base Balance

Summary

Review Questions

OBJECTIVES

After studying the chapter, you should be able to:

- Distinguish among and explain the component variables of pulmonary ventilation, external respiration, and internal respiration.
- Identify the conductive and respiratory zones of the respiratory system and compare the functions of the two zones.
- Explain the mechanics of breathing.
- Differentiate between pulmonary circulation and bronchial circulation.
- Describe static and dynamic lung volumes.

- Distinguish between the conditions under which respiratory measures are collected and reported.
- Calculate minute and alveolar ventilation, the partial pressure of a gas in a mixture, the amount of oxygen carried per deciliter of blood, and the arteriovenous oxygen difference.
- Explain how respiration is regulated at rest and during exercise.
- Explain how oxygen and carbon dioxide are transported in the circulatory system and how oxygen is released to the tissues.
- Explain the role of respiration in acid-base balance.

Introduction

The common denominator for all sports, exercise, and physical activity is muscle action. For muscles to be able to act, energy must be provided. The first link in the chain supplying a large portion of this energy is respiration, which provides oxygen to and removes carbon dioxide from the body. In reality, most of us take respiration for granted (we don't have to think to do it), and we are unaware of it until something like hard exercise increases the sensation. Thus, it comes as quite a surprise to many exercise physiology students that respiration is relatively complex physiologically. In addition, there are a great many abbreviations used for the multitude of respiratory terms. The reader is referred to the two pages entitled "Commonly Used Symbols and Abbreviations" directly inside the front of the textbook as a quick reference to help keep these terms straight. In addition, summaries of the variables important for the major divisions of respiration (pulmonary ventilation, external respiration, and internal respiration) are provided at the end of the appropriate sections of text.

Although it is typical to think of respiration as being the same as breathing and/or ventilation, technically, it is not. **Figure 9.1** presents an overview of respiration. The volume of air flowing into the lungs from the external environment through either the nose or the mouth is called **pulmonary ventilation**. Ventilation is accomplished by breathing, the alternation of inspiration and expiration that causes the air to move. The actual exchange of the gases oxygen (O₂) and carbon dioxide (CO₂) between the lungs

and the blood is known as **external respiration**. At the cellular level, oxygen and carbon dioxide gases are again exchanged; this exchange is called **internal respiration**. The cardiovascular system transports the gases between the sites of external and internal respiration. **Cellular respiration** is the utilization of oxygen by the cells to produce energy with carbon dioxide as a by-product. Cellular respiration includes both aerobic (with O₂) and anaerobic (without O₂) energy production and is discussed in [Chapter 2](#). This chapter concentrates on pulmonary ventilation, external respiration, and internal respiration. Technically, the respiratory system is responsible for only pulmonary and external respiration, but its mission is not accomplished without internal respiration ([Marieb and Hoehn, 2019](#)).

Pulmonary Ventilation The process by which air is moved into and out of the lungs.

External Respiration The exchange of gases between the lungs and the blood.

Internal Respiration The exchange of gases between the blood and the tissues at the cellular level.

Cellular Respiration The utilization of oxygen by the cells to produce energy.

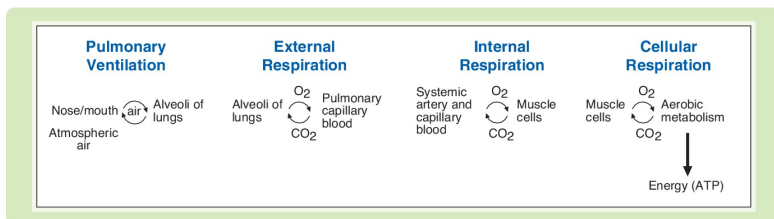


Figure 9.1 Overview of Respiration.

Respiration consists of four separate processes. The first is

pulmonary ventilation, in which air is moved into and out of the body. The second, external respiration, involves the exchange of oxygen and carbon dioxide between the lungs and the blood. The third is internal respiration, which involves the exchange of oxygen and carbon dioxide at the cellular or tissue level. Finally, cellular respiration is the utilization of oxygen to produce energy, which also produces carbon dioxide as a by-product.

Structure of the Pulmonary System

The respiratory system consists of two major portions: (1) the conductive zone, which transports the air to the lungs, and (2) the respiratory zone, where gas exchange takes place.

The Conductive Zone

The basic structure of the conductive zone is shown in **Figure 9.2**. The structures from the nose or mouth to the terminal bronchioles comprise the *conductive zone*. The primary role of the conductive zone is to transport air. Because no exchange of gases takes place here, this zone is also called *anatomical dead space*. As a general guideline, the amount of anatomical dead space can be estimated as 1 mL for each 1 lb of “ideal” body weight ([Slonim and Hamilton, 1976](#)). Hence, a 130-lb female who is at her ideal weight has an estimated 130 mL of anatomical dead space. However, if this individual were to gain 20 lb, she would still have the same anatomical dead space and that estimate would remain at 130 mL. Anatomical dead space is important in determining the alveolar ventilation, as discussed later.

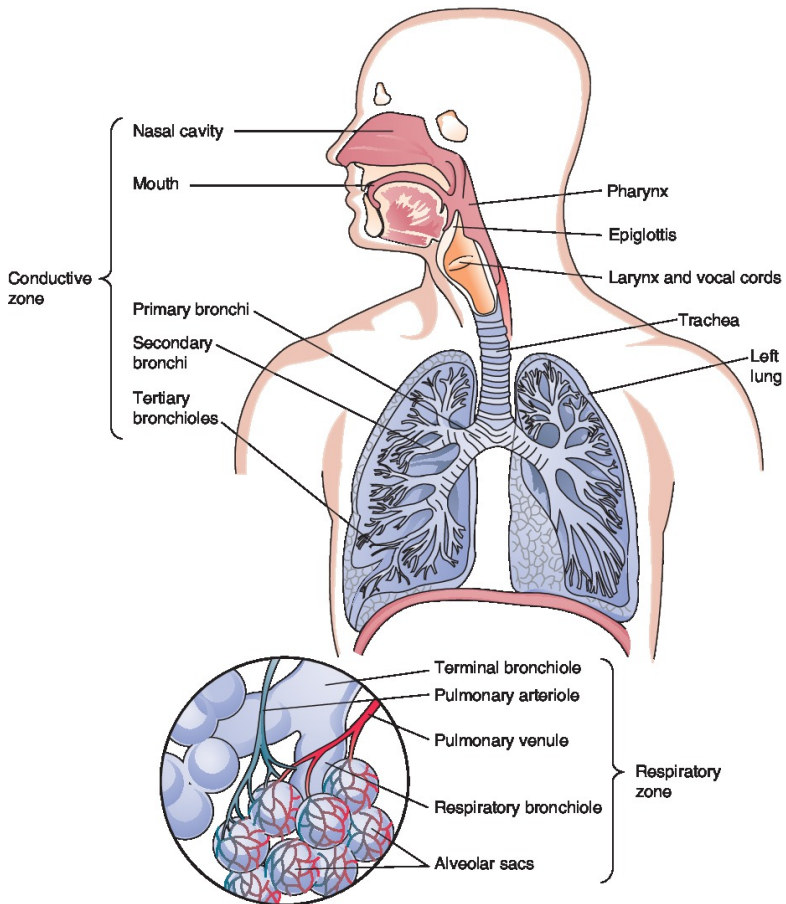


Figure 9.2 Anatomy of the Pulmonary System.

The pulmonary system is divided into two zones: the conductive zone transports air to the lungs and the respiratory zone where gas exchange takes place.

The second important role of the conductive zone is to warm and humidify the air. By the time the air reaches the lungs, it will be warmed to body temperature (normally $\sim 37^{\circ}\text{C}$) and will be 99.5% saturated with water vapor. This protective mechanism helps maintain core body temperature and protects the lungs from injury (Slonim and Hamilton, 1976). The warming and humidifying of air is easily accomplished over a wide range of environmental temperatures under resting conditions, when the

volume of air transported is small and the air is inhaled through the nose. During heavy exercise, however, large volumes of air are inhaled primarily through the mouth, thus bypassing the warming and moisturizing sites of the nose and nasal cavity. As a result, the mouth and the throat may feel dry. If heavy exercise takes place in cold weather (especially at subzero temperatures), dryness increases and throat pain may be felt. These uncomfortable feelings are not a symptom of freezing of the lungs; rather, they are the result of the drying and cooling of the upper airway. The lower portions of the conductive zone still moisturize and warm the air sufficiently before it reaches the lungs. A scarf worn across the mouth will trap moisture and heat from the exhaled air and thereby decrease or eliminate the uncomfortable sensations.

The third role of the conductive zone is to filter the incoming air. The nasal cavity, pharynx, larynx, trachea, and bronchial system are all lined with ciliated mucous membranes (**Figure 9.2**). These membranes with their hair-like projections trap impurities and foreign particles (particulates) that are inhaled. Both smoke and environmental air pollutants diminish ciliary activity and can ultimately destroy the cilia. Air pollution is discussed in the following chapter.

The Respiratory Zone

The *respiratory zone* consists of the respiratory bronchioles, the alveolar ducts, alveolar sacs (grape-like clusters), and the alveoli (**Figure 9.2**). The alveoli are the actual site of gas exchange between the pulmonary system and the cardiovascular system. At birth, humans have about 24 million alveoli. This number increases to about 300 million by 8 years of age and remains constant until age 30, when it begins a gradual decline. Although each individual alveolus is small, only about 0.2 mm in diameter, collectively, the alveoli in a young adult have a total surface area of 50–100 m² (West, 2005). This area would cover a badminton court or even a tennis court if flattened out. Despite this large surface area, the lungs weigh only about 2.2 lb (1 kg). The volume of the respiratory zone is about 2.5–3.0 L · min⁻¹ (West, 2005). The membrane between the alveoli and the capillaries is actually composed of five very thin layers, two of which are the

endothelial cells of the alveoli and the capillaries. The endothelium of the alveoli produces a substance called surfactant that reduces surface tension and helps prevent alveoli from collapsing (Seifter et al., 2005). Despite the number of layers, the thickness is less than the paper this book is printed on, and gas exchange takes place easily (West, 2005).

In addition to the anatomical dead space where no exchange takes place, some alveoli have no capillary blood supply or the capillaries are pathologically blocked and therefore cannot participate in gas exchange; these alveoli make up an *alveolar dead space*. The anatomical plus the alveolar dead space combined makes up the *physiological dead space*. Because alveolar dead space is minimal in healthy individuals, the physiological dead space is only slightly larger than the anatomical dead space (West, 2005).

Mechanics of Breathing

The movement of air into the lungs from the atmosphere depends on two factors: pressure gradient (ΔP) and resistance (R). The relationship between these factors is expressed by the equation for *airflow* (\dot{V}):

$$\text{airflow (L} \cdot \text{min}^{-1}) = \frac{\text{pressure gradient (mmHg)}}{\text{resistance (R unit)}}$$

or

$$9.1 \quad \dot{V} = \frac{\Delta P}{R}$$

A *pressure gradient* is simply the difference between two pressures. Difference is represented by the Greek capital letter delta: Δ . The larger the differences in pressure, the larger the pressure gradient is. Gases—in this case, air, which is a mixture of gases—move from areas of high pressure to areas of low pressure.

Resistance is the sum of the forces opposing the flow of the gases. About 20% of resistance to airflow is caused by tissue

friction as the lungs move during inspiration and expiration. The remaining 80% is due to the friction between the gas molecules and the walls of the airway (airway resistance) and the internal friction between the gas molecules themselves (viscosity). Airway resistance is determined by the size of the airway and the smoothness or turbulence of the airflow.

As [Equation 9.1](#) shows, in order for air to flow, the pressure gradient must be greater than the resistance to the flow. Thus, for inspiration to take place, pressure must be higher in the atmosphere than in the lungs; for expiration, pressure in the alveoli of the lungs must be higher than in the atmosphere. **Figure 9.3** shows how the inspiratory pressure gradient is created.

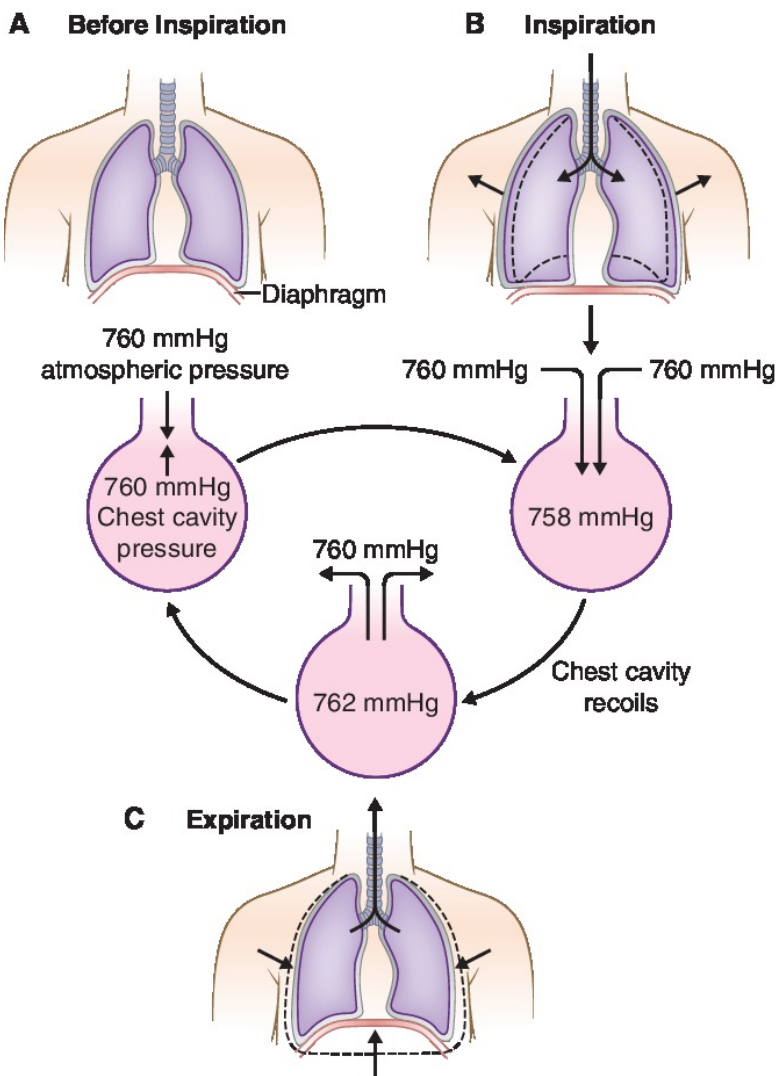


Figure 9.3 Inspiratory and Expiratory Pressure Gradients.

Inspiration and expiration are accomplished through pressure gradients. **A.** The respiratory musculature is relaxed, and the atmospheric pressure equals the chest cavity pressure, so no air movement occurs. **B.** The external intercostals and diaphragm contract, moving the ribs up and out and the diaphragm down, respectively. This muscle

action enlarges the chest cavity laterally, anteroposteriorly, and downward, increasing the volume. Chest cavity pressure is therefore lower than atmospheric pressure, and air flows in. C. The inspiratory muscles relax, and the chest cavity recoils, creating a pressure higher than atmospheric pressure. Air flows out. The respiratory cycle then begins again.

A key to understanding **Figure 9.3** is Boyle's law. *Boyle's law* states that the pressure of a gas is inversely related to its volume (or vice versa) under conditions of constant temperature: low pressure is associated with large volume, and high pressure is associated with small volume.

For pulmonary ventilation, an increase in chest cavity volume is accomplished by muscle contraction for inspiration. This increase in volume leads to an internal lung pressure decrease according to Boyle's law. As a result, a negative pressure exists in the chest cavity relative to the atmosphere outside the body. Thus, a pressure gradient has been created. Air flows into the chest cavity in an attempt to equalize this pressure difference. The volume change per unit of pressure is called *compliance* (West, 2005). Healthy lungs tend to have high compliance that, in turn, favors efficient ventilation.

The main inspiratory muscle is the dome-shaped diaphragm. With neural stimulation, the diaphragm contracts and moves downward, elongating the chest cavity (**Figure 9.3B**). In normal resting breathing, the diaphragm moves about 1 cm; in heavy or forced breathing, it may move as much as 10 cm (West, 2005). During exercise, the chest cavity is further enlarged by the action of the external intercostal muscles and others, known collectively as the accessory muscles, which elevate the rib cage and cause expansion both laterally (side to side) and anteroposteriorly (front to back). The extent of accessory muscle activity and the resultant drop in pressure depend on the depth of the inspiration.

FOCUS ON APPLICATION

Face Masks

Since the outbreak of the COVID-19 pandemic, wearing a face mask has become commonplace for people who work or exercise in public places. There are also professions that require face masks on a routine basis, such as health care workers and EMS response crews. Face masks help to mitigate the spread of diseases by filtering both inspired and expired air. However, wearing a face mask may feel restrictive to some people, especially when performing high levels of physical work. This raises questions about the effects of face masks on exercise. Do they limit exercise performance? Do they change the physiology of exercise? Are they safe to wear during strenuous work? Recent research has explored the effects of face masks on exercise performance and physiology.

1. Shaw et al. (2020) conducted a randomized, counterbalanced crossover design study to examine the effects of wearing a surgical mask, a cloth mask, or no mask on vigorous exercise performance. A total of 14 young and healthy participants (7 males and 7 females) completed maximal cycle ergometry tests and showed no difference in time to exhaustion or peak power among the three conditions. Additionally, there were no differences in arterial oxygen saturation, tissue oxygenation index, rating of perceived exertion, or heart rate between masked and unmasked exercise at any point during the test.
2. In another study using a cycle ergometry ramp protocol, Epstein et al. (2020) assessed the performance of 16 young males on a maximal exercise test while wearing no mask, wearing a surgical mask, and wearing an N95 respirator. No differences were seen in heart rate, respiratory rate, blood pressure, oxygen saturation, or time to exhaustion. There was, however, significantly higher end-tidal carbon dioxide for trials using the N95 respirator, which increased with exercise intensity.

Both studies concluded that face masks are safe to wear during exercise and result in little to no changes in exercise performance, feeling of exertion, or exercise physiology. It should be noted that recent research has included only healthy young adults, and for safety purposes, people with lung diseases and older populations should consult their doctor before performing strenuous exercise while wearing a face mask.



Sources: Epstein et al. (2020); Shaw et al. (2020)

These changes in the chest cavity volume transfer themselves to the lungs through the pleura. Pleurae are thin, double-layered membranes that line both the chest cavity (the inner surfaces of the thorax, sternum, ribs, vertebrae, and diaphragm) and the external lung surfaces. The portion covering the chest cavity is called the parietal pleura; the portion covering the external lung surfaces is called the visceral or pulmonary pleura. A fluid secreted by the pleura fills the space between the pleurae (the intrapleural space), allowing the lungs to glide smoothly over the chest cavity walls. It also causes the parietal and the pulmonary pleurae to adhere to each other in the same way that two pieces of glasses are held together by a thin film of water. Because of this adhesion, the lungs themselves move when muscle actions

move the chest cavity ([Hall and Hall, 2021](#); [Martin et al., 1979](#)).

During normal resting conditions, expiration occurs simply because the diaphragm and other inspiratory muscles relax. When these muscles relax, both the lungs and the muscles, which are highly elastic, recoil to their original positions. This elastic recoil decreases lung volume and thus creates a pressure inside the chest cavity that is higher than the atmospheric pressure. As the chest cavity volume decreases, the intrathoracic pressure increases slightly above that of the atmosphere. The result is that the air moves out of the lungs into the atmosphere. The pressures equalize again, and the cycle repeats with the next inspiration. A complete respiratory cycle includes both inspiration and expiration.

During heavy breathing, as in exercise, expiration is an active process. The primary expiratory muscles are the abdominals and the internal intercostals. The abdominals (rectus abdominis, the obliques, and the transverse abdominus) push the abdominal organs—and hence the diaphragm—upward; the internal intercostals pull the ribs inward and down. This decrease in chest volume increases intrathoracic pressure more quickly than passive elastic recoil alone, and the air is forced out of the lungs faster.

The pleurae also serve a purpose during expiration. Pressure in the intrapleural space fluctuates with breathing in a way that parallels pressure within the lungs. However, the intrapleural pressure is always negative (approximately -4 mmHg) relative to the intrapulmonary (lung) pressure. This negative pressure protects the lungs from collapsing. If the intrapleural pressure were equal to the atmospheric pressure, the lungs would collapse at the end of expiration because of the elastic recoil.

Because muscle activity is involved during the respiratory cycle of inhalation and exhalation, energy is consumed. During rest, however, this energy consumption (restricted to inspiratory muscles) amounts to only 1–2% of the total body energy expenditure in nonsmokers ([Pardy et al., 1984](#)).

Respiratory Circulation

The lung has two different circulatory systems. *Pulmonary circulation* serves the external respiratory function, and *bronchial circulation* supplies the internal respiratory needs of the lung tissue (**Figure 9.4**).

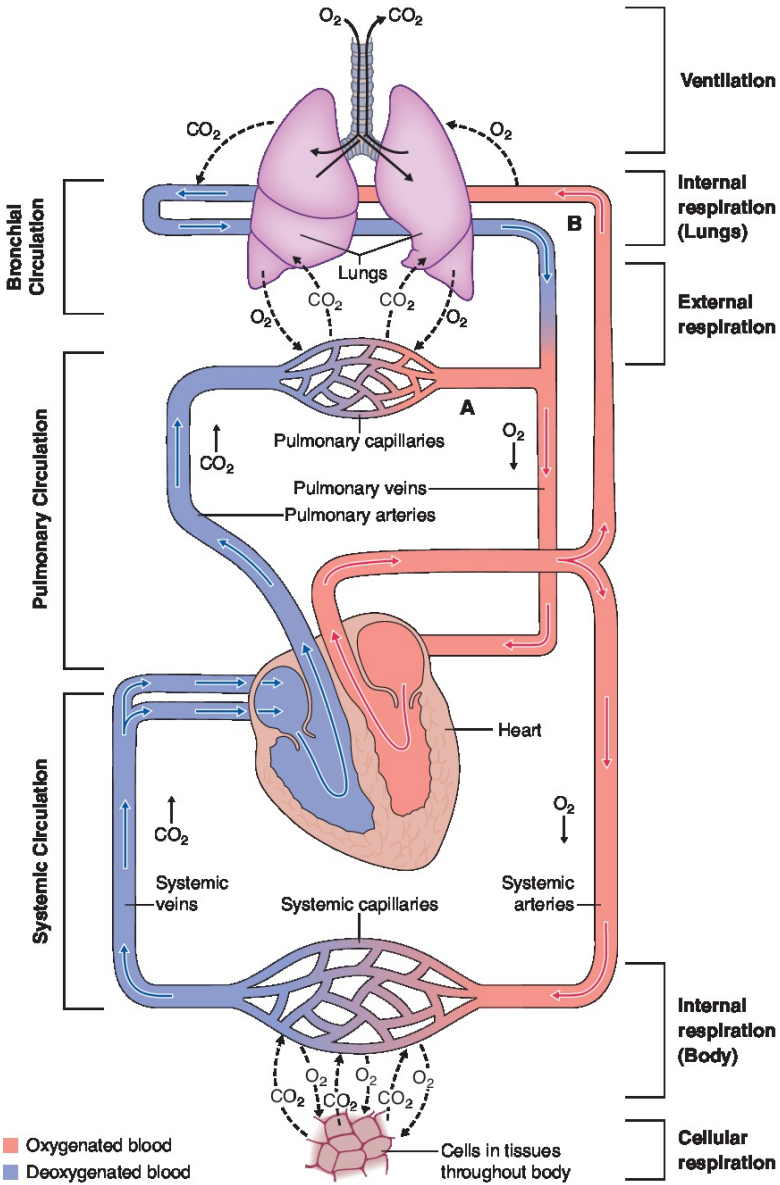


Figure 9.4 Lung Circulatory Systems.

Pulmonary circulation (**A**) serves the process of external respiration, picking up oxygen for and unloading carbon dioxide from the body as a whole at the alveoli. Bronchial circulation (**B**) serves the process of internal respiration, unloading oxygen to and picking up carbon dioxide from the lung tissue. **Source:** Modified from [Germann and Stanfield \(2002\)](#).

The structure of the pulmonary circulation parallels the divisions of the structures in the conductive zone, branching in a tree-like manner called *arborization* (**Figure 9.5**). Arborization ends in a dense alveolar capillary network, blanketing most alveoli, to greatly increase the exchange of respiratory gases. The capillary blood flow through this network is called **perfusion of the lung** ([Hall and Hall, 2021](#); [West, 2005](#)).

Perfusion of the Lung Pulmonary circulation, especially capillary blood flow.

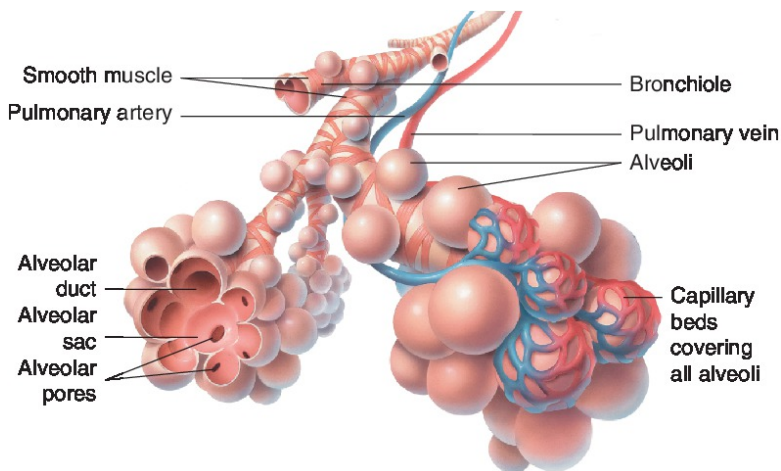


Figure 9.5 Pulmonary Circulation Showing Arborization around the Alveoli.

Source: Asset provided by Anatomical Chart Co.

The pulmonary artery exits the right ventricle of the heart and gives rise to the capillary network in the lungs (**Figure 9.4A**). The pulmonary vein originates from this capillary network and enters the heart at the left atrium. As with pulmonary airflow and the rest of the circulatory system, blood flows through this circuit due to differences in pressure that produce a pressure gradient large enough to overcome resistance to the flow. Normal pulmonary artery blood pressure is low, only 25/10 mmHg, but venous pulmonary blood pressure is even lower, only 7 mmHg. Although this pressure gradient between the pulmonary artery and vein is not large, it is enough to bring about the blood flow. Because of the low gradient, however, gravity affects the pulmonary circulation more than the systemic or total body circulation. The lowest portion of the lungs, therefore, is perfused best. The portion of the lungs that is best perfused with blood is also ventilated best ([Hall and Hall, 2021](#); [Leff and Schumacker, 1993](#); [Martin et al., 1979](#)). Which portion it is varies with the body posture.

The bronchial circulation (**Figure 9.4B**) consists of relatively small systemic arteries that originate from the descending portion of the aorta, called the thoracic artery, travel through the lungs, and return as veins that empty into the pulmonary venous system. Thus, not all of the pulmonary venous blood is fully oxygenated ([Slonim and Hamilton, 1976](#)).

Minute Ventilation/Alveolar Ventilation

The amount of air inspired or the amount of air expired in 1 minute is known as **minute ventilation or minute volume**. The most common units of measurement are liters per minute ($\text{L} \cdot \text{min}^{-1}$) and milliliters per minute ($\text{mL} \cdot \text{min}^{-1}$). Inspired minute

ventilation is symbolized as \dot{V}_I , where V is volume, the “dot” indicates per unit of time, and the subscript upper case I stands for inspired. The symbol for expired ventilation with the subscripted upper case E, indicates expired rather than inspired air.

Minute Ventilation or Minute Volume (\dot{V}_I or \dot{V}_E) The amount of air inspired or expired each minute, or the pulmonary ventilation rate per minute; calculated as tidal volume times frequency of breathing.

Minute ventilation depends on **tidal volume (V_T)**, the amount of air inhaled or exhaled per breath, and the frequency (f) of breaths per minute ($\text{br} \cdot \text{min}^{-1}$).

Tidal Volume (V_T) The amount of air that is inspired or expired in one breath.

The equation is:

$$\begin{aligned} &\text{minute ventilation (mL} \cdot \text{min}^{-1}\text{)} \\ &= \text{tidal volume (mL} \cdot \text{br}^{-1}\text{)} \times \text{frequency (br} \cdot \text{min}^{-1}\text{)} \\ &\text{or} \end{aligned}$$

$$9.2 \quad \dot{V}_E = V_T \times f$$

Milliliters per minute are then commonly converted to liters per minute by dividing by 1,000.

At rest, a normal young adult breathes at a frequency of 12–15 times per minute and has a tidal volume of 400–600 mL. Children ventilate at a much faster rate but with a smaller tidal volume.

Example

Compute \dot{V}_E when $f = 15 \text{ br} \cdot \text{min}^{-1}$ and $V_T = 400 \text{ mL}$. You should set up and solve the equation as follows:

$$\begin{aligned} \dot{V}_E &= (400 \text{ mL} \cdot \text{br}^{-1}) \times (15 \text{ br} \cdot \text{min}^{-1}) \\ &= 6,000 \text{ mL} \cdot \text{min}^{-1} \end{aligned}$$

$$\frac{6,000 \text{ mL} \cdot \text{min}^{-1}}{1,000 \text{ mL} \cdot \text{L}^{-1}} = 6.0 \text{ L} \cdot \text{min}^{-1}$$

Because minute ventilation represents the total amount of air moved into or out of the lungs per minute, it includes the portion of air that fills the conduction zone. Thus, minute ventilation does not represent the amount of air that is available for gas exchange. The amount of air that is available for gas exchange is termed **alveolar ventilation** (or *anatomical effective ventilation*).

Alveolar ventilation (\dot{V}_A) takes into account tidal volume (V_T), dead space (V_D), and the frequency (f) of breathing. The equation for calculating \dot{V}_A is:

Alveolar Ventilation (V_A) The volume of air available for gas exchange; calculated as tidal volume minus dead space volume times frequency.

$$\begin{aligned} &\text{alveolar ventilation (mL} \cdot \text{min}^{-1}\text{)} \\ &= [\text{tidal volume (mL} \cdot \text{br}^{-1}\text{)} - \text{dead space} \\ &\quad \text{(mL} \cdot \text{br}^{-1}\text{)}] \times \text{frequency (br} \cdot \text{min}^{-1}\text{)} \end{aligned}$$

or

$$9.3 \quad \dot{V}_A = (V_T - V_D) \times f$$

Typically, approximately 70% of \dot{V}_I reaches the alveoli for gas exchange. The ratio of anatomical dead space (V_D) to tidal volume (V_T) is abbreviated as V_D/V_T . This is not a mathematical constant because both factors can change. The dead space actually increases from some stretching of the respiratory passages as breathing becomes deeper and the deeper breathing increases the inspired volume of air. The increase in dead space is only a very small portion of the increase in the volume of air inspired.

Major Pulmonary Ventilation Variables

In summary, the major variables for pulmonary ventilation are minute ventilation (\dot{V}_E or \dot{V}_I), tidal volume (VT), frequency of breathing (f), and the ratio of dead space to tidal volume (V_D/VT). Alveolar ventilation (\dot{V}_A) is considered part of external respiration.

Example

Calculate alveolar ventilation using the values of f and VT given in the previous example, for a female who is at her ideal weight of 130 lb. Also calculate the percent of \dot{V}_I that is available for gas exchange. This problem becomes:

$$\begin{aligned}\dot{V}_A &= [(400 \text{ mL} \cdot \text{br}^{-1}) - (130 \text{ mL} \cdot \text{br}^{-1})] \\ &\quad \times (15 \text{ br} \cdot \text{min}^{-1}) \\ &= 4,050 \text{ mL} \cdot \text{br}^{-1} \div 1,000 \text{ mL} \cdot \text{L}^{-1} \\ &= 4.05 \text{ L} \cdot \text{min}^{-1}\end{aligned}$$

The percentage is 67.5% $[(4,050 \text{ mL} \cdot \text{min}^{-1}) / (6,000 \text{ mL} \cdot \text{min}^{-1})]$.

As with minute ventilation, alveolar ventilation can be calculated from either \dot{V}_E or \dot{V}_I . The alveolar dead space is so negligible in normal healthy individuals that it does not need to be considered in these calculations.

To be sure you understand the implications of this concept, complete the problems in the [Check Your Comprehension 1](#) box.

CHECK YOUR COMPREHENSION 1

1. Given two breathing patterns, A and B, in the accompanying table, calculate the alveolar ventilation.

On the basis of your calculations, is it better to breathe fast and shallowly or slowly and deeply?

| Breathing Pattern | | |
|---------------------------------------|-----|-----|
| | A | B |
| \dot{V}_E (L · min ⁻¹) | 6 | 6 |
| V_T (mL · br ⁻¹) | 600 | 200 |
| f (br · min ⁻¹) | 10 | 30 |
| V_D (mL · br ⁻¹) | 150 | 150 |
| \dot{V}_A (mL · min ⁻¹) | ? | ? |

2. Individuals learning the front crawl swimming stroke are often reluctant to exhale air while their faces are in the water. They then try to inhale more air when their faces are turned to the side or to both exhale and inhale in the short time available during the head turn. Why is this not an effective breathing technique?
3. What is the effect of using a snorkel on the dead space? What implication does this effect have for breathing through a snorkel?

Check your answers in Appendix C.

Measurement of Lung Volumes

Lung volumes can be measured either statically or dynamically. Static lung volumes are anatomical measures, are independent of time, and thus do not measure flow. Dynamic lung volumes depend on time and measure both airflow and air volume.

Static Lung Volumes

Take as deep a breath as you can. At this point, your lungs

contain the maximum amount of air they can hold. This amount of air is called **total lung capacity (TLC)**. TLC can be divided into four volumes and three other capacities, as depicted in **Figure 9.6**. Note that the four capacities are combinations of two or more volumes. All of these volumes and capacities have clinical significance, but only those important in the study of exercise physiology are discussed briefly here.

Total Lung Capacity (TLC) The greatest amount of air that the lungs can contain.

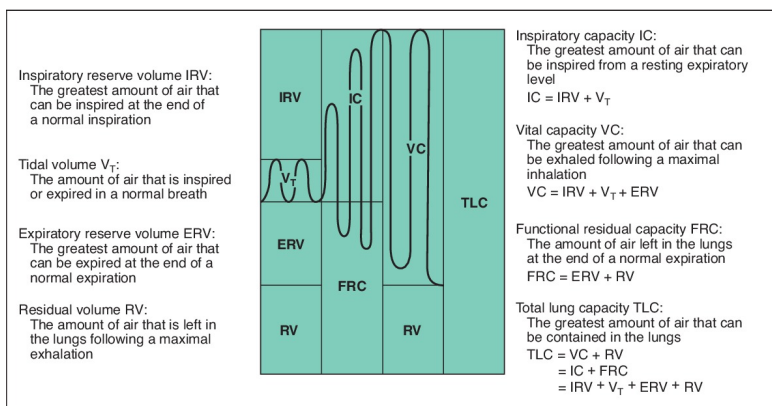


Figure 9.6 Static Lung Volume Spirogram.

TLC can be subdivided into four volumes (IRV, V_T , ERV, and RV) and into three other capacities (IC, FRC, and VC). From the normal resting depth of inspiration and expiration (indicated by the *wavy line* moving up and down, respectively, in the box labeled V_T), both inspiration and expiration can be expanded into the IRV and ERV until all possible air is inhaled and exhaled (VC). The RV remains in the lungs at all times.

As mentioned previously, tidal volume (V_T) is the amount of air either inhaled or exhaled in a single breath. A normal V_T inflates the lungs to about half of the TLC when sitting erect or standing and only about a third when lying supine ([Slonim and](#)

Hamilton, 1976). When the demand for energy increases during exercise, V_T increases by expanding into both the inspiratory reserve volume (IRV) and expiratory reserve volume (ERV). Thus, the limits of vital capacity ($VC = IRV + V_T + ERV$) represent the absolute limit of the tidal volume increase during exercise.

Residual volume (RV) is the amount of air left in the lungs following a maximal exhalation. This leftover air is important because it allows for a continuous gas exchange between the alveoli and the capillaries between breaths. If all air were forced out of the lungs, no gas would be available for exchange. During exercise, when V_T expands, the functional residual capacity (FRC) helps maintain a smooth exchange, in the following way. The V_T can and does expand into both the IRV and the ERV, but it expands more into the IRV than the ERV, leaving a relatively large FRC intact. This large FRC dilutes the gas changes (the decrease in oxygen and increase in carbon dioxide) caused by the increased energy production and expenditure of exercise. By reducing fluctuation, the FRC stabilizes and smoothes the gas exchange.

Residual Volume (RV) The amount of air left in the lungs following a maximal exhalation.

Despite its beneficial physiological aspects, RV presents a measurement difficulty. When body composition is determined by hydrostatic (underwater) weighing (see Chapter 7), RV must be measured or estimated. Residual air makes the body more buoyant; if not accounted for, it would reduce the underwater weight. The less an individual weighs underwater, the higher the measured percentage of body fat. Thus, if RV were not accounted for, it would erroneously increase the measurement of fat. This is the most common reason for determining RV in exercise physiology. Sometimes, RV is estimated from VC; however, it is more accurate if measured directly either outside or inside the tank (Wilmore, 1969). RV is estimated as 24% of VC for males and 28% of VC for females.

Vital capacity (VC) is simply the largest amount of air that can be exhaled following a maximal inhalation. The common way

of testing VC is to ask the individual to inhale maximally and then forcefully exhale all of the air as quickly as possible. Because the exhalation is forced, the designation forced vital capacity, FVC, is used.

Vital Capacity (VC) The greatest amount of air that can be exhaled following a maximal inhalation.

Dynamic Lung Volumes

When volumes are measured at specified time intervals (usually 1 and 3 seconds) during a forced VC test, the name is changed to *forced expiratory volume*, specifically, FEV₁ and FEV₃. This provides information not only about the total volume of air moved but also the rate of flow. Normal healthy individuals should be able to exhale at least 80% of their FVC in 1 second; this measurement is labeled FEV₁. FEV₁ values below 65–70% indicate moderate to severe restriction to airflow (Adams, 1994).

The second commonly measured dynamic lung volume is a test of ventilatory capacity called *maximal voluntary ventilation* (MVV). In this test, a timed maximal ventilation of either 12 or 15 seconds is recorded and then multiplied by 5 (if 12 seconds) or 4 (if 15 seconds) to extrapolate to the volume that could be ventilated in 1 minute. This value is usually higher than what can actually be achieved during exercise in untrained individuals, but it gives a rough estimate of exercise ventilation potential. Low values may reflect airway resistance and poorly conditioned or poorly functioning ventilatory muscles.

Both FEV and MVV tests are often used as screening tests before maximal exercise tests of oxygen consumption.

Spirometry

All of the lung volumes and capacities described previously—except for TLC, FRC, and RV—can be measured using a spirometer. Historically, most spirometers had an inverted container, called a bell that fit inside another container usually filled with water. When air is exhaled into the tube, the bell is

pushed up; inhalation moves the bell down. In this way, the volumes of air inspired and expired can be measured, and the various volumes, capacities, and flow rates can be calculated. A spirometer that measures air volumes over water is called a wet spirometer. Newer dry spirometers are often digital and do not use water (**Figure 9.7**). Spirometry measures can also be obtained by many metabolic carts that assess oxygen consumption (see [Chapter 4](#)).



Figure 9.7 A Spirometer.

Gas Dilution

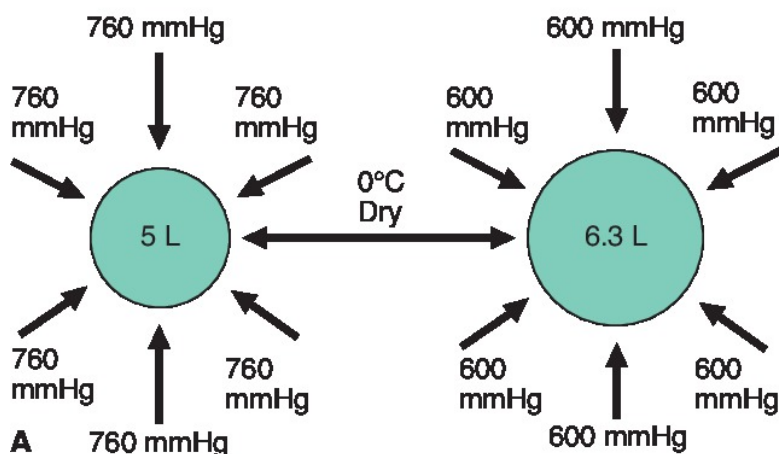
TLC, FRC, and RV cannot be measured by simple spirometry because these involve air that cannot be exhaled voluntarily from the lungs. Thus, it is necessary to determine the volume of air that remains in the lungs. The most common technique for this measurement is gas dilution. One technique involves the dilution of an inert, insoluble, foreign gas such as helium (He). A second technique involves the dilution of medical grade oxygen and the measurement of nitrogen (N_2) and is called the nitrogen washout technique. In each case, the participant inhales a known volume of either helium or oxygen and then rebreathes the gas mixture. Helium ultimately equilibrates with the gases in the lungs and

spirometer, and calculations are based on the dilution of the original helium mixture. Oxygen dilutes the original nitrogen concentration in the lungs, and calculations are based on these values.

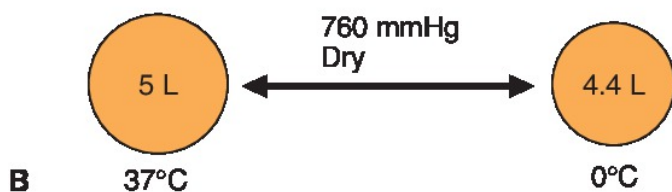
Standardization

The respiratory measurements detailed above are performed under ambient or atmospheric conditions. This means that the temperature measured is the temperature in the room or in the spirometer just before testing, and the pressure is the barometric pressure of the room. Because the air is exhaled from inside the human body, which is a wet environment, the air is saturated with water vapor. These measurements are therefore designated as *ATPS*: ambient (A) temperature (T) and pressure (P) saturated (S). Because *ATPS* volumes vary by environmental conditions, they must be converted to standardized conditions for purposes of comparison and assessment. Standardization is based on the known effects of pressure, temperature, and water vapor on gas volumes. **Figure 9.8** illustrates these three effects, which are as follows:

Effect of Pressure on Volume



Effect of Temperature on Volume



Effect of Water Vapor on Volume



Figure 9.8 Effects of Pressure, Temperature, and Water Vapor on Air Volume.

A. The volume of a given quantity of a gas is inversely related to the pressure exerted on it, if the temperature remains constant (Boyle's law). **B.** The volume of a given quantity of gas is directly related to the temperature of the

gas if the pressure remains constant (Charles' law). C. The volume of a gas increases as the content of water vapor increases.

FOCUS ON RESEARCH

The Relationship between Forced Expiratory Volume and Health

Evidence is mounting that forced expiratory volume in 1 second (FEV₁) is related to health.

An epidemiological study by [Schunemann et al. \(2000\)](#) explored the link between low values of FEV₁ (expressed as a *percentage of predicted FEV₁* [FEV₁%pred]) and mortality (death from all causes).

A randomly selected sample of 554 adult men and 641 adult women were part of a 29-year follow-up. During that time, 302 (54.5%) of the men and 278 (43.4%) of the women died. Only 39 of these deaths (29 males and 10 females) were directly attributed to respiratory disease. Nevertheless, pulmonary function was a significant predictor of all-cause mortality. Each 1% increase in FEV₁%pred (pred = predicted) was associated with a 1–1.5% decrease in all-cause mortality. The risk of an early death for individuals scoring in the lowest quintile (≤ 80.2 FEV₁%pred for males and ≤ 80.5 FEV₁%pred for females) was approximately twice as great (2.24 for males and 1.81 for females) as the risk in the highest quintile (≥ 108.8 FEV₁%pred for males and ≥ 113.6 FEV₁%pred for females).

The reasons for these results are unknown. It was initially thought that FEV₁%pred was simply a proxy for smoking status, but other research has shown that this association is independent of smoking status. Possible explanations for the association include the following:

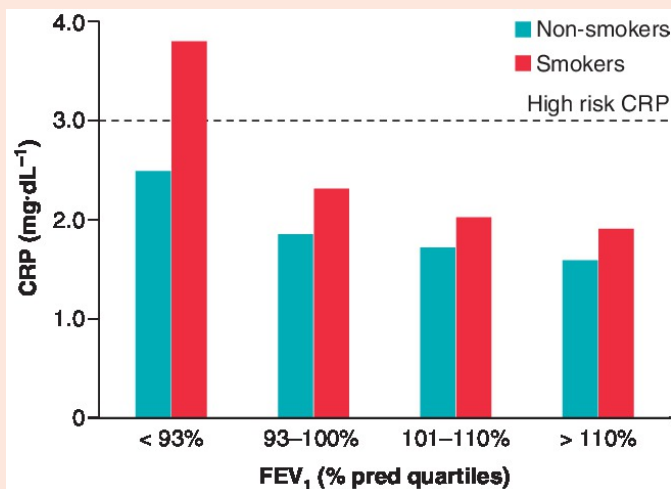
1. Impaired pulmonary function could lead to a decreased

tolerance against environmental toxins.

2. Oxidative stress (see the Focus on Application box in [Chapter 5](#) for a discussion of oxidative stress), which is negatively related to FEV1%pred, could adversely affect the overall health status. Conversely, reduced pulmonary function could be an underlying factor responsible for increased oxidative stress.
3. Low FEV1 values may simply adversely affect physical activity patterns, such that inactivity is the true causal factor.

Exploring the idea of environmental toxins, a study by [Aronson et al. \(2006\)](#) investigated whether a decline in lung function (defined as FEV1) in apparently healthy individuals was associated with systemic inflammation as measured by C-reactive protein (CRP). The immune system and inflammation are detailed in [Chapter 22](#), but briefly, the lungs are a major location for immune cell function. During breathing and gas exchange, the lungs must protect themselves against a variety of infectious agents, noxious gases, and various particulates. (Look ahead to [Figure 9.12](#) and note the macrophage immune cell.) Thus, the investigators speculated that lung function could indicate an immune response occurring, along with resultant low-grade inflammation, before any overt disease appeared. Participants were 1,131 individuals without known pulmonary disease. Median values for CRP by FEV1%pred are presented in the graph for smokers and nonsmokers. The lowest quartile (<93% FEV1pred) contained 96 individuals with below normal (defined as <80% FEV1pred) values. The association between CRP and FEV1 was highly significant and remained so even after adjustment for age, sex, body mass index, metabolic abnormalities, and cardiorespiratory fitness level. These results indicate that systemic inflammation may be linked to early declines in pulmonary function.

These combined results may mean that FEV1 can be used as a convenient health assessment tool.



Sources: Aronson, D., I. Roterman, M. Yigia, et al.: Inverse association between pulmonary function and C-reactive protein in apparently healthy subjects. *American Journal of Respiratory and Critical Care Medicine*. 174(6):626–632 (2006); Schunemann, H. J., J. Dorn, B. J. B. Grant, W. Winkelstein, & M. Trevisan: Pulmonary function is a long-term predictor of mortality in the general population: 29-Year follow-up of the Buffalo Health Study. *Chest*. 118(3):656–664 (2000).

1. The volume of a given quantity of gas is inversely related to the pressure exerted on it when the temperature remains constant. This effect is described by Boyle's law and was discussed previously in the section on the mechanics of breathing. As shown in **Figure 9.8A**, when pressure is reduced from 760 to 600 mmHg, 5 L of air expands to 6.3 L. The reverse is also true. If the pressure increases from 600 to 760 mmHg, the volume is reduced from 6.3 to 5.0 L ([Slonim and Hamilton, 1976](#); [West, 2005](#)).
2. The volume of a given quantity of gas is directly related to the temperature of the gas when the pressure remains constant. This relationship is described by *Charles' law*. As shown in **Figure 9.8B**, if the temperature is reduced from 37

to 0°C, the volume of the gas is reduced from 5 to 4.4 L. The reverse is also true. If the temperature rises from 0 to 37°C, the volume also rises from 4.4 to 5.0 L.

3. Water molecules evaporate into a gas, such as air, and are responsible for part of the pressure of that gas. The amount of pressure accounted for by the water vapor is related exponentially to temperature. As shown in **Figure 9.8C**, when a volume of air is converted from saturated to dry air at a constant temperature, the volume is reduced from 5.0 to 4.7 L. Once again, the reverse is also true. If a gas volume goes from dry to wet at a given temperature, the volume increases (Slonim and Hamilton, 1976; West, 2005).

The numbers for temperature and pressure in **Figure 9.8** were not chosen arbitrarily. They are involved in the two standardized conditions to which ATPS lung volumes are converted: BTPS and STPD. The abbreviation *BTPS* means body (B) temperature (T) (37°C), ambient pressure (P), and fully saturated (S) with water vapor. Remember that in its passage through the conduction zone, the air is both warmed and humidified to achieve these values. When converting from ATPS to BTPS, the temperature usually increases and the pressure, adjusted for the effects of temperature on water vapor pressure, decreases. Because of Charles' law (an increase in temperature causes an increase in volume) and Boyle's law (a decrease in pressure causes an increase in volume), the volume expressed as BTPS has to be larger than the volume originally measured as ATPS.

BTPS is typically used when the anatomical space from which the volume of gas originated is of primary importance. Thus, most lung volumes and capacities are conventionally expressed as BTPS.

STPD means standard (S) temperature (T) (0°) and pressure (P) (760 mmHg), dry (D). The STPD volume is smaller than the originally measured ATPS volume because under typical testing conditions, temperature decreases from ATPS (~20°C) to STPD (0°C) and, by Charles' law, so does volume. Unless the testing is done at sea level, pressure increases from ATPS (variable, but around 735–745 mmHg before considering the influence of water vapor) to STPD (760 mmHg), and according to Boyle's law,

volume decreases. Going from wet to dry conditions also decreases the volume.

STPD volumes are used when it is necessary to know the amount of gas molecules present. Inspired or expired minute ventilation is typically converted and reported in STPD conditions, although sometimes it may be expressed as BTPS.

Partial Pressure of a Gas: Dalton's Law

Dry, unpolluted atmospheric air is a mixture of gases including oxygen, carbon dioxide, nitrogen, argon, and krypton. Because the last three are considered inert in humans, they are generally grouped together and labeled simply as nitrogen (the largest component). Thus, air is said to be composed of 79.04% nitrogen, 20.93% oxygen, and 0.03% carbon dioxide. These percentages are also referred to as fractions of each gas and are given in decimal form: 0.7904 nitrogen, 0.2093 oxygen, and 0.0003 carbon dioxide. Another way to describe the composition of a gas mixture such as air is by the partial pressures exerted by each gas (Leff and Schumacker, 1993).

All gases exert pressure. In a mixture of gases, the total pressure is the sum of the pressure of individual gases making up the mixture. Total pressure is standardized to the sea-level barometric pressure of 760 mmHg, but at altitudes above sea level, the actual barometric pressure, P_B , must be used. The **partial pressure of a gas (P_G)** is that portion of the total pressure exerted by any single gas in the mixture. The partial pressure of any gas is proportional to its percentage in the total gas mixture. These relationships are described by *Dalton's law of partial pressures*.

Partial Pressure of a Gas (P_G) The pressure exerted by an individual gas in a mixture; determined by multiplying the fraction of the gas by the total barometric pressure.

The partial pressure of any gas is the product of the total pressure and the fraction of the gas (expressed as a decimal).

Partial pressure of a gas (mmHg) = total pressure
(mmHg) \times fraction of the gas

or

$$9.3 \quad P_G = P_B \times F_G$$

For example, the standardized partial pressure of nitrogen, P_{N_2} , is $760 \text{ mmHg} \times 0.7904 = 600.7 \text{ mmHg}$.

Table 9.1 provides the important information about the partial pressure of different gases in atmospheric air at different altitudes (panels a and b) and in the alveoli (panel c). This table shows that the concentration of oxygen is 20.93% in dry atmospheric air and the concentration of carbon dioxide is 0.03%. If these fractions are multiplied by the barometric pressure (P_B), it provides the P_{O_2} and the P_{CO_2} in dry atmospheric air at sea level. To better understand the effect of changes in P_B at altitude, and changing concentration of gases in the alveoli, calculate the partial pressure of dry atmospheric air at altitude and of alveolar air. Look first at **Table 9.1**, panels (a) and (b). Notice that the gas percentages are the same at sea level and at altitude, in this case 4,268 m (14,000 ft). These percentages remain the same at any altitude. Conversely, the barometric pressure (P_B) is lower at both the selected altitude and any other altitude in comparison with sea level. Exactly how much lower than sea level the P_B is depends on how high the altitude is. At 4,268 m (14,000 ft), P_B is 440 mmHg. As the altitude increases, the barometric pressure decreases. Now, compare **Table 9.1**, columns (a) and (c). Notice that now the barometric pressure values are the same; that is, they are both standardized to 760 mmHg, which is the barometric pressure at sea level. However, the fractions of the gases are different. This difference between alveolar air and atmospheric air occurs because the percentage of oxygen in the alveoli is decreased by diffusion of oxygen into the capillary blood. Similarly, the percentage of carbon dioxide is increased by the diffusion of carbon dioxide out of the capillary blood. In addition, water vapor now occupies a percentage of the total air mixture. At normal body temperature (37°C), water vapor exerts a pressure of 47 mmHg. This value must be subtracted from 760 mmHg prior to multiplying by the fraction of each gas to determine the partial pressure of each gas.

TABLE 9.1 Approximate Gas Partial Pressures in Ambient Air and Selected Ventilatory and Respiratory Sites

| Gases | (a) Dry Atmospheric Air, Sea Level | | | (b) Dry Atmospheric Air, Altitude = (4,268 m) 14,000 ft (Pike's Peak) | | | (c) Alveolar Air, Sea Level | | |
|------------------|---------------------------------------|-----------------------|----------|---|-----------------------|----------|-----------------------------|-----------------------|----------|
| | % | P _a (mmHg) | P (mmHg) | % | P _a (mmHg) | P (mmHg) | % | P _a (mmHg) | P (mmHg) |
| O ₂ | 20.93 | 760 | 159 | 20.93 | 440 | 92.0 | 13.7–14.6 | 760 – 47 = 713 | 104–98* |
| CO ₂ | 0.03 | 760 | 0.3 | 0.03 | 440 | 0.1 | 5.3 | 760 – 47 = 713 | 40 |
| N ₂ | 79.04 | 760 | 600.7 | 79.04 | 440 | 348.0 | 78.7–79.8 | 760 – 47 = 713 | 561–569 |
| H ₂ O | 0.00 | 760 | 0.0 | 0.00 | 440 | 0.0 | | | 47† |
| | | | 760 | | | 440 | | | 760 |

*Owing to minor variations in percentage, the PO₂ in alveolar air is often rounded to 100 mmHg.

†Assumes a body temperature of 37°C and PH₂O of 47 mmHg, which must be subtracted from 760 mmHg before determining PO₂, PCO₂, PN₂.

Knowledge of the partial pressure of gases is important for at least two reasons. The first is that the partial pressures of oxygen (PO₂) in the blood and more specifically the partial pressure of carbon dioxide (PCO₂) help to regulate pulmonary ventilation to bring air into the lungs. The PO₂ and PCO₂ are sensed by various chemoreceptors and influence respiratory centers in the brain (see full discussion later in this chapter). The second reason is that both external and internal respirations depend on pressure gradients.

CHECK YOUR COMPREHENSION 2

Using the information presented in **Table 9.1**, return to **Figure 9.1** and superimpose the partial pressure of O₂ in dry atmospheric air at sea level, and the partial pressure of O₂ in the alveoli at sea level in the appropriate place.

Check your answer in Appendix C.

Regulation of Pulmonary Ventilation

Breathing, or pulmonary ventilation, results from inspiratory and expiratory muscle contraction and relaxation. The muscle action—and therefore the rate, depth, and rhythm of breathing—is controlled by the brain and nervous system and is tightly coupled to the body's overall need for oxygen and the subsequent production of energy and carbon dioxide. The coordinated control of respiratory muscles, especially during exercise, is a very complex process that is not yet fully understood.

The Respiratory Centers

There are two main regions in the brain that function together to control breathing: the pons and the medulla. These are schematically diagrammed in the middle of **Figure 9.9**.

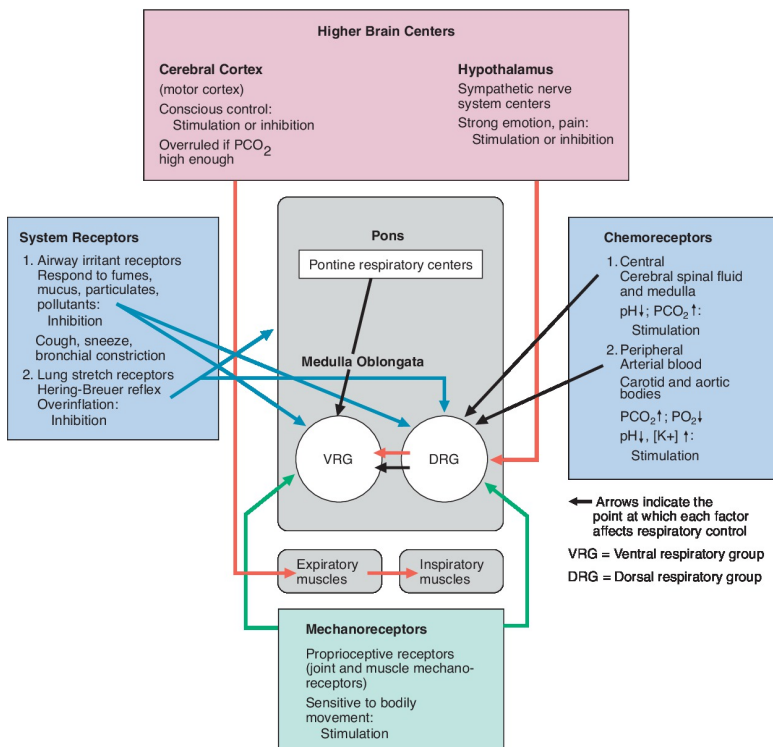


Figure 9.9 Anatomical Sensors and Factors That Influence the Control of Pulmonary Ventilation.

Schematic representation of the respiratory centers that control ventilation and the pathways of action of factors influencing the control of ventilation.

Within the medulla oblongata of the brainstem are two respiratory centers composed of anatomically distinct neural networks. The ventral respiratory group (VRG) is the rhythm-generating and integrative center. It contains neurons that fire during inspiration and other neurons that fire during expiration. Each group inhibits the other. When the inspiratory neurons fire, nerve impulses traveling via motor neurons stimulate the diaphragm and the external intercostal inspiratory muscles to contract. Inhalation occurs when the thoracic cavity is enlarged and intrathoracic pressure decreases. When the VRG's expiratory neurons fire, the motor nerve output stops, the inspiratory muscles relax, and exhalation occurs. Active contraction of the expiratory muscles (internal intercostals and abdominals) occurs when forceful breathing is required, such as during moderate to heavy exercise (Leff and Schumacker, 1993; West, 2005).

Without outside influence and at rest, the VRG center causes a **respiratory cycle** of approximately 2 seconds for inspiration and 3 seconds for exhalation (for a rate of $12\text{--}15 \text{ br} \cdot \text{min}^{-1}$). This normal respiratory rate and oscillating rhythm is known as **eupnea**.

Respiratory Cycle One inspiration and expiration.

Eupnea Normal respiration rate and rhythm.

The dorsal respiratory group (DRG) until recently was thought to act as an inspiratory center. However, it now appears as if the DRG integrates input from peripheral stretch receptors and chemoreceptors and communicates this information to the VRG (Leff and Schumacker, 1993; Marieb and Hoehn, 2019). The pons area of the brainstem contains pontine respiratory centers. They seem to be important for ensuring that the transitions between inhalation and exhalation are smooth. The pontine respiratory

centers transmit impulses to the VRG of the medulla to fine tune breathing rhythms generated by the VRG during such specialized activity as exercise. Inspiratory depth is determined by how actively the respiratory centers stimulate the motor neurons serving the respiratory muscles; this is achieved by varying the number of motor units stimulated. Respiratory rate is determined by how long the stimulation lasts ([Marieb and Hoehn, 2019](#)).

Anatomical Sensors and Factors Affecting Control of Pulmonary Ventilation

The respiratory centers are influenced by a number of factors through a variety of anatomical sensors that are important during exercise ([Dempsey et al., 1985](#)).

Figure 9.9 includes these factors and schematically presents the major areas where each factor operates. Without outside influences, the rate (frequency) of breathing and the depth (tidal volume) of breathing exhibit the greatest changes. A disruption in rhythm rarely occurs except under voluntary control ([Hall and Hall, 2021](#); [Leff and Schumacker, 1993](#); [Whipp et al., 1982](#)).

Higher Brain Centers

Both the hypothalamus and the cerebral cortex can influence breathing. The first operates involuntarily and the second voluntarily.

HYPOTHALAMUS The sympathetic nervous system centers in the hypothalamus are activated by pain or strong emotions. In turn, they send neural messages to the respiratory centers. The reaction can either stimulate or inhibit breathing. Hyperventilation by an individual who is emotionally upset is an example of hypothalamic stimulation. Feeling your breath taken away when you jump into very cold water exemplifies hypothalamic inhibition. This hypothalamic control mechanism may be very important for increasing respiration on initiation of movement and sustaining the increase throughout the duration of an activity ([Eldridge, 1994](#)).

CEREBRAL CORTEX Cerebral control of breathing originates in

the motor cortex. Such voluntary control is important to musicians, singers, and athletes such as swimmers, weight lifters, archers, and shooters, to name just a few. The motor cortex may also operate such that the conscious anticipation of exercise unconsciously increases ventilation. Neural impulses from the motor cortex pass directly to the respiratory muscles, bypassing the control centers in the medulla. Voluntary control is limited, however (Leff and Schumacker, 1993; West, 2005). If you doubt this, take a reading break at the end of this paragraph and jog in place at a fast pace for 3 minutes. Then sit down immediately and try holding your breath for 1 minute. If you can do that, great; but even if you can, you will probably feel a desire to breathe. More than likely, the automatic drive to breathe will overrule your conscious signal not to, and at some point before the end of the minute, you will gasp for air.

Systemic Receptors

The lungs themselves contain several types of receptors that provide sensory information to the respiratory control centers and result in reflex action. Chief among them are irritant receptors and stretch receptors (Leff and Schumacker, 1993; Martin et al., 1979; West, 2005). Both of these receptors are more important for protection than for regulating normal resting or exercise ventilation.

IRRITANT RECEPTORS Irritant receptors within the conduction zone respond to foreign substances such as chemicals, noxious gases, cold air, mucus, dust, and other pollutant particulates. They cause a reflex response. Depending on the irritating substance and the anatomical location, the response may be a cough, a sneeze, or a bronchial constriction, all of which disrupt the normal breathing pattern (Leff and Schumacker, 1993; West, 2005).

STRETCH RECEPTORS Stretch receptors in the airway smooth muscle respond to deep, fast inflation. Increases in the lung volume stimulate the stretch receptors, which send inhibitory impulses to the medullary respiratory centers. As a result, respiratory frequency is slowed because of the increased

expiratory time (inflation reflex). An opposing deflation reflex tends to increase inspiratory activity. These reflexes are called the *Hering-Breuer reflexes*. The threshold for these is quite high. They do not appear to function in adults at rest and play a minor role in exercise only if tidal volume exceeds 1 L (Leff and Schumacker, 1993; West, 2005).

Mechanoreceptors

Specific mechanoreceptors called proprioceptors exist in skeletal muscles (including respiratory muscles) and joint capsules. These receptors provide information to a variety of sites in the brain about movement and body position in space. Input from these receptors probably plays some minor role in the stimulation of respiration during exercise, but they are not involved in respiration during rest (Leff and Schumacker, 1993).

Chemoreceptors

Chemoreceptor sensors that respond to fluctuations in chemical substances important to respiration are found in two major anatomical locations. Central chemoreceptors are located in the medulla oblongata; these are sensitive to an increased partial pressure of carbon dioxide ($\uparrow\text{PCO}_2$) and increased acidity ($\downarrow\text{pH}$). Peripheral chemoreceptors are located in large arteries, specifically, at the aortic body and the carotid body. In addition to being sensitive to $\uparrow\text{PCO}_2$ and $\downarrow\text{pH}$, these receptors are also sensitive to a decrease in the partial pressure of oxygen ($\downarrow\text{PO}_2$) and an increase in the concentration of potassium ions ($\uparrow[\text{K}^+]$). Each of these factors is explained in the following subsections.

INFLUENCE OF PO_2 The peripheral carotid body chemoreceptors are the main oxygen sensors. Under normal circumstances, a decline in PO_2 has very little impact on minute ventilation other than enhancing sensitivity to PCO_2 (Leff and Schumacker, 1993).

Figure 9.10 shows that from the normal arterial PO_2 (PaO_2) of 95 mmHg (Table 9.1) to a value of about 60 mmHg, minute ventilation does not change if the arterial PCO_2 (PaCO_2) remains normal (~ 36 to 42 mmHg). However, if the PaO_2 decreases below 50 mmHg, minute ventilation is stimulated to rise

exponentially. Such a large decrease is not a normal physiological event at sea level, even at very heavy levels of exercise, because the PaO_2 mmHg is maintained within narrow limits. However, PaO_2 may fall below 50 mmHg at altitudes above 4,268 m (14,000 ft), triggering the graphically depicted increase in minute ventilation (Leff and Schumacker, 1993; West, 2005).

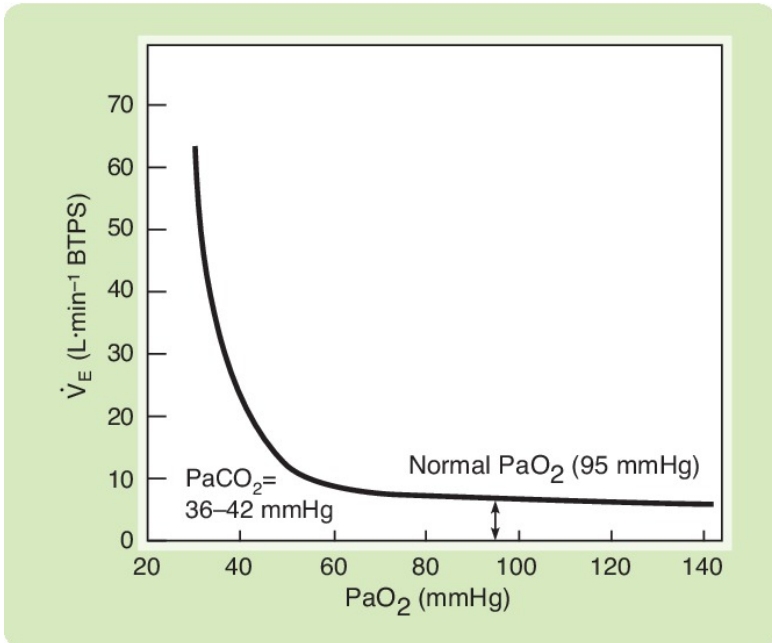


Figure 9.10 The Effect of Arterial PO_2 (PaO_2) on Minute Ventilation.

At a normal PaCO_2 of 36–42 mmHg, reduced PaO_2 does not stimulate ventilation until the value of PaO_2 is less than 60 mmHg. **Source:** From Leff, A. R., & P. T. Schumacker: Respiratory Physiology: Basics and Applications. Philadelphia, PA: W. B. Saunders (1993). Reprinted with permission from Alan R. Leff, MD.

INFLUENCE OF PCO_2 Both the central and the peripheral chemoreceptors are sensitive to an increase in the PaCO_2 . However, the peripheral chemoreceptors respond more directly to

PCO₂ than the central chemoreceptors do. The central chemoreceptors respond initially to rising PCO₂ levels but thereafter primarily to the effect these rising PCO₂ levels have on pH. The ventilatory response to PCO₂ is split 40–60% between peripheral and central chemoreceptors, respectively. At rest, the regulation of pH in the cerebrospinal fluid (CSF) is the primary control mechanism (by the central receptors) of ventilation.

Figure 9.11 shows the influence of increasing arterial PCO₂ values on minute ventilation. Even a small deviation from the normal value of PaCO₂ of approximately 40 mmHg (**Table 9.1**) causes a large rectilinear increase in minute ventilation when arterial PO₂ is maintained at approximately normal levels (~95 mmHg). The steepness and the immediacy of this rectilinear increase indicate that ventilation is much more sensitive to an increase in PCO₂ than to a decrease in PO₂. Hence, PaCO₂ is the strongest regulating factor. An increase as small as 5 mmHg in PaCO₂ increases ventilation by approximately 67%. The ventilatory response to CO₂ is reduced in sleep, with increasing age, in trained athletes, and in underwater divers ([West, 2005](#)).

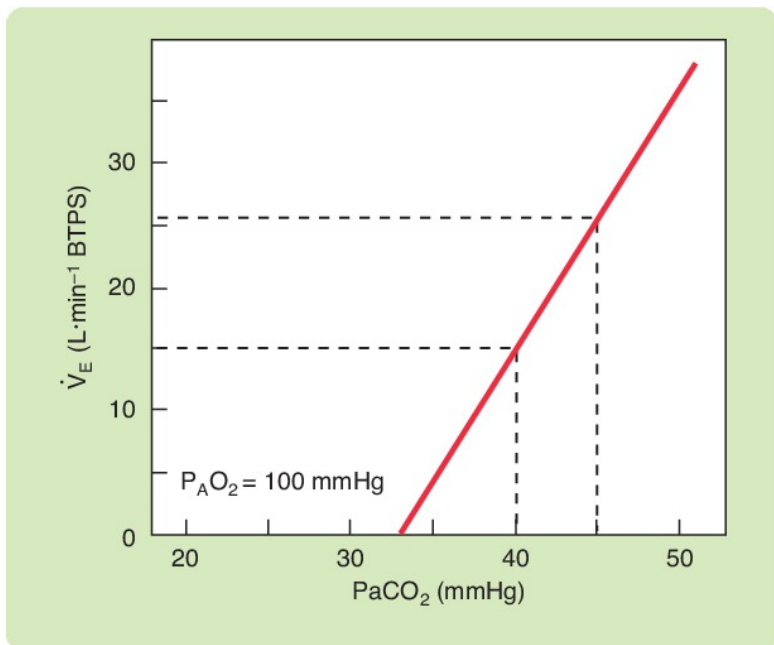


Figure 9.11 The Ventilatory Response to Carbon

Dioxide.

At a normal PAO_2 of approximately 100 mmHg, a rise in $PaCO_2$ from the normal value of 40 mmHg to just 45 mmHg almost doubles pulmonary ventilation (from ~ 15 to 26 $L \cdot min^{-1}$, as indicated by the *dashed lines* intersecting the y-axis), showing how sensitive ventilation is to $PaCO_2$.

Source: Modified from Nielsen, M., & H. Smith: Studies on the regulation of respiration in acute hypoxia. *Acta Physiologica Scandinavica*. 24(4):293–313 (1952). Copyright © 1952 Scandinavian Physiological Society. Reprinted by permission of John Wiley & Sons, Inc.

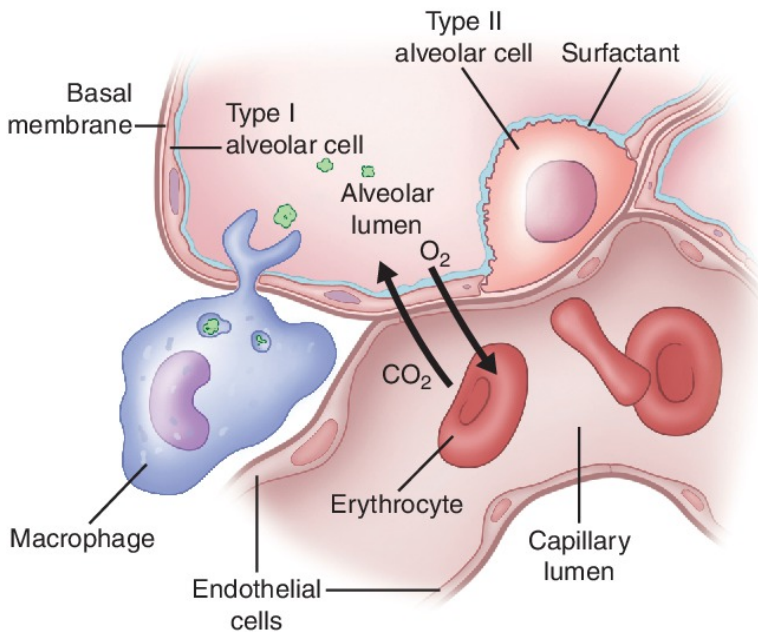


Figure 9.12 Anatomical Diagram of External Respiration.

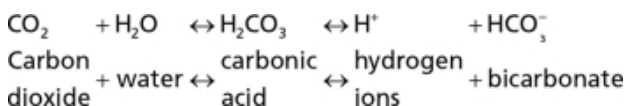
Source: Reprinted with permission from Seifter, J., A. Ratner, & D. Sloane: *Concepts in Medical Physiology*. Philadelphia, PA: Lippincott Williams & Wilkins (2005).

Holding your breath will cause an increase in the arterial PCO₂, called *hypercapnia*. Sometimes, an excess of CO₂ in the blood can lead to labored or difficult ventilation, which is termed **dyspnea**. Excess CO₂ is not the only possible cause of dyspnea, and this is not a normal exercise response. A buildup of CO₂ is the reason your conscious control of respiration was overruled if you tried not to breathe after the 3-minute jog in place earlier. Conversely, **hyperventilation**—defined as increased pulmonary ventilation, especially ventilation that exceeds metabolic requirements—decreases PCO₂ (called *hypocapnia*) and the drive to breathe. Hyperventilation may be caused by an altitude response to a decreased PO₂, an involuntary response during an anxiety attack, or a conscious attempt to extend breath-holding time.

Dyspnea Labored or difficult breathing.

Hyperventilation Increased pulmonary ventilation, especially ventilation that exceeds metabolic requirements; carbon dioxide is blown off, leading to a decrease in its partial pressure in arterial blood.

INFLUENCE OF pH As with an increase in arterial PCO₂, both the peripheral and the central chemoreceptors are sensitive to the decreases in pH. The pH level is partially related to the CO₂ level. When CO₂ is hydrated, it forms carbonic acid (H₂CO₃), which degrades readily into hydrogen ions (H⁺) and bicarbonate (HCO₃⁻) according to the following reactions:



In blood, the H⁺ can be buffered, but if that capacity is exceeded, any change in arterial pH is detected by the aortic and carotid bodies. The brain and the spinal cord are bathed by a protein-free solution known as the cerebrospinal fluid (CSF). CO₂ easily diffuses across the blood-brain barrier and into the CSF.

Because brain cells also produce CO_2 and because CSF has no protein buffers, the pH of the CSF is slightly more acidic than that of blood. An excess of H^+ in the CSF acts directly on the central chemoreceptors to increase respiration; a decrease in H^+ suppresses respiration.

All changes in pH, of course, are not caused by CO_2 . For example, during high-intensity exercise, large quantities of lactate accumulate. If the blood's ability to buffer the resultant H^+ is exceeded, pH decreases, and respiration increases.

INFLUENCE OF $[\text{K}^+]$ Of the chemical factors mentioned so far ($\downarrow\text{PO}_2$, $\uparrow\text{PCO}_2$, and $\downarrow\text{pH}$), only $\downarrow\text{pH}$ changes enough during exercise to cause the needed increase in ventilation known as hyperpnea. **Hyperpnea** is increased pulmonary ventilation that matches an increased metabolic demand. An increase in the concentration of potassium $[\text{K}^+]$ may be another factor that changes sufficiently in exercise to increase ventilation. Potassium moves from the working muscles to blood during exercise of any intensity. This increased $[\text{K}^+]$, called *hyperkalemia*, directly stimulates the carotid bodies. At rest, $[\text{K}^+]$ is not a factor (Eldridge, 1994; Forster and Pau, 1994; Nye, 1994).

Hyperpnea Increased pulmonary ventilation that matches an increased metabolic demand, such as during exercise.

Figure 9.9 earlier schematically depicted factors that control the rate and depth of ventilation. It would be logical to assume that changes in the arterial PO_2 and PCO_2 occur during exercise and have the primary role in control. However, this is not what happens. Neither PO_2 nor PCO_2 changes enough, especially early in exercise or at low to moderate to heavy intensities in untrained individuals, to play a major role in ventilatory control during exercise. Exactly which factor is most important is not known precisely. Changes may take place in the sensitivity of the medullary respiratory control centers themselves during exercise. Neural messages from the motor cortex, muscle proprioceptors, and hypothalamic sympathetic nervous system activity, as well as increases in the hydrogen ion and potassium ion concentrations, all appear to have a role during exercise (Eldridge, 1994; West,

2005).

Gas Exchange and Transport

Gas Exchange: Henry's Law

As mentioned previously, knowledge of the partial pressure of gases is important for two reasons. First, the partial pressures of gases play a role in the control of pulmonary ventilation, as discussed above. Second, the movement of O₂ and CO₂ between the alveoli and the capillaries (external respiration) and between the capillaries and the tissues (internal respiration) occurs by the process of diffusion. **Diffusion** is the tendency of gaseous, liquid, or solid molecules to move from an area of higher concentration to an area of lower concentration by constant random action. Diffusion can occur only if there is a pressure gradient (a difference in the partial pressures of the gas) between the capillary and the tissue. Gases diffuse down the pressure gradient from a higher pressure area to a lower pressure area. The rate of diffusion depends on the magnitude of the pressure gradient (millimeters of mercury at the high end minus millimeters of mercury at the low end), the surface area available for diffusion, the thickness of the barrier between the two locations, and the solubility (ability to be dissolved) of the gas in the barrier liquid. *Henry's law* states that when a mixture of gases is in contact with a liquid, each gas dissolves in the liquid in proportion to its partial pressure and solubility until equilibrium is achieved and the gas partial pressures are equal in both locations (Leff and Schumacker, 1993; Martin et al., 1979).

Diffusion The tendency of gaseous, liquid, or solid molecules to move from an area of higher concentration to an area of lower concentration by constant random action.

FOCUS ON APPLICATION | *Clinically Relevant*

Decreased PCO₂ and Drowning

The decrement in PCO₂ with hyperventilation can have serious consequences. Many people know that hyperventilating can extend breath-holding time but erroneously believe it does so because more oxygen is taken in. They are unaware that it is not O₂ but CO₂ levels that are changing (being blown off) and that respiration is more sensitive to PCO₂ changes than to PO₂ changes. [Craig \(1976\)](#) summarized 58 cases of loss of consciousness during underwater swimming and diving following hyperventilation. Of these 58 cases, 23 (40%) ended as fatalities, with most occurring in guarded pools. Because an individual continues patterned motor activity (swimming) for a short time after loss of consciousness caused by a lack of oxygen to the brain, these life-threatening cases are difficult for a lifeguard to detect quickly. Beginning swimmers frustrated by trying to coordinate their arms, legs, and breathing who simply put their head in the water and try to go as far as possible without breathing are also vulnerable. As a result of these findings, it is recommended that underwater swimming be limited to one length of a standard 25-yd or 25-m pool.



Source: [Craig \(1976\)](#).

External Respiration

External respiration is the movement of gases at the alveolar-pulmonary capillary level (**Figure 9.12**). Specifically, oxygen diffuses from the alveoli into the pulmonary capillaries, and carbon dioxide diffuses from the pulmonary capillary into the alveoli. This exchange is diagrammed in **Figure 9.13A and D**.

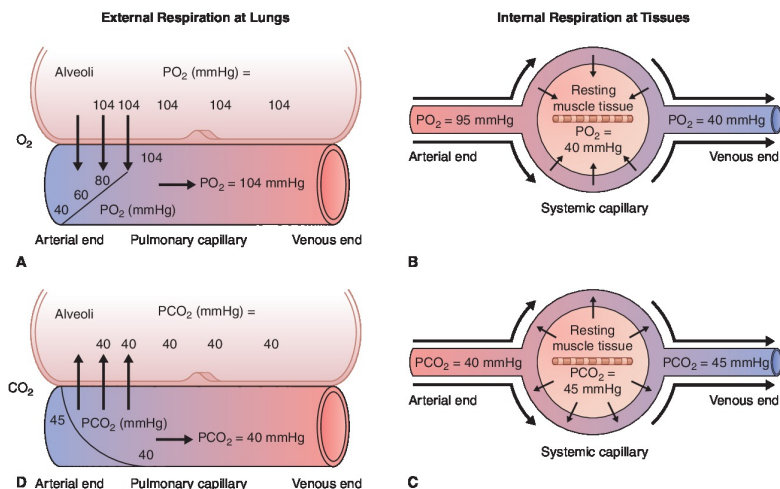


Figure 9.13 Oxygen and Carbon Dioxide Exchange.

Gas exchange takes place at two anatomical locations: the alveoli-pulmonary capillary interface (external respiration) and the systemic capillary-tissue interface (internal respiration). **A.** External respiration for oxygen. **B.** Internal respiration for oxygen. **C.** Internal respiration for carbon dioxide. **D.** External respiration for carbon dioxide.

Remember that pulmonary circulation originates from the right ventricle of the heart. By definition, an artery is a vessel carrying blood away from the heart. Unlike systemic arteries, the pulmonary artery carries partially deoxygenated blood. The pulmonary arteries quickly branch into capillaries that parallel the alveoli. While they are small, the capillaries do have a definable length.

At the arterial end of the capillary, PO₂ in the alveoli (PAO₂) is high (98–104 mmHg) (**Table 9.1**), and PO₂ in the pulmonary

capillary is low (40 mmHg). Oxygen diffuses down the pressure gradient until equilibrium is reached at approximately 104 mmHg. Note in **Figure 9.13A** that this equalization of pressure occurs within the first third of the capillary length. This means that blood can actually flow three times as fast through the pulmonary capillaries and still be adequately oxygenated.

Refer again to **Table 9.1** and **Figure 9.13A and B**. In **Figure 9.13B**, notice that PaO_2 (systemic arterial blood) is listed as 95 mmHg, not as 98–104 mmHg as given in **Table 9.1** and **Figure 9.13A**. The lower value in the systemic arterial blood (95 mmHg) versus the venous pulmonary blood (104 mmHg) results because the fully oxygenated blood in the pulmonary vein is diluted with partially oxygenated venous blood ($\text{PvO}_2 = 40$ mmHg) from the bronchial vein as blood from both the veins flows into the left atrium. Remember that the bronchial artery, the capillaries, and the vein provide blood to and return it from the lungs as part of the systemic circulation and so have the normal systemic gas contents and pressures (**Figure 9.4**). Although blood from the bronchial vein amounts to only about 2% of the total blood returning to the left atrium, it is sufficient to bring the PaO_2 of systemic arterial blood down to approximately 95 mmHg ([Hall and Hall, 2021](#)).

At the same time that oxygen is diffusing from the alveoli to the pulmonary capillaries, carbon dioxide diffuses from the pulmonary capillaries to the alveoli. At the arterial end of the capillary, PCO_2 in the capillary is high (about 45 mmHg) and PCO_2 in the alveoli is low (40 mmHg) (**Table 9.1**). Carbon dioxide also diffuses from high to low pressure down the gradient until equilibrium is reached. Note in **Figure 9.13D** that this equalization of pressure occurs within the first third of the capillary length. Note also that the pressure gradient for CO_2 is not nearly as steep (5 mmHg) as it is for O_2 (64 mmHg). Carbon dioxide diffuses with a lesser pressure gradient because it is more soluble.

Remember that each gas moves down its own concentration gradient, regardless of the concentration of any other gas present. Work through the [Check Your Comprehension 3 box](#) to ensure that you understand the partial pressure of oxygen at different sites.

Major External Respiration Variables

The major variables that are important in external respiration are alveolar ventilation (\dot{V}_A), the partial pressure of oxygen at the alveoli (PAO_2), partial pressure of oxygen in the arterial blood (PaO_2), oxygen or partial pressure of oxygen gradient between the alveoli and the arteries (A-a) PO_2 , the percent saturation of arterial blood with oxygen ($SaO_2\%$), and the partial pressure of carbon dioxide at the alveoli ($PACO_2$).

Internal Respiration

Internal respiration is the movement of gases at the capillary-tissue level. Although the tissue can be any tissue, it is particularly relevant for exercise physiology students to think of the tissue as skeletal muscle tissue. Internal respiration is diagrammed in **Figure 9.13B and C**. Skeletal muscle fibers are well supplied with capillaries. When blood enters the arterial end of any systemic capillary, PaO_2 is high (95 mmHg). Within the tissue, PO_2 at rest is low (40 mmHg). Oxygen diffuses from high to low pressure until equilibrium is reached. Blood exiting into the systemic venous system thus has a PvO_2 of 40 mmHg, which is maintained until oxygenation occurs again at the alveoli.

CHECK YOUR COMPREHENSION 3

Using the information presented in **Figure 9.13**, return to **Figure 9.1** and superimpose the partial pressure of O_2 in alveoli (at sea level), the partial pressure of O_2 in the pulmonary capillaries, the partial pressure of O_2 in the systemic arterial blood, and the partial pressure of O_2 in the muscle cells at rest in the appropriate place on **Figure 9.1**.

Check your answer in Appendix C.

Carbon dioxide is produced at the tissue level as a direct result of cellular respiration. Thus, PCO_2 levels are higher (about 45 mmHg) in the tissues than elsewhere in the system. Diffusion occurs as always from the area of high pressure (45 mmHg in the

tissue) to the area of low pressure (40 mmHg in the systemic capillary), once again achieving equilibrium. The venous level of PCO_2 is also maintained until it reaches the alveoli, where the carbon dioxide diffuses out of the bloodstream and is exhaled. To make sure that you understand the movement of respiratory gases during rest, complete the task in the [Check Your Comprehension 4 box](#).

CHECK YOUR COMPREHENSION 4

Trace the movement of oxygen through the body from the alveoli to the pulmonary capillaries, through the left side of the heart, the systemic arteries, the tissue, the systemic capillaries, the systemic veins, the right side of the heart, and back to the alveoli. Give the PO_2 at each site and any diffusion of oxygen. Do the same thing for carbon dioxide, giving PCO_2 , but start at the tissue level where carbon dioxide is produced and follow it through the systemic capillaries and veins, the right side of the heart, the pulmonary capillaries and the alveoli, the pulmonary vein, the left side of the heart, the systemic arterial system, and back to the systemic capillaries at the tissue level.

Check your answer in Appendix C.

Major Internal Respiration Variables

The respiratory variables that are important for internal respiration are the partial pressure of oxygen in arterial blood (PaO_2), the partial pressure of oxygen in the venous blood (PvO_2), the amount of oxygen carried in the arteries minus the amount carried in the veins ($a\text{-vO}_2\text{diff}$), the partial pressure of carbon dioxide in arterial blood (PaCO_2), the partial pressure of carbon dioxide in venous blood (PvCO_2), and the percent saturation of venous blood with oxygen ($\text{SvO}_2\%$).

Oxygen Transport

Oxygen is carried in two ways in the blood. First, oxygen is transported in a dissolved form in the liquid portion of the blood.

The amount of oxygen transported this way is only about 1.5–3% of the total oxygen transported. However, it is this dissolved component that is responsible for the partial pressure of oxygen in the blood (**Figure 9.13**). The dissolved oxygen content is determined by the PO₂ and the solubility of oxygen, which is a constant of 0.00304 mL·dL⁻¹·mmHg⁻¹ at 37°C. The formula is as follows ([Hall and Hall, 2021](#); [Leff and Schumacker, 1993](#)):

$$\text{dissolved O}_2 \text{ content (mL} \cdot \text{dL}^{-1}) = \text{PO}_2 \text{ (mmHg)} \times \text{solubility (mL} \cdot \text{dL}^{-1} \cdot \text{mmHg}^{-1})$$

The second way oxygen is transported in the blood is bound to hemoglobin. The vast majority, 97–98.5%, of the oxygen in the bloodstream is transported this way.

Example

In normal systemic arterial blood with a PaO₂ of 95 mmHg, the calculation becomes

$$\begin{aligned} \text{dissolved O}_2 \text{ content} &= (95 \text{ mmHg}) \\ &\times (0.00304 \text{ mL} \cdot \text{dL}^{-1} \cdot \text{mmHg}^{-1}) \\ &= 0.29 \text{ mL} \cdot \text{dL}^{-1} \end{aligned}$$

Thus, only 0.29 mL of oxygen is dissolved in a deciliter of arterial blood. You may also see this result expressed in units of milliliters per 100 milliliters (mL·100 mL⁻¹) of blood or as milliliters percent (mL %). All are comparable.

Red Blood Cells and Hemoglobin

Red blood cells (RBCs), or erythrocytes, are small, flexible cells shaped like biconcave disks, or miniature doughnuts, with the area where the hole would be just squished in (**Figure 9.14**). Females typically have an RBC count of 4.3–5.2 million per cubic milliliter of blood; the male count is generally 5.1–5.8 million per cubic milliliter of blood. The average person has about 35 trillion

RBCs, which have a total surface area of about 2,000 times the body's total surface area. Hemoglobin, which is the protein portion of the RBC that binds with oxygen, constitutes about one third of an RBC by weight if water is included or 97% if water is excluded. Normal values for hemoglobin for adult females are 12–16 g·100 mL⁻¹ blood (g·dL⁻¹), with a mean of 14 g·100 mL⁻¹ blood. For adult males, the values are 14–18 g·100 mL⁻¹ blood, with a mean of 16 g·100 mL⁻¹ blood. Children have slightly lower values ([Hall and Hall, 2021](#)).



Figure 9.14 Red Blood Cells.

An electron micrograph of erythrocytes, or RBCs, showing the biconcave disk shape. See animation, Oxygen Transport,

on Lippincott Connect



Hemoglobin consists of four iron-containing pigments called *hemes* (**Figure 9.15**) and a protein called *globin*. Each of the four iron atoms in a hemoglobin molecule can combine reversibly

with one molecule of oxygen. Therefore, each hemoglobin molecule can transport four molecules of oxygen. The chemical symbol for hemoglobin is Hb. The chemical symbol for hemoglobin not bound to oxygen, sometimes called *deoxyhemoglobin* or reduced hemoglobin (negatively charged) such that it binds quickly and easily to H^+ ions, is often designated as HHb. Hemoglobin bound to oxygen, symbolized HbO_2 , is called *oxyhemoglobin*. None of the oxygen bound to hemoglobin is used by the RBC.

Hemoglobin (Hb) The protein portion of the red blood cell that binds with oxygen, consisting of four iron-containing pigments called hemes and a protein called globin.

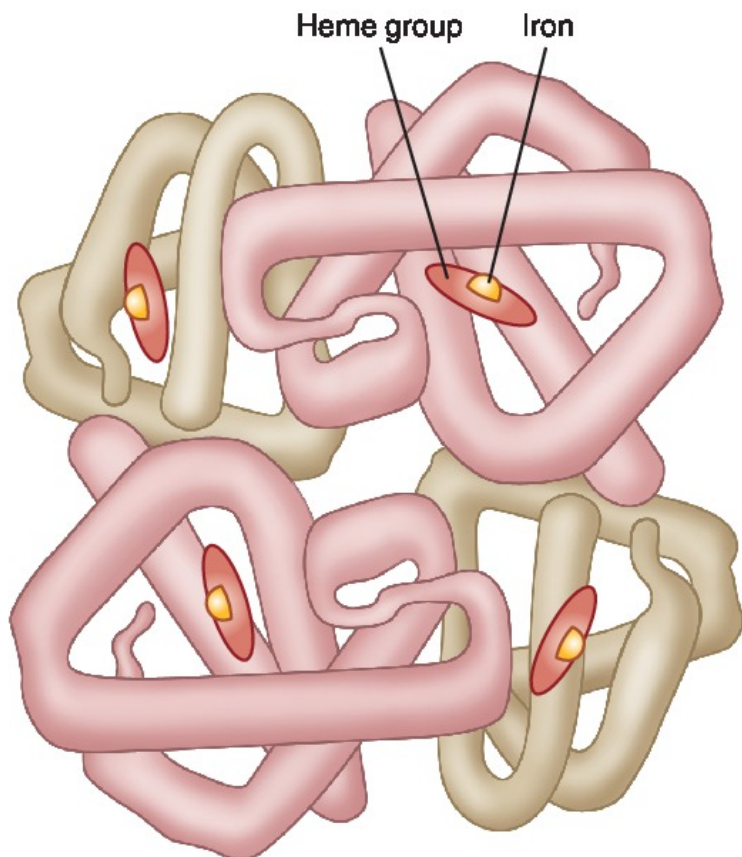


Figure 9.15 Hemoglobin.

Hemoglobin consists of four iron-containing heme units and the protein globin (the chains surrounding the heme units).

Source: Reprinted with permission from Seifter, J., A.

Ratner, & D. Sloane: *Concepts in Medical Physiology*.

Philadelphia, PA: Lippincott Williams & Wilkins (2005).

The Binding of Oxygen with Hb: The Oxygen Dissociation Curve

All four oxygen molecules do not bind with the four heme atoms at the same time (**Figure 9.16**). **Figure 9.16A** shows deoxyhemoglobin. Binding of one oxygen molecule onto a fully deoxygenated hemoglobin molecule (**Figure 9.16B**) changes the spatial arrangement (conformation) of all of the other subunits (**Figure 9.16C**). This change increases the binding affinity for oxygen. Thus, each succeeding oxygen molecule binds more easily and quickly than the preceding one (**Figure 9.16D–F**) (Seifter et al., 2005). The result is a fully oxygenated hemoglobin molecule (HbO₂). When graphed, this process of successive molecules of oxygen binding to hemoglobin produces the characteristic sigmoid-shaped curve of oxyhemoglobin shown in **Figure 9.17**. This curve is called the *oxygen dissociation curve*, for reasons that will become clear later.

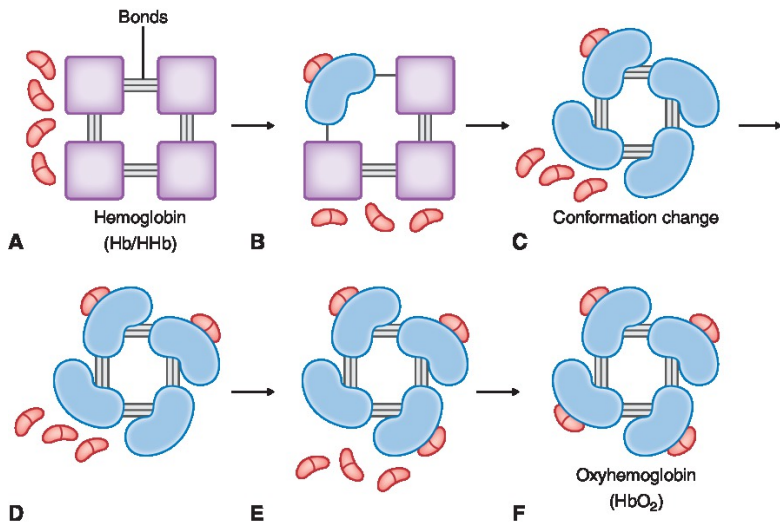
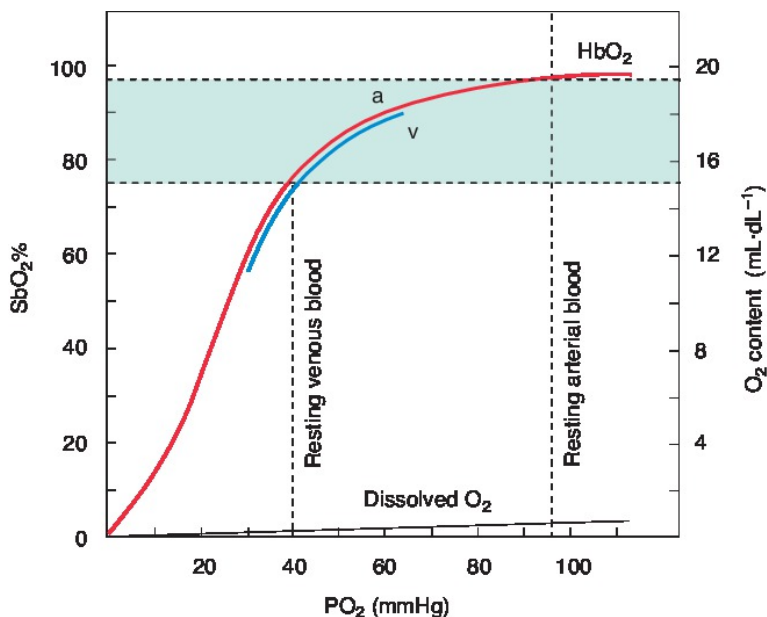


Figure 9.16 Oxygenation of Hemoglobin.

A. Hemoglobin consists of four heme subunits tightly bonded to each other with a relatively low affinity for oxygen (O_2). B. Once oxygen binds to one heme subunit, the spatial arrangement (conformation) of all other subunits changes (C), increasing the affinity for oxygen. As a result, second (D), third (E), and fourth (F) oxygen molecules bind increasingly faster. This results in fully oxygenated hemoglobin (HbO_2). **Source:** Seifter et al. (2005). Used with permission.



Assumes Hb = $15 \text{ g}\cdot\text{dL}^{-1}$ blood; Body temperature = 37°C

a = arterial blood; $\text{PaCO}_2 = 40 \text{ mmHg}$; pH = 7.4

v = venous blood; $\text{PvCO}_2 = 45 \text{ mmHg}$; pH = 7.38

Figure 9.17 Oxygen Dissociation Curve.

Under normal resting conditions, approximately 25% of the oxygen being transported in arterial blood is dissociated (released or exchanged by internal respiration) for use by body tissues. The *shaded area* at the top of the diagram represents this in percentage on the left y-axis and as an absolute amount on the right y-axis. See text for full explanation and calculations.

When all four of its heme groups are bound to oxygen, the hemoglobin molecule is said to be fully saturated. All hemoglobin molecules may not be fully saturated, though. The amount of the oxygen-carrying capacity being used at a given time is referred to as **percent saturation of hemoglobin**, designated as $\text{SbO}_2\%$. Percent saturation is calculated as:

Percent Saturation of Hemoglobin (SbO₂%) The ratio of the amount of hemoglobin combined with oxygen to the total hemoglobin capacity for combining with oxygen, expressed as a percentage; indicated generally as SbO₂% or specifically as SaO₂% for arterial blood or as SvO₂% for venous blood.

$$9.6 \quad \text{SbO}_2\% = \frac{\text{Hb combined with O}_2}{\text{Hb capacity for combining with O}_2} \times 100$$

The symbols SaO₂% and SvO₂% may be used to distinguish percent saturation of blood in the arteries and the veins, respectively, whereas Sb refers nonspecifically to blood.

Percent saturation depends primarily on the partial pressure of oxygen. In the arterial blood at a PO₂ of 95 mmHg, the percent saturation is 97%. Find this value on **Figure 9.17**, where PO₂ is on the x-axis and SbO₂% is on the left y-axis. On the same figure, determine the SbO₂% in normal venous blood. Since the PO₂ in normal venous blood is 40 mmHg, you should have determined that the SbO₂% was 75%. This value is also shown in **Figure 9.17**.

In addition to knowing the % saturation of blood with oxygen, it is also useful to know how much oxygen is carried in the blood. The *oxygen content* is the amount of oxygen (in mL) carried in 100 mL (or 1 dL) of blood. The oxygen content of hemoglobin depends upon the hemoglobin level and the physiological oxygen-binding capacity, according to the following formula:

$$\begin{aligned} \text{oxygen content of hemoglobin (mL} \cdot \text{dL}^{-1}\text{)} = \\ \text{hemoglobin level (gm} \cdot \text{dL}^{-1}\text{)} \times \text{oxygen-binding} \\ \text{capacity (mL O}_2 \cdot \text{gm} \cdot \text{Hb}^{-1}\text{)} \times \text{percent saturation} \\ \text{(expressed as a decimal fraction)} \end{aligned}$$

or

$$9.7 \quad \text{HbO}_2 = \text{Hb} \times 1.34 \times \text{SbO}_2\%$$

In this equation, the hemoglobin level will, of course, vary from individual to individual. The physiological oxygen-binding capacity is a constant 1.34 mL O₂·g Hb⁻¹, and the percent saturation of hemoglobin will vary between arterial and venous

blood and resting and exercise conditions.

Example

Using an average hemoglobin level of $15 \text{ g} \cdot \text{dL}^{-1}$, calculate the oxygen content for arterial blood where the percent saturation is 97%.

$$\begin{aligned} \text{HbO}_2 \text{ mL} \cdot \text{dL}^{-1} &= 15 \text{ g} \cdot \text{dL}^{-1} \\ &\quad \times 1.34 \text{ mL O}_2 \cdot \text{g Hb}^{-1} \times 0.97 \\ &= 19.5 \text{ mL} \cdot \text{dL}^{-1} \end{aligned}$$

In **Figure 9.17**, look at the right side y-axis. This axis is of O_2 content in $\text{mL} \cdot \text{dL}^{-1}$ rounded to the nearest whole number. The value for normal arterial blood at 97% saturation is the value just calculated.

Arteriovenous Oxygen Difference

The amount of oxygen released (dissociated) from the blood during one circuit through the systemic system is called the **arteriovenous oxygen difference (a-vO₂diff)**. That is, the a-vO₂diff is the difference between the amount of oxygen originally carried in the arterial blood and that returned in the venous blood. Before subtracting to obtain the a-vO₂diff, we must calculate the total oxygen in both the arteries and the veins. The amount normally carried in the arterial blood has already been determined. It is:

Arteriovenous Oxygen Difference (a-vO₂diff) The difference between the amount of oxygen originally carried in arterial blood and the amount returned in venous blood.

$$\begin{aligned}
 \text{dissolved O}_2 &= 0.29 \text{ mL} \cdot \text{dL}^{-1} \\
 + \text{HbO}_2 &= \frac{19.50 \text{ mL} \cdot \text{dL}^{-1}}{19.79 \text{ mL} \cdot \text{dL}^{-1}} = \text{total O}_2 \text{ content} \\
 &\quad \text{in arterial blood (aO}_2\text{)}
 \end{aligned}$$

Example

Calculate the dissolved O₂ and HbO₂ in venous blood.
The calculations are as follows:

$$\begin{aligned}
 \text{dissolved O}_2 (\text{venous}) &= 40 \text{ mmHg} \\
 &\quad \times 0.00304 \text{ mL O}_2 \cdot \text{dL}^{-1} \cdot \text{mmHg}^{-1} \\
 &= 0.12 \text{ mL} \cdot \text{dL}^{-1} \\
 \text{HbO}_2 (\text{venous}) &= 15 \text{ g} \cdot \text{dL}^{-1} \times 1.34 \text{ mL} \cdot \text{g}^{-1} \\
 &\quad \times 0.75 = 15.08 \text{ mL} \cdot \text{dL}^{-1}
 \end{aligned}$$

Now, add these values:

$$\begin{aligned}
 \text{dissolved O}_2 &= 0.12 \text{ mL} \cdot \text{dL}^{-1} \\
 \text{HbO}_2 &= 15.08 \text{ mL} \cdot \text{dL}^{-1} \\
 \frac{15.20 \text{ mL} \cdot \text{dL}^{-1}}{15.20 \text{ mL} \cdot \text{dL}^{-1}} &= \text{total O}_2 \text{ content} \\
 &\quad \text{in venous (vO}_2\text{)}
 \end{aligned}$$

The a-vO₂diff is calculated by the formula:

$$\begin{aligned}
 \text{a-vO}_2 \text{ diff} &= \text{O}_2 \text{ in arterial blood (mL} \cdot \text{dL}^{-1}\text{)} \\
 &\quad \text{O}_2 \text{ in venous blood (mL} \cdot \text{dL}^{-1}\text{)} \\
 &\quad \text{or}
 \end{aligned}$$

$$9.8 \quad \text{a-vO}_2 \text{ diff} = \text{aO}_2 - \text{vO}_2$$

Therefore, the difference, using the values already calculated, is:

$$\begin{aligned}
 \text{a-vO}_2 \text{ diff} &= 19.79 \text{ mL} \cdot \text{dL}^{-1} - 15.20 \text{ mL} \cdot \text{dL}^{-1} \\
 &= 4.59 \text{ mL} \cdot \text{dL}^{-1}
 \end{aligned}$$

Approximately $5 \text{ mL O}_2 \cdot \text{dL}^{-1}$ is used by the tissue to support cellular metabolism under normal resting conditions. This means that approximately 25% ($4.59 \div 19.79 = 0.23 \times 100 = 23\%$) of the oxygen is actually released from the hemoglobin to the tissues and used during rest. And, thus, approximately 75% of the oxygen is held in reserve for use when needed such as during physical exercise. The actual amount of oxygen used, measured as the $a-v\text{O}_2\text{diff}$, is called the *coefficient of oxygen utilization*. The normal resting coefficient of oxygen utilization is $4.59 \text{ mL} \cdot \text{dL}^{-1}$.

Note that the 75% is also the $\text{SbO}_2\%$ value for venous blood. Refer to **Figure 9.17** again. The shaded area at the top—between the $\text{SbO}_2\%$ values of 97% and 75% on the left axis and between the values 19.79 and $15.20 \text{ mL} \cdot \text{dL}^{-1}$ on the right axis—represents these relative and absolute amounts of O_2 that have been released or dissociated primarily from the RBCs. The separation or release of oxygen from the RBCs to the tissues is called **oxygen dissociation**, and the curve is called the oxygen dissociation curve. Complete the [Check your comprehension Box 5](#) on oxygen transport.

Oxygen Dissociation The separation or release of oxygen from the RBCs to the tissues.

CHECK YOUR COMPREHENSION 5-CASE STUDY

Shu Fang runs high school cross-country. As a junior, her best time for 3 mi was 17:54, but in her senior season, she has not been able to break 19:10 on the same course and always feels tired. A medical examination revealed that last year her hemoglobin level was $13.2 \text{ g} \cdot \text{dL}^{-1}$, but this year it is $11.9 \text{ g} \cdot \text{dL}^{-1}$. As her coach or athletic trainer, explain to her the impact of this difference on her ability to transport oxygen to produce energy by calculating the difference in oxygen-carrying capacity.

Check your answer in Appendix C.

Carbon Dioxide Transport

Carbon dioxide is carried in three ways in the blood from muscles or other tissues where it is produced to the lungs, where it is eliminated from the body. As with O₂, the first way it is transported is by dissolving in blood plasma. The amount of CO₂ transported in this fashion is only about 5–10% of the total. However, as with dissolved O₂, it is this dissolved CO₂ component that determines the partial pressure of CO₂ in the blood. Because the exchange of CO₂ in internal and external respiration depends largely upon the PCO₂, this is a critical role for dissolved CO₂. The remaining 90–95% of the CO₂ diffuses into the RBCs.

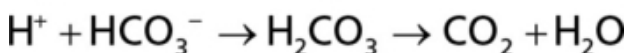
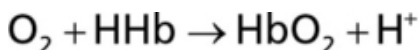
As CO₂ enters the RBC, about 20% combines chemically with the globin portion of the Hb molecule to produce *carbaminohemoglobin* (HbCO₂). This is the second form of CO₂ transport. Some small quantity may combine with proteins in the plasma as well. Note that when CO₂ combines with Hb, the CO₂ is not competing with oxygen for space on the heme units, because it combines with the globin portion. However, more CO₂ can combine with Hb if it is deoxygenated. That is, the lower the PO₂ and SbO₂%, the greater is the amount of CO₂ that can be carried in the blood. This reaction is known as the *Haldane effect* (Leff and Schumacker, 1993).

At the lungs, the situation is reversed: as O₂ saturates the Hb, less CO₂ can be bound, so the release of CO₂ is stimulated. Thus, Hb is really a transport vehicle carrying O₂ from the alveoli to the cells and CO₂ from the cells to the alveoli. This task is made easier by the fact that the dissociation (dropping off) of one molecule facilitates the binding (picking up) of the other at both sites.

The third way that CO₂ is transported is as bicarbonate ions. Approximately 70–75% of the CO₂ is transported in this way. When CO₂ diffuses into the RBCs, it combines with water under the influence of the enzyme carbonic anhydrase and forms carbonic acid (H₂CO₃). Carbonic acid is weak and unstable and quickly dissociates into hydrogen ions (H⁺) and bicarbonate ions (HCO₃⁻). The chemical reaction is depicted as:



The H^+ binds to Hb ($H^+ + Hb \rightarrow HHb$), thus preventing much change in pH, and the HCO_3^- diffuses into plasma. To counteract this loss of negative charges, chloride ions (Cl^-) move from plasma to the RBC. This ion exchange is called the *chloride shift*. At the lungs, where PCO_2 is relatively low, the reactions are all reversed. The chemical reactions are:



Once back in the form of CO_2 , the CO_2 diffuses along its partial pressure gradient from the blood to the alveoli and is exhaled ([Hall and Hall, 2021](#); [Leff and Schumacker, 1993](#)). See animation, Transport of Carbon Dioxide, on Lippincott Connect.



Figure 9.18 summarizes the transport of both oxygen and carbon dioxide in arterial and venous blood. Take time to study this in terms of the processes that have been described above.

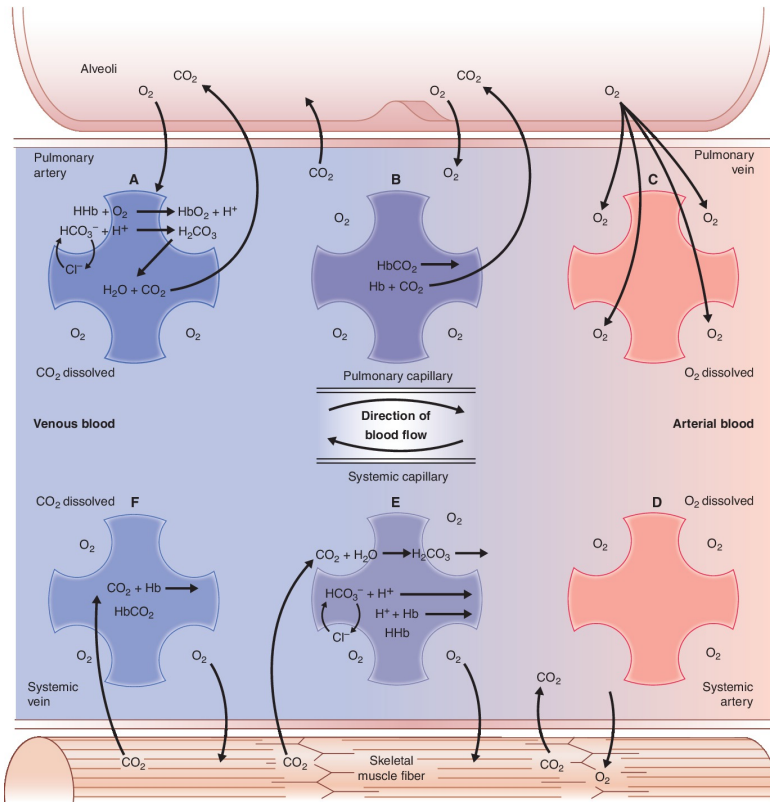


Figure 9.18 Summary of Oxygen and Carbon Dioxide Transport.

Oxygen is transported in two ways in the circulatory system—dissolved and bound to the heme units of hemoglobin (RBC). Carbon dioxide is transported in three ways in the circulatory system—dissolved, bound to the globin portion of hemoglobin, and as bicarbonate. **A.** The oxygenation of hemoglobin and diffusion of carbon dioxide from bicarbonate. **B.** The diffusion of carbon dioxide from the globin portion of hemoglobin. **C.** The transport of oxygen on heme portions of hemoglobin. **D.** The diffusion of oxygen from the heme portion of hemoglobin. **E.** The transport of carbon dioxide as bicarbonate. **F.** The transport of carbon dioxide on the globin portion of hemoglobin.

The Respiratory System and Acid-Base Balance

The ability of hemoglobin to bind the hydrogen ions (H^+) produced during the transport of carbon dioxide and the ability of pulmonary ventilation both to respond to (**Figures 9.9 and 9.12**) and eliminate carbon dioxide are very important for maintaining acid-base balance. These reactions exemplify two of the three lines of defense for regulating acid-base balance: the chemical buffer system and the respiratory system. Furthermore, the partial pressure of carbon dioxide is also directly involved in the third line of defense, renal regulation by the kidneys.

Acid-base balance involves a series of mechanisms that attempt to regulate the concentration of hydrogen ions in body fluids. This balance is vitally important, because virtually all biochemical reactions in the human body require the pH to be maintained within very narrow limits for proper functioning ([Hall and Hall, 2021](#)). Hydrogen ions come from several sources in the human body, but most result from the aerobic production of energy when oxygen is used and carbon dioxide is generated or when lactate is produced and H^+ accumulates in anaerobic energy production.

A *chemical buffer* is a system of one or more compounds that resists changes in pH when a strong acid or base is added. This is done by binding H^+ ions whenever the pH drops and releasing them when the pH rises. Chemical buffers respond instantaneously. Hemoglobin is not the only chemical buffer, but in terms of capacity, it is the most important buffer in the blood. Bicarbonate (HCO_3^-) constitutes the alkaline reserve in the blood. The H_2CO_3 content of the blood is subject to respiratory controls.

The carbonic acid-bicarbonate system (H_2CO_3/HCO_3^-) provides temporary buffering of hydrogen ions as they travel through the circulatory system. It also provides for the removal from the body of the carbon dioxide formed in the buffering process through *respiratory regulation*. The sensitivity of pulmonary ventilation to PCO_2 and pH enables varying amounts of acid production to be dealt with, because increased levels of carbon dioxide can be exhaled as needed. As explained earlier, an elevated PCO_2 level in the blood activates medullary

chemoreceptors that respond by increasing respiratory rate and depth; peripheral chemoreceptors respond to rising plasma H^+ levels to also stimulate deeper and more rapid respiration. This expels more CO_2 (an acid), converts the H^+ to water, and quickly restores pH homeostasis. This respiratory compensation reacts within a matter of 1–3 minutes (Marieb and Hoehn, 2019; Slonim and Hamilton, 1976).

Renal regulation of acid-base balance has two goals: (1) to conserve or eliminate bicarbonate ions (HCO_3^-), thereby stabilizing the amount in the body, and (2) to excrete hydrogen ions (in urine). Renal bicarbonate retention or excretion depends on the PCO_2 in arterial blood. The kidneys are the most potent of the acid-base mechanisms. While the lungs can dispose of carbonic acid by eliminating CO_2 , only the kidneys can rid the body of the other acids generated by cellular metabolism including lactic acid and ketone bodies. However, they require hours or even days to effectively change pH (Marieb and Hoehn, 2019).

In the normal resting condition, venous pH is slightly lower than arterial pH (venous = 7.35, arterial = 7.4), but this difference has very little effect on most physiological functions. The key is maintaining arterial pH levels. Overall, then, not only does the respiratory system deliver oxygen for the production of energy and remove carbon dioxide, but it also contributes to the effective functioning of all biochemical reactions in the body through its role in maintaining acid-base balance.

Summary

1. Respiration consists of pulmonary ventilation and oxygen and carbon dioxide gas exchanges at the alveolar (external) and tissue (internal) levels.
2. Structurally, the pulmonary system can be divided into the conductive and respiratory zones. The conductive zone serves to transport air, warm and humidify air, and filter the air. Gas exchange takes place in the respiratory zone.
3. At rest, inspiration is an active process brought about by a

pressure gradient that exceeds resistance to the flow of air. Resting expiration is a passive process accomplished primarily by elastic recoil.

4. The conductive zone makes up the anatomical dead space. An alveolar dead space occurs when functional alveoli are not supplied with capillaries or capillaries are pathologically blocked. Together, anatomical and alveolar dead spaces are called the physiological dead space.
5. Minute ventilation (\dot{V}_E or \dot{V}_D) is the respiratory variable most commonly measured in exercise situations. Alveolar ventilation is the best measure of air available for gas exchange.
6. Residual volume, the amount of air remaining in the lungs after a maximal expiration, must be accounted for when one determines body composition by underwater weighing.
7. Respiratory measures are collected under ambient conditions (ATPS). Most lung volumes and capacities are then converted to body temperature values (BTPS). Minute ventilation may also be converted to standard temperature and pressure, dry (STPD) conditions.
8. Primary control of respiration resides in the ventral respiratory group (VRG) and dorsal respiratory group (DRG) in the medulla oblongata with input from the pontine respiratory center in the pons. Factors affecting these centers include the following:
 - a. Conscious thought through the cerebral cortex
 - b. Sympathetic nerve reactions through the hypothalamus
 - c. Irritants or lung inflammation through systemic receptors in the airways and lungs
 - d. PO_2 , PCO_2 , pH, and K^+ through central and peripheral chemoreceptors
 - e. Movement via proprioceptive stimulation from mechanoreceptors and muscles and joints
9. Major variables for pulmonary ventilation include minute ventilation (\dot{V}_E), tidal volume (V_T), and frequency (f).
10. Major variables for external respiration include alveolar

ventilation (\dot{V}_A), partial pressure of oxygen in the alveoli (PAO₂) and partial pressure of oxygen in the systemic arterial blood (PaO₂), partial pressure of oxygen gradient between the alveoli and arteries (A-a) PO₂, and the percent saturation of arterial blood with oxygen (SaO₂%).

11. Major variables for internal respiration include partial pressure of oxygen in the systemic arterial blood (PaO₂), partial pressure of oxygen in the systemic venous blood (PvO₂), the a-vO₂ difference, the percent saturation of arterial blood with oxygen (SaO₂%), and the percent saturation of arterial venous blood with oxygen (SvO₂%).
12. Gases always flow down a pressure gradient. Each gas moves independently. Oxygen moves from the alveoli into arterial blood and red blood cells (RBCs) and then from the RBCs and capillary blood into the muscle tissues. Carbon dioxide moves from the muscle tissues into capillary blood and RBCs and then from the venous blood and RBCs into the alveoli.
13. Hemoglobin is composed of four iron heme units and one globin (a protein). When fully saturated, each hemoglobin molecule will have oxygen bound to each of its four heme structures. Normal arterial saturation (SaO₂%) is 97%; normal resting venous saturation (SvO₂%) is 75%.
14. Oxygen released from the RBCs at the tissue level is said to have been dissociated. At rest, the primary drive for this dissociation is the pressure gradient for oxygen.
15. At rest, only about 25% of circulated oxygen is used; 75% remains saturated in venous blood. These percentages translate into an arteriovenous oxygen difference of approximately 4.6 mL · dL⁻¹.
16. Carbon dioxide is transported from the tissue to the lungs in three ways: dissolved, as carbaminohemoglobin, and as bicarbonate ions. The chemical buffering and removal of carbon dioxide from the body are important for maintaining acid-base balance.

Review Questions

1. Define pulmonary ventilation, external respiration, and internal respiration. Define the following variables and classify each as involved in pulmonary ventilation, external respiration, or internal respiration. Some may be classified in more than one way.

| | | | |
|---------------------------------------|-----------------|------------------|-----------|
| (A-a) $\text{PO}_2 \cdot \text{diff}$ | \dot{V}_T | PvCO_2 | V_D/V_T |
| a-v O_2 diff | \dot{V}_A | PvO_2 | |
| V_D | PaO_2 | $\text{SaO}_2\%$ | |
| f | PaO_2 | $\text{SbO}_2\%$ | |
| \dot{V}_E and \dot{V}_I | PaCO_2 | $\text{SvO}_2\%$ | |

2. Diagram the conductive and respiratory zones of the respiratory system. Compare the function of the two zones.
3. Why does air flow into and out of the lungs?
4. What is the functional difference between pulmonary circulation and bronchial circulation? How does bronchial circulation affect the PaO_2 ?
5. Identify the three capacities and four volumes into which TLC can be divided. Which is most responsive during exercise? Which must be accounted for when one determines body composition by hydrostatic (underwater) weighing?
6. Explain the conditions represented by the volume designations ATPS, BTPS, and STPD. Where is each condition most appropriately used? Which volume is typically the largest? Which is the smallest? Name and explain the gas laws that cause these differences.
7. Discuss the primary control of respiration and the factors that affect such control.
8. Describe how oxygen and carbon dioxide are transported in the circulatory system. Explain the importance of each transport form and any interaction between the movements of the individual gases.
9. Explain why the saturation of venous blood with oxygen (SvO_2) is lower than arterial blood.
10. Explain how the transport and removal of carbon dioxide relate to acid-base balance. Why is it important to maintain

acid-base balance?

11. Graph a normal resting oxygen dissociation curve. What percentage of the available oxygen is normally dissociated at rest?

For further review and study tools, visit Lippincott Connect.

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10 Respiratory Exercise Response, Training Adaptations, and Special Considerations



CHAPTER OUTLINE

Introduction

Response of the Respiratory System to Exercise

Short-Term, Light to Moderate Submaximal Aerobic Exercise

Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

Incremental Aerobic Exercise to Maximum

Static Exercise

Locomotor-Respiratory Coupling (Entrainment) during Exercise

Respiratory Limitations to Exercise

Exercise-Induced Arterial Hypoxemia

Respiratory Muscle Fatigue

Excessive Fluctuations in Intrathoracic Pressures

The Influence of Sex and Age on Respiration at Rest and during Exercise

Male-Female Respiratory Differences

Children and Adolescents

Older Adults

Respiratory Muscle Training Principles and Adaptations

Controlled-Frequency Breathing Training

Whole Body Respiratory Training Principles and Adaptations

Lung Volumes and Capacities

Pulmonary Ventilation

External and Internal Respiration

Why Are There So Few Respiratory Adaptations to Whole Body Exercise Training?

Special Considerations

Altitude

Physical Activity and Pollution

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Graph and explain the pattern of response for the major respiratory variables during short-term, light to moderate

submaximal aerobic exercise.

- Graph and explain the pattern of response for the major respiratory variables during long-term, moderate to heavy submaximal aerobic exercise.
- Graph and explain the pattern of response for the major respiratory variables during incremental aerobic exercise to maximum.
- Graph and explain the pattern of response for the major respiratory variables during static exercise.
- Compare and contrast the pulmonary ventilation, external respiration, and internal respiration responses to short-term, light to moderate submaximal aerobic exercise; long-term, moderate to heavy submaximal aerobic exercise; incremental aerobic exercise to maximum; and static exercise.
- Explain why respiration may be a limitation to performance in elite athletes.
- Differentiate between respiratory muscle training and adaptations and whole body respiratory training and adaptations.
- Identify variations in resting volumes, exercise responses, and exercise training adaptations between males and females and among young adults, children and adolescents, and older adults.
- Determine the value of altitude training and training in polluted conditions.

Introduction

During exercise, the demand for energy increases. The demand varies, of course, with the type, intensity, and duration of the exercise. In most exercise situations, much of the body's ability to respond to the demand for more energy depends on the availability of oxygen. To provide the needed oxygen for aerobic energy production, the respiratory system—including pulmonary ventilation, external respiration, and internal respiration—must respond. Pulmonary ventilation increases to enhance alveolar ventilation, external respiration adjusts to maintain the relationship between ventilation and perfusion in most cases, and internal respiration responds with an increased extraction of

oxygen by the muscles. These changes in respiration not only provide adequate oxygenation for the muscles but also play a major role in maintaining acid-base balance, which is, in turn, closely related to carbon dioxide levels.

In general, all levels of respiratory activity are precisely matched to the rate of work being done. Furthermore, because of this precise control and the large reserve built into the system, respiration in normal, healthy, sedentary, or moderately fit individuals is generally not a limiting factor in activity. This is true despite the perception of feeling out of breath during exercise. Occasionally, however, the capacities of the cardiovascular and metabolic systems exceed that of the respiratory system such that respiration can be considered a limitation to primarily high-intensity or maximal work and, paradoxically, that happens most frequently, but not exclusively, in elite highly trained athletes.

Of course, changes in pulmonary ventilation would be of little benefit if parallel changes in pulmonary blood volume and flow and total body systemic circulation did not also occur. These accompanying cardiovascular responses will be detailed in the chapters in the cardiovascular unit.

This chapter concentrates on pulmonary ventilation, external respiration, and internal respiration responses to aerobic activity, including short- and long-term, constant-load submaximal activity and incremental exercise to maximum. The most prominent changes in the respiratory system occur within these classifications of activity. Static exercise responses will also be briefly discussed. However, respiratory responses to dynamic resistance activity and high-intensity anaerobic exercise have not been specifically documented and, therefore, cannot be discussed here. When reading this discussion and studying the accompanying graphs, you may wish to refer to the glossary of respiratory symbols in **Table 10.1** and to review the way that oxygen is taken into the body (pulmonary ventilation), absorbed into the blood stream (external respiration), and delivered to cells of the body to be used in cellular respiration as presented in **Figure 9.1**. Note that several variables can be considered as part of more than one process.




TABLE 10.1 Respiratory Symbols

| Pulmonary Ventilation | External Respiration | Internal Respiration |
|---|--|---|
| \dot{V}_E = minute ventilation | \dot{V}_A alveolar ventilation | a-vO ₂ diff = amount of oxygen carried in the arteries minus the amount carried in the veins |
| V_D = dead space | P_AO_2 = partial pressure of oxygen at the alveoli | PaO_2 = partial pressure of oxygen in the arterial blood |
| V_T = tidal volume | PaO_2 = partial pressure of oxygen in the arterial blood | $PaCO_2$ = partial pressure of carbon dioxide in the arterial blood |
| f = frequency | (A-a)PO ₂ diff = oxygen or PO ₂ pressure gradient between the alveoli and the arteries | $PvCO_2$ = partial pressure of carbon dioxide in venous blood |
| V_D/V_T = ratio of dead space to tidal volume | $SaO_2\%$ = percent saturation of arterial blood with oxygen | $SvO_2\%$ = percent saturation of venous blood with oxygen |
| | P_ACO_2 = partial pressure of carbon dioxide at the alveoli | PvO_2 = partial pressure of oxygen in venous blood |

Response of the Respiratory System to Exercise

Short-Term, Light to Moderate Submaximal Aerobic Exercise

The responses of major pulmonary variables to short-term (5–10 minutes), light to moderate submaximal (30–69% of maximal work capacity) aerobic exercise are shown in **Figure 10.1**. Variables related to pulmonary ventilation are shown in the first column, variables related to external respiration are presented in the middle column, and variables related to internal respiration are shown in the third column of figures.

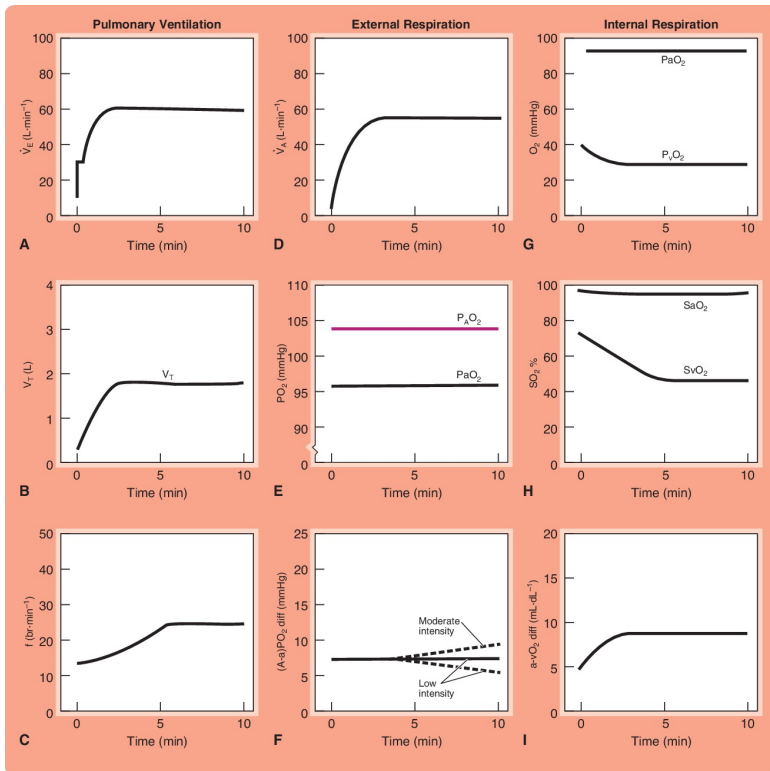


Figure 10.1 Responses of Respiratory Variables to Short-Term, Light to Moderate Submaximal Aerobic Exercise.

A. Minute ventilation (\dot{V}_E). B. Tidal volume (V_T). C.

Frequency (f). D. Alveolar ventilation (\dot{V}_A). E. Partial pressure of oxygen at the alveoli (P_{AO_2}) and in arterial blood (P_{aO_2}). F. Partial pressure difference between the alveoli and the arteries ($(A-a)PO_2\text{diff}$). G. Partial pressure of oxygen in arterial blood (P_{aO_2}) and venous blood (P_{vO_2}). H. Percent saturation of arterial blood (S_{aO_2}) and venous blood with oxygen (S_{vO_2}). I. Arterio-venous oxygen difference ($a-vO_2\text{diff}$).

Pulmonary Ventilation

The most obvious response to an increased metabolic demand,

such as exercise, is the increase in pulmonary minute ventilation (\dot{V}_E L·min⁻¹), called hyperpnea. What is perhaps a little surprising is the initial immediate reaction. In **(Figure 10.1A)**, note that between the onset of exercise at 0 and 2 minutes into

the exercise, a triphasic response in \dot{V}_E occurs. Within the first respiratory cycle at the onset of exercise, there is an initial abrupt

increase in \dot{V}_E , termed phase 1. This increase is maintained for approximately 10–20 seconds. Phase 2 is a slower exponential rise from the initial elevation to a steady-state leveling off. At the low to moderate workload depicted here, this exponential rise is generally completed in 2–3 minutes. At this point, phase 3, a new steady state, is achieved. The actual level of this achieved exercise steady state depends on a number of factors, including the workload, the fitness status of the individual, and the environmental conditions. In the time span depicted in this graph, the steady-state level is maintained. The three-phase

response at the onset of activity is typically not seen when \dot{V}_E is reported or graphed minute by minute, rather than second by second or even breath by breath, but it does occur (Bell, 2006; Pardy et al., 1984; Whipp, 1977; Whipp and Ward, 1980; Whipp et al., 1982).

The initial rise in ventilation occurs primarily because of an increase in tidal volume (Leff and Schumacker, 1993). Theoretically, tidal volume ranges from the resting level to the limits of vital capacity (VC). In reality, rarely is more than 50–65% of VC reached before a plateau occurs. Furthermore, although tidal volume encroaches into both the inspiratory reserve volume (IRV) and the expiratory reserve volume (ERV), it encroaches much more into IRV than ERV (Koyal et al., 1976; Pearce and Milhorn, 1977; Turner et al., 1968; Younes and Kivinen, 1984).

At light to moderate workloads, the contribution of increased breathing frequency to increased minute ventilation is minimal and gradual. Both tidal volume and frequency level off at a steady state that satisfies the oxygen requirements to perform the metabolic work necessary for short submaximal activity (**Figure 10.1B and C**).

Airway resistance decreases because of bronchodilation as soon as exercise begins. Likewise, the ratio of dead space (V_D) to tidal volume (V_T) decreases, and in this case, the largest changes are evident at the lowest work rate (Wasserman et al., 1967; Whipp and Ward, 1980). This resulting drop in V_D/V_T is moderate at low to moderate exercise intensities. The V_D itself changes minimally with bronchodilation, but with the proportionally larger increase in V_T , the ratio declines (Grimby, 1969). This result is important because alveolar ventilation (\dot{V}_A) thus increases from about 70% of the total pulmonary ventilation at rest to a higher percentage during exercise. Since \dot{V}_A is the critical ventilation in terms of facilitating gas exchange with the blood in the pulmonary capillaries, the reduction in the V_D/V_T ratio means that the appropriate level of \dot{V}_A can be achieved with a smaller rise in \dot{V}_E than would be needed if the ratio did not change (Wasserman and Whipp, 1975).

External Respiration

Exercise responses of key variables of external respiration are shown in panels D–F of **Figure 10.1**. The \dot{V}_A response to low to moderate exercise is depicted in **Figure 10.1D**. This curve parallels the change in \dot{V}_E , except that the initial adjustments seen in \dot{V}_E are not depicted for \dot{V}_A . The rise in \dot{V}_A is sufficient to maintain PO_2 at the alveolar level (PAO_2) at a constant level during short-term submaximal exercise (**Figure 10.1E**). Maintenance of PAO_2 is important because it represents the driving force for oxygen transfer across the alveolar-capillary interface (Powers et al., 1993; Wasserman, 1978).

As explained in **Chapter 9**, under resting conditions, there is a difference in PO_2 between the alveoli (PAO_2) and systemic arterial blood (PaO_2) owing to the dilution of the systemic arterial blood with the bronchial venous blood. During short-term, low-intensity submaximal exercise, PaO_2 is maintained. The alveolar to arterial oxygen partial pressure difference, depicted as

(A-a)PO₂diff in **Figure 10.1F**, either does not change or decreases slightly (Jones, 1975; Leff and Schumacker, 1993; Wasserman and Whipp, 1975). At moderate workloads, a slight increase may occur. The (A-a)PO₂diff reflects the efficiency and/or adequacy of oxygen transfer in the lungs during exercise. At the steady-state submaximal levels described here, there is no noticeable change in this efficiency.

Internal Respiration

Gas exchange and blood perfusion in the lungs during low to moderate exercise are sufficient to maintain the saturation of red blood cells with oxygen (SaO₂%) within a narrow range approximating resting levels **Figure 10.1H** (Gurtner et al., 1975).

Recall that internal respiration involves the dissociation of oxygen from the red blood cells so that it may diffuse down the pressure gradient into the muscles and other tissues. Before detailing the exercise response of internal respiration variables, it is important to understand how oxygen moves from the systemic capillaries to the cells.

Refer to **Figure 10.2** and refamiliarize yourself with the resting relationships for oxygen dissociation described in **Chapter 9** and labeled in this figure. The perpendicular line on the x-axis at 95 mmHg represents the PaO₂ for resting arterial blood. It intersects with the middle curve and indicates a percent saturation of hemoglobin (SbO₂%) of approximately 97% (seen on the left y-axis). The perpendicular line on the x-axis at 40 mmHg represents the PvO₂ for resting venous blood. This line also intersects with the middle curve and indicates a SbO₂ of 75%. This indicates that at rest, 75% of the oxygen remains associated with the red blood cells in venous blood. Stated another way, under resting conditions, only about 23% of oxygen is dissociated from hemoglobin and diffuses out of the capillaries and into the cells of the body. This means that much more oxygen can be extracted when it is needed to do cellular work, and this is precisely what happens during exercise. Recall that in the metabolic chapter, maximal oxygen consumption was defined as the maximal amount of oxygen that could be taken in, transported, and utilized to produce ATP during heavy exercise. Pulmonary ventilation explained how oxygen is taken in, and the

cardiovascular system is responsible for transporting the oxygen bound to hemoglobin. But for oxygen to be available to be “consumed” by the cells, it must dissociate from hemoglobin and diffuse across the capillary wall into a cell. Once the oxygen has been taken up, or consumed, by the cells (for our purposes mostly skeletal muscle cells), then there is less oxygen in the venous blood as it leaves the capillaries.

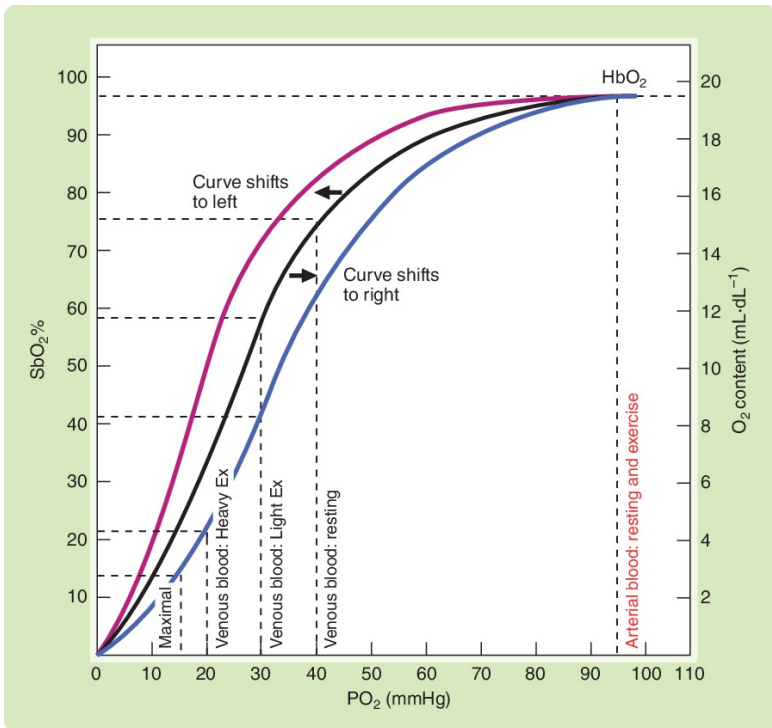


Figure 10.2 Oxygen Dissociation during Exercise.

The center curve represents normal resting values: $PaCO_2 = 40$ mmHg, $pH = 7.4$, body temperature = $37^\circ C$. During all intensities and types of exercise, the $PaCO_2$ increases, pH decreases (becomes more acidic), and body temperature increases. Each of these conditions causes the curve to shift to the right, with the result that more oxygen is dissociated from red blood cells to be used by the muscles.

Consequently, the higher the intensity of exercise, the lower

both the oxygen content of the venous blood and the $\text{SbO}_2\%$ in venous blood.

Four factors are involved in the increased oxygen extraction during exercise:

1. Increased PO_2 gradient
2. Increased PCO_2
3. Decreased pH
4. Increased temperature

Each of these factors is depicted in the oxygen dissociation curve in **Figure 10.2**. The first factor, an increased PO_2 gradient (seen along the x-axis), occurs as more oxygen is used to produce the additional energy needed during exercise. Factors 2, 3, and 4 (an increased PCO_2 , decreased pH, and increased temperature) all cause the oxygen dissociation curve to shift to the right. This shift is indicated in **Figure 10.2** by the arrow pointing to the right-hand curve. As the oxygen dissociation curve is shifted to the right, more oxygen is extracted (or dissociated so it can be used by the muscle cells) for any given PO_2 . How each factor operates specifically to increase oxygen extraction is described below.

INCREASED PO_2 GRADIENT Under resting conditions, sufficient oxygen remains in the muscle tissue to maintain a PO_2 of 40 mmHg. When exercise increases the demand for energy, the oxygen already present in the muscle tissue is used immediately. Since the oxygen is used, the muscle tissue partial pressure is reduced. The PO_2 of arterial blood remains unchanged, as described earlier because of an increase in pulmonary ventilation and blood flow. This is a key element of understanding how the respiratory system responds to exercise. The PaO_2 is unchanged but the PvO_2 decreases as oxygen moves into the muscle. The pressure gradient can widen from 55 mmHg at rest (95 mmHg at the arterial end of the capillary minus 40 mmHg in the resting muscle tissue) up to possibly 65 mmHg (95 mmHg at the arterial end of the capillary minus 30 mmHg in the exercising muscle tissue) during light submaximal exercise.

Equilibrium is reached between the muscle tissue and the

blood by the venous end of the capillary (based on Henry's law), so the venous blood has a PO_2 equal to the PO_2 in the exercising muscle. Refer to **Figure 10.2** and find the line labeled light exercise venous blood above the x-axis value of 30 mmHg. Assume for the moment that this is the only change that occurs (a false assumption, as will be seen later, but which does no harm here). In the figure, you can see the effect this increased pressure gradient (due to decrease in PvO_2) has on the dissociation of oxygen. By following this light exercise venous blood line to where it intersects the solid line middle curve, and then to the left y-axis, you can see that the corresponding $SbO_2\%$ (actually $SvO_2\%$ now) is approximately 59%, not 75%. Instead of 23% of the oxygen being dissociated as it was at rest ($97\% SaO_2\% - 75\% SvO_2\% = 22\%$; $22\% \div 97\% = 23\%$), now 39% ($97\% SaO_2\% - 59\% SvO_2\% = 38\%$; $38\% \div 97\% = 39\%$) has been released from the red blood cells just because of the change in the pressure gradient. Thus, an additional 16% of the available oxygen ($39\% - 23\% = 16\%$) has been dissociated and is used for energy production. Because all submaximal exercise does not require the same amount of oxygen, these numbers simply illustrate the effect of a widening pressure gradient.

INCREASED PCO_2 When oxygen is used to provide energy, carbon dioxide is produced as a by-product. Increasing levels of carbon dioxide mean that PCO_2 increases. An increase in PCO_2 shifts the oxygen dissociation curve to the right. This shift occurs in a minimal way even at rest, as seen in the venous curve in **Figure 9.18**. However, during exercise, the shift is further to the right. Exactly how far the curve shifts depends on the intensity of the exercise and how much carbon dioxide is produced. The shift in the curve to the right means that at any given PO_2 , more dissociation occurs, as shown in **Figure 10.2**. Follow the line labelled light exercise venous blood from the value of 30 mmHg on the PO_2 x-axis until it intersects the right-hand curve. A horizontal line from this intersection to the left y-axis yields a saturation value of about 41%. Thus, more oxygen is dissociated than would have been if the pressure gradient had been operating alone. **Figure 10.3** emphasizes that the actual shift occurs during the red blood cell transit through the capillary before reaching the venous blood vessel.

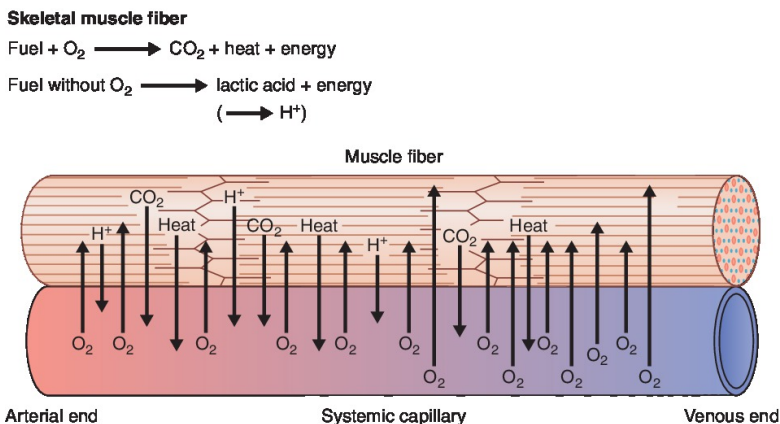


Figure 10.3 Factors Influencing Oxygen Dissociation during Exercise.

The by-products of the production of energy—namely, carbon dioxide, heat, and hydrogen ions (H⁺)—in skeletal muscle fibers stimulate the dissociation of oxygen from red blood cells as they traverse systemic capillaries.

DECREASED pH OR INCREASED HYDROGEN ION CONCENTRATION Hydrogen ions (H⁺) come from two primary sources during exercise. First, carbon dioxide combines with water to form carbonic acid. The carbonic acid then breaks down into hydrogen ions and bicarbonate. Second, increased glycolysis results in additional lactate and hydrogen ions. The presence of increased levels of hydrogen ions lowers the pH to a more acidic level. This more acidic pH shifts the oxygen dissociation curve to the right in the same way that an increased PCO₂ does and with the same results: a greater dissociation of oxygen from red blood cells. How far to the right the curve shifts depends on the amount of hydrogen ions released. As with carbon dioxide, this effect is taking place during the red blood cells transit through the muscle capillaries (**Figure 10.3**). The effect of carbon dioxide and pH on the affinity of hemoglobin for oxygen is known as the *Bohr effect* (Hall and Hall, 2021; Kenney, 1982).

INCREASED TEMPERATURE Heat is another by-product of muscle energy production. Heat is transferred from the muscle tissue (high heat) to the capillary (low heat) (**Figure 10.3**). The

resultant rise in temperature shifts the oxygen dissociation curve to the right. The action is precisely the same as that of the increased PCO₂ and decreased pH, and so it is again depicted by the same rightward shift in **Figure 10.2**. Thus, the elevation in the use of oxygen to produce energy during exercise and the by-products of that energy production operate together to make the reserves of oxygen available: the first (increased oxygen consumption) by widening the pressure gradient and the other three (increased PCO₂, decreased pH, and increased body temperature) by shifting the oxygen dissociation curve to the right.

The exercise responses for the respiratory variables that reflect internal respiration, and that are directly related to the oxygen dissociation curve, are depicted in **Figure 10.1G–I**. Because PaO₂ and SaO₂% (**Figure 10.1G and H**) do not change with short-term, low-intensity exercise, the actual amount of oxygen being carried in the arteries (in milliliters per deciliter) also does not change ([Wasserman et al., 1967](#)). However, because muscle energy production uses more oxygen during exercise, the venous oxygen values, for PvO₂ and SvO₂% (**Figure 10.1G and H**), decrease. With an equal arterial oxygen and lower venous oxygen content, the a-vO₂diff (**Figure 10.1I**) increases. Because this is a steady-state submaximal situation, the a-vO₂diff will level off when sufficient oxygen is being extracted to supply the needs of the cell ([Davies et al., 1972](#); [Dempsey et al., 1977](#); [Kao, 1974](#)). The arteriovenous oxygen difference (a-vO₂diff) is the best overall indicator of internal respiration ([Gurtner et al., 1975](#)).

Because energy production also produces carbon dioxide, PvCO₂ also increases. The extra carbon dioxide is exhaled easily from the lungs, and the hyperpnea of exercise may even blow off a little extra carbon dioxide, resulting in a slight decrement in PaCO₂. Regulation of ventilation maintains PaCO₂ very close to resting values ([Davies et al., 1972](#); [Dempsey et al., 1977](#); [Kao, 1974](#); [Wasserman et al., 1967](#); [Whipp and Ward, 1980](#)).

These combined responses are well within the reserve capacity of the respiratory system for normal, healthy individuals.

Long-Term, Moderate to Heavy Submaximal

Aerobic Exercise

Respiratory responses to long-term, moderate to heavy (60–75% of maximal working capacity) aerobic exercise are similar to those changes just discussed for short-term, light to moderate submaximal exercise but, for most variables, they differ in magnitude. In addition, several of the variables have a drifting pattern, where the values drift slightly higher or lower after approximately 30 minutes of exercise.

Pulmonary Ventilation

Figure 10.4A shows that \dot{V}_E increases to a higher level than during light to moderate submaximal exercise before plateauing at a steady state. Achievement of the steady state may take somewhat longer than at lower work intensities and often is not held throughout the duration of the exercise. Note that after

about 30 minutes, a gradual rise in \dot{V}_E occurs, despite an unchanging workload, an effect called *ventilatory drift* (Dempsey et al., 1977; Hanson et al., 1982; Wasserman, 1978). The precise reason for this drift is unknown (Sawka et al., 1980), although a rising body temperature is most often speculated as the primary reason. This drift is both inefficient and advantageous. It is inefficient because the extra air breathed is in excess of the workload demand. It is advantageous for gas exchange because alveolar ventilation parallels the drift, and acid-base balance is maintained (Dempsey et al., 1977; Hanson et al., 1982).

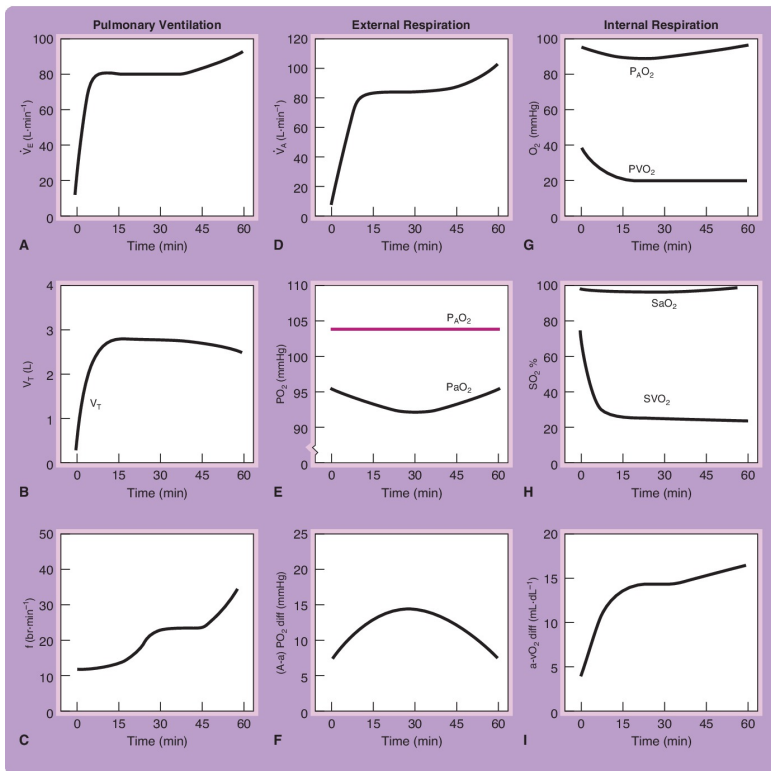


Figure 10.4 Responses of Respiratory Variables to Long-Term, Moderate to Heavy Submaximal Aerobic Exercise.

A. Minute ventilation (\dot{V}_E). **B.** Tidal volume (V_T). **C.** Frequency (f). **D.** Alveolar ventilation (\dot{V}_A). **E.** Partial pressure of oxygen at the alveoli (P_{AO_2}) and in arterial blood (P_{aO_2}). **F.** Partial pressure difference between the alveoli and the arteries ($(A-a)PO_2\text{diff}$). **G.** Partial pressure of oxygen in arterial blood (P_{aO_2}) and venous blood (P_{vO_2}). **H.** Percent saturation of arterial blood (S_{aO_2}) and venous blood with oxygen (S_{vO_2}). **I.** Arterio-venous oxygen difference ($a-vO_2\text{diff}$).

As for lower-intensity submaximal exercise, the initial change in \dot{V}_E is due primarily to an increase in V_T (Figure 10.4B).

However, in the later stages of heavy submaximal work, tidal volume may decrease slightly. The drift in \dot{V}_E comes about primarily as a result of increased breathing frequency (**Figure 10.4C**). The V_D/V_T ratio still decreases primarily at the onset of activity, but it does so to a greater extent with a heavier workload than at lighter loads.

External Respiration

As stated earlier, the drift in \dot{V}_E is paralleled by a similar drift in **Figure 10.4D**). PAO_2 remains constant (**Figure 10.4E**), as it did with lower-intensity submaximal work. However, PaO_2 decreases slightly (**Figure 10.4E**) until about the time when ventilatory drift begins, forming a shallow U-shaped curve ([Dempsey et al., 1977](#)). It then returns toward baseline values. This variation in PaO_2 while PAO_2 remains unchanged is reflected in a mirror image relationship in the $(A-a)PO_2\text{diff}$ (**Figure 10.4F**), depicted as a truncated, inverted U-shaped curve. The initial increase in the $(A-a)PO_2\text{diff}$ indicates inefficiency in gas exchange. However, the small loss of efficiency has very little practical meaning and does not limit the exercise ([Hanson et al., 1982](#); [Wasserman et al., 1967](#)).

Internal Respiration

Other than differences in magnitude, all of the internal respiration variables respond in the same way during long-term, moderate to heavy submaximal dynamic exercise as during shorter, lighter dynamic exercise, as previously described.

Despite the higher workload and the shallow U-shaped response, PaO_2 is relatively constant (**Figure 10.4G**). Because of the heavier workload, more oxygen is dissociated and used, and the PvO_2 and $SvO_2\%$ (**Figure 10.4G and H**) decrease to lower levels than in shorter, lighter workloads. The result is an increase in the $a-vO_2\text{diff}$ (**Figure 10.4I**). The factors responsible for the dissociation of oxygen are the same as those for lower-intensity exercise. $PaCO_2$ decreases slightly due to the increased volume of air being exhaled. $PvCO_2$ increases because the greater use of oxygen to produce energy also results in more carbon dioxide

being carried in the venous system to the lungs to be exhaled.

Incremental Aerobic Exercise to Maximum

Incremental aerobic exercise to maximum consists of a series of progressively increasing work intensities, which stop when the individual cannot do any more work. The length of each work intensity, often called a stage, generally varies from 1 to 3 minutes to allow for the achievement of a steady state at that level, at least until the higher workloads, when a steady state cannot be either attained or maintained.

Pulmonary Ventilation

It might be anticipated, because \dot{V}_E rises and levels off at submaximal workloads, that this rise would be proportional, direct, and rectilinear throughout the entire range from rest to maximal exercise. However, as shown in **Figure 10.5A**, the response is not a smooth rise. At light to moderate and even heavy workloads, up to approximately 50–75% of maximum

workload, \dot{V}_E does increase in a rectilinear fashion. At this point, a break in the linearity occurs, and a second, steeper linear rise ensues. This proportional rise continues until approximately 85–95% of maximum workload, when a second break in linearity occurs. The slope of the third linear rise to maximum that follows is even steeper (Koyal et al., 1976; Powers and Beadle, 1985; Wasserman, 1978). These points where the rectilinear rise in minute ventilation breaks from linearity during incremental exercise to maximum have had a variety of names. The first breakpoint has been called the “point of optimal ventilatory efficiency” reflecting the point at which a maximum amount of oxygen can be taken up with a minimum of ventilation. The second breakpoint is often called the “respiratory compensation point” reflecting a relative hyperventilation (Binder et al., 2008). However, in this text, we use the most acceptable simple terminology that does not imply causation, that is, **ventilatory thresholds**. The first breakpoint is called the first ventilatory threshold (VT1), and the second breakpoint is called the second

ventilatory threshold (VT2).

Ventilatory Thresholds Points where the rectilinear rise in minute ventilation breaks from linearity during an incremental exercise to maximum.

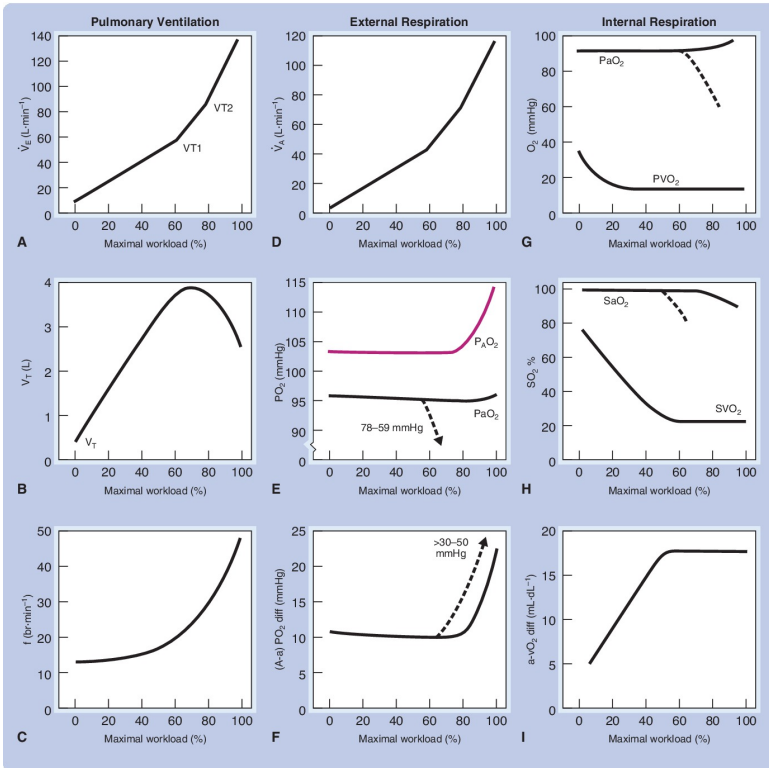


Figure 10.5 Responses of Respiratory Variables to Incremental Aerobic Exercise to Maximum.

A. Minute ventilation (\dot{V}_E). B. Tidal volume (V_T). C. Frequency (f). D. Alveolar ventilation (\dot{V}_A). E. Partial pressure of oxygen at the alveoli (P_{AO_2}) and in arterial blood (P_{AO_2}). F. Partial pressure difference between the alveoli and the arteries ((A-a)PO₂diff). G. Partial pressure of oxygen in arterial blood (P_{aO_2}) and venous blood (P_{vO_2}).

H. Percent saturation of arterial blood (SaO_2) and venous blood with oxygen (SvO_2). I. Arterio-venous oxygen difference (a-vO_2 diff).

Precisely what causes these breakpoints is unknown. One theory has linked ventilatory breakpoints with an excess of carbon dioxide resulting from the buffering of lactic acid; this theory calls the breakpoints anaerobic thresholds. Although carbon dioxide is a known respiratory stimulator, this theory is probably not completely accurate (for reasons that are fully discussed in the unit on metabolism). Therefore, the term “ventilatory threshold” is preferable. Other possible mechanisms—including catecholamine or potassium stimulation of the carotid bodies; limitations in changes in V_T , frequency, and V_D/V_T to maintain \dot{V}_A ; increasing body temperatures; and feedback from the skeletal muscle proprioceptors—have been suggested as causes of the VTs, but their role remains unproven. A combination of factors likely is responsible (Loat and Rhodes, 1993; Skinner and McLellan, 1980; Walsh and Banister, 1988).

Knowledge of the VTs, regardless of why they occur, has some practical benefit. The workloads at which the VTs occur are related to endurance exercise training and performance. VT1 is thought to indicate the upper boundary of moderate exercise and VT2 to separate heavy but sustainable exercise intensity from very heavy nonsustainable intensity. This means that VT1 and VT2 can be used for exercise prescription. That is, training at work rates between VT1 and VT2 would stimulate aerobic metabolism but allow for activity of long duration (Neder and Stein, 2006). It also means that the VTs can be useful for predicting performance (Amann et al., 2006). The higher the workload where the breaks occur, the greater the intensity of activity that can be sustained (Loat and Rhodes, 1993; Walsh and Banister, 1988).

As described for submaximal exercise, the changes in minute ventilation (\dot{V}_E) during low to moderate workloads are achieved primarily by an increase in V_T (Figure 10.5B). At very heavy workloads, however, the depth of breathing may actually

decrease, forming a truncated, inverted U pattern (Dempsey, 1986; Younes and Kivinen, 1984). When V_T reaches its highest point, any further increase in ventilation can occur only as the result of an increased breathing frequency (Wasserman, 1978). The rise in breathing frequency is exponential at the higher work levels (Figure 10.5C). As with submaximal workloads, the V_D/V_T ratio decreases the most in the initial light to moderate stages of the incremental work (Grimby, 1969). The maximal reduction is reached at about 60% of maximal work, and this value is maintained to maximum.

External Respiration

Changes in \dot{V}_A parallel the changes in \dot{V}_E , including the slope of the rectilinear rises and the two breakpoints (Figure 10.5D).

The breakpoints in \dot{V}_A , however, occur before the breakpoints in \dot{V}_E —that is, at slightly lower percentages of maximal work (Jones, 1975; Wasserman, 1978).

The rise in \dot{V}_A is sufficient to maintain PAO_2 and subsequently PaO_2 through the light to moderate submaximal exercise workloads (Figure 10.5E). As the workload becomes higher, PAO_2 rises exponentially to maximum. This rise provides the driving force to reach an equilibrium of alveolar gas with mixed venous blood and, in so doing, maintains PaO_2 (solid line) within narrow limits in normal or moderately fit individuals (Figure 10.5E) (Dempsey, 1986; Grimby, 1969; Segal, 1992; Wasserman and Whipp, 1975; Wasserman et al., 1967). The $(A-a)PO_{2diff}$ follows the pattern of the exponential rise in PAO_2 (Figure 10.5F) (Amann, 2012; Jones, 1975).

Internal Respiration

The dissociation or release of oxygen reaches its limit during incremental exercise to maximum as this is the condition under which the most oxygen must be supplied to the muscle to support ATP production. At low and moderate workloads, more and more oxygen is released, as depicted by the sigmoid-shaped curve in

Figure 10.2. The factors that stimulate the dissociation of oxygen during incremental exercise to maximum are the same as previously described, but they typically are greater in magnitude, that is, the pressure gradient is wider, PCO_2 is higher, pH is lower, and temperature is higher as the exercise load increases (**Figure 10.5G**) (Richardson et al., 1995a, b; Wagner, 2006).

The ability to sustain PaO_2 (**Figure 10.5G**) during heavy exercise means that the blood is nearly 100% saturated with oxygen under normal conditions. On the other hand, venous saturation ($\text{SvO}_2\%$) of oxygen drops to approximately 15% (**Figure 10.5H**) as oxygen is taken up by the exercising muscle. The $\text{a-vO}_2\text{diff}$ (**Figure 10.5I**) parallels the changes in oxygen dissociation, gradually increasing with the incremental workloads until it can increase no more. It plateaus at approximately 50–60% of the maximal workload (Rowell, 1969; Saltin, 1969). Thus, even maximal exercise is well within the respiratory reserves for most individuals.

As more oxygen is used to produce more energy, more carbon dioxide is produced during maximal exercise than during submaximal exercise. This metabolic carbon dioxide as well as nonmetabolic carbon dioxide produced in the effort to buffer the lactate and H^+ that accumulates at the higher workloads results in a rise in PvCO_2 . Conversely, PaCO_2 is maintained at first and then decreases. This fall in arterial carbon dioxide partial pressure reflects the excess removal of carbon dioxide brought about by alveolar hyperventilation (Dempsey et al., 1977; Grimby, 1969; Jones, 1975).

FOCUS ON APPLICATION | *Clinically Relevant*

Breathing Patterns during Exercise

Beginning exercisers often ask how and when they should breathe. The best advice for most land exercise appears to be to breathe in whatever pattern comes naturally, whether spontaneously or synchronized with the movement. The primary exception is weight training. The static component of

weight training can cause the **Valsalva's maneuver**, a breath-holding action that involves closing of the glottis (which keeps air in the lungs) and contraction of the diaphragm and abdominal musculature. The result is an increase in intra-abdominal pressure and a large increase in blood pressure. Individuals may become light-headed or faint. Blood pressure remains much lower when breathing during the contraction. For this reason, it is generally recommended to inhale during the lowering phase of a resistance exercise and to exhale during the lifting phase of each repetition (Fleck and Kraemer, 2004; Narloch and Brandstater, 1995).

Valsalva's Maneuver Breath holding that involves closing of the glottis and contraction of the diaphragm and abdominal musculature.







Static Exercise

Static exercise involves the production of force or tension with no mechanical work being done. Therefore, gradations of static exercise are usually expressed relative to the individual's ability to produce force in a given muscle group (called the maximal voluntary contraction, or MVC) held for a specified period of time. For example, an individual might perform a 30% MVC for 5 minutes.

Figure 4.5 (in [Chapter 4](#)) presents respiratory and metabolic responses to heavy static exercise. Note that, despite this being “heavy” static activity, the rise in $\dot{V}O_2$ representing energy cost is minimal. As always, pulmonary ventilation (\dot{V}_E) increases to provide the needed additional oxygen. At the onset of static exercise, however, the initial (0–2 minutes) three-phase response in \dot{V}_E that occurs in aerobic exercise is absent. The a-vO₂diff either remains the same or decreases slightly during static exercise. This is undoubtedly related to the occlusion of blood flow in statically contracting muscles. For both \dot{V}_E and a-vO₂diff, a rebound rise occurs in recovery. In all other aspects, the respiratory responses are similar in static exercise and in low-intensity, aerobic exercise. Static exercise does not push the reserve capacity of the respiratory system ([Asmussen, 1981](#)).

Table 10.2 summarizes the respiratory responses to exercise discussed in preceding sections.

TABLE 10.2 Normal Respiratory Responses to Exercise

| | Short-Term, Light to Moderate Submaximal Aerobic Exercise  | Long-Term, Moderate to Heavy Submaximal Aerobic Exercise [†]  | Incremental Aerobic Exercise to Maximum  | Static Exercise  |
|------------------------------|---|---|---|---|
| Pulmonary Ventilation | | | | |
| \dot{V}_E | Increases rapidly; plateaus | Increases rapidly; plateaus; positive drift | Shows initial rectilinear rise; has two breakpoints | Minor gradual increase; rebound rise in recovery All responses the same as for short-term, light to moderate submaximal exercise |
| V_D | Decreases | Decreases | Decreases | |
| V_T | Increases rapidly; plateaus | Increases rapidly; plateaus | Has truncated, inverted U-shaped curve; increases greatly; has incomplete reversal | |
| f | Slowly increases; plateaus | Increases slowly; plateaus; positive drift | Positive curvilinear rise | |
| V_D/V_T | Decreases initially; plateaus | Decreases rapidly initially; plateaus | Decreases rapidly initially; levels off at 60% of maximum and is maintained | |
| External Respiration | | | | |
| \dot{V}_A | Increases rapidly; plateaus | Increases rapidly; plateaus; positive drift | Shows initial rectilinear rise; has two breakpoints | |
| P_{iO_2} | Shows no change | Shows no change | Shows no change until approximately 75% of maximum; then positive exponential rise | |
| P_{aO_2} | Shows no change | Has small U-shaped curve | Shows no change until approximately 75% of maximum; then increases slightly [†] | |
| (A-a) PO_2 diff | Decreases slightly or shows no change (light); increases slightly (moderate) | Has truncated, inverted U-shaped curve; increases rapidly initially; has incomplete reversal | Shows no change until approximately 75% of maximum; then positive exponential rise [†] | |
| $SAO_2\%$ | Decreases less than 1%; has U-shaped curve | — | Remains steady until approximately 75% of maximum; then declines slightly [†] | |
| Internal Respiration | | | | |
| $PaCO_2$ | Is level; then decreases slightly | Is level; then decreases slightly | Is level; then decreases | Shows no change or decreases slightly No change during; rebound rise in recovery |
| $PvCO_2$ | Shows slight linear rise | Shows a gradual linear rise; plateaus | Has sharp rise; levels slightly | |
| PvO_2 | Decreases rapidly; plateaus | Decreases rapidly; plateaus | Decreases sharply; levels off | |
| $SV_{O_2}\%$ | Decreases initially; plateaus | Decreases initially; plateaus | Decreases sharply; never reaches 0 | |
| a- vO_2 diff | Increases rapidly; plateaus | Increases rapidly; plateaus; positive drift | Shows rectilinear rise to 40–60% $\dot{V}O_{2\max}$; plateaus | |

*Resting values are taken as baseline.

†The difference between leveling during the short-term, light to moderate and long-term, moderate to heavy submaximal exercise responses is one of magnitude; that is, leveling occurs at a higher value with higher intensities.

‡Exercise-induced arterial hypoxemia may change the response.

Locomotor-Respiratory Coupling (Entrainment) during Exercise

Entrainment is defined as frequency and phase locking between two periodic (rhythmic) systems. In some but not all individuals,

the performance of rhythmical exercise (such as walking, running, cycling, and rowing) is accompanied by a synchronization of limb movement and breathing frequency, a form of entrainment known as **locomotor-respiratory coupling (LRC)** (Hill et al., 1988; Mahler et al., 1991; O'Halloran et al., 2012; Sporer et al., 2007). For example, an individual may always inhale during the recovery phase of rowing and always exhale during the drive portion of the stroke. Or a walker, runner, or cyclist may always exhale during the push-off phase of one leg or the other. In these patterns, the same number of locomotor movements occurs per breath. Entrainment appears to occur most frequently in experienced runners working at high intensities. Unlike swimming, in which breathing coordination is a function of head placement during the stroke as a learned response, LRC entrainment occurs without conscious thought. Not all individuals entrain during exercise; not all who do entrain use the same pattern. Humans frequently use LRC movements per breath ratios of 2:1, 2.5:1, 3:1, or 4:1, although in running the 2:1 foot strike to breath pattern often predominates (Daley et al., 2013).

Locomotor-Respiratory Coupling (LRC) A form of entrainment that involves the synchronization of limb movement and breathing frequency that accompanies rhythmical exercise.

The precise mechanisms responsible for LRC are not known although mechanical consequences of locomotion (e.g., the impact on internal structures surrounding the respiratory muscles caused by foot strikes) and neural control mechanism including central locomotor control and peripheral feedback (from extremity muscle action) have been suggested (Daley et al., 2013; Hoffman et al., 2012; Romer et al., 2012).

Entrainment may serve a number of beneficial physiological functions including reducing the work of ventilator muscles, delaying respiratory muscle fatigue, and/or improving respiratory efficiency (Daley et al., 2013). Indeed, individuals who entrain naturally have a slightly improved ventilatory efficiency (Bonsignore et al., 1998) and a lower-energy cost during exercise

when they entrain but not when they breathe randomly. Subjects forced to breathe in specific entrainment patterns rather than being allowed to breathe spontaneously do not exhibit any reduction in energy cost or perceive any less breathing effort with entrained breathing (MacIennan et al., 1994). Conversely, an increase in LRC and a corresponding decrease in energy consumption have been shown when individuals were instructed to cycle in cadence with an auditory stimulation whose rhythm corresponded to the preferred limb movement frequency (Hoffman et al., 2012).

Respiratory Limitations to Exercise

As has been discussed in the previous sections, the respiratory system of most healthy young to middle-aged individuals is usually not considered a major limiting factor for high-intensity aerobic exercise. The small change in $(A-a)PO_2\text{diff}$ indicates an adequate rate of O_2 diffusion across the alveolar-capillary membrane. Furthermore, alveolar hyperventilation can increase sufficiently to raise alveolar PAO_2 high enough to compensate for the widened $(A-a)PO_2\text{diff}$ thus maintaining PaO_2 with minimal reductions in arterial hemoglobin saturation ($SaO_2\%$) from light to maximal exercise. Airway resistance and the change in volume per unit change in pressure (compliance) of the lung are maintained close to resting levels. Breathing requires $\leq 10\%$ of both maximal oxygen consumption and maximal cardiac output and intrathoracic pressure changes approximate only 40–50% of their maximal dynamic capacity in most untrained individuals.

While it might be anticipated that these more than adequate changes with exercise would persist or even improve in a highly trained individual, such is not the case. There are three major situations in which the respiratory system may be inadequate for the task at hand and thus limit high-intensity exercise. These are

1. Exercise-induced arterial hypoxemia
2. Respiratory muscle fatigue
3. Excessive fluctuations in intrathoracic pressure (Amann, 2012)

Exercise-Induced Arterial Hypoxemia

Refer again to **Figure 10.5E, F and H**). Note the dotted lines, which show steep decreases in PaO_2 and $\text{SaO}_2\%$ and a steep rise in $(A-a)\text{PO}_2\text{diff}$. A decrease of at least 10 mmHg PaO_2 or 4% $\text{SaO}_2\%$ (if persistent) is called **exercise-induced arterial hypoxemia (EIAH)**. This is a condition in which the amount of oxygen carried in arterial blood is decreased. The decline in PaO_2 (**Figure 10.5E and G**) with EIAH can be considerable, ranging from 18 to 37 mmHg below resting values ($96-18 = 78$ mmHg; $96-37 = 59$ mmHg). At the same time, the $\text{SaO}_2\%$ (**Figure 10.5H**) is reduced. Mild EIAH is indicated by a $\text{SaO}_2\%$ of 93–95%, moderate as 88–93%, and severe as less than 88% ([Amann, 2012](#); [Richards et al., 2004](#)).

Exercise-Induced Arterial Hypoxemia (EIAH) A condition in which the amount of oxygen carried in arterial blood is severely reduced by $\geq 4\%$ consistently.

Surprisingly, 40–50% of highly trained, healthy, elite male cyclists and runners with $\dot{\text{V}}\text{O}_2 \text{ max}$ in excess of $4.5 \text{ L}\cdot\text{min}^{-1}$ or $55 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ exhibit this response at work rates from 60% to 90% of maximum. Even the highly successful Kenyan runners have been shown to experience EIAH ([Foster et al., 2014](#)). Although most of the original data were collected from elite adult male athletes, there is now evidence of EIAH in females as well as younger and older adult athletes of both sexes ([Prefaut et al., 2000](#)). Recently, EIAH has also been reported in untrained, low-fit females ([Dominelli et al., 2013](#); [Richards et al., 2004](#)) and nonelite sportsmen and sportswomen following high-intensity interval training ([Mucci et al., 2004](#)). However, no published reports have shown EIAH in untrained males. Thus, females may be more prone to developing EIAH than males. The pattern of respiratory changes differs greatly among those who experience EIAH at submaximal exercise intensities ([Dominelli et al., 2013](#)).

During EIAH, the individual's ability to process oxygen and, therefore, to perform high-intensity activity is lower than it would be without EIAH, although both may be higher than in untrained or moderately trained individuals. It has been

estimated that $\dot{V}O_2 \text{ max}$ is reduced by approximately 1.5–2% for each 1% reduction in SaO_2 once the reduction is more than 3% below resting values (Dempsey and Wagner, 1999; Romer et al., 2012). One study tested participants with and without EIAH

who had similar $\dot{V}O_2 \text{ max}$ and power outputs (Legrand et al., 2005). The EIAH athletes, however, had higher muscle deoxygenation. That is, their working muscles had adapted and were able to compensate, at least to some extent, for the reduced oxygen delivery by extracting more oxygen from what was available. Thus, external respiration may be a limitation for exercise in some individuals with full or partial compensation from internal respiration. Athletes who exhibit EIAH at sea level suffer more severe gas exchange impairments during short-term exposure to higher altitudes than athletes who do not exhibit EIAH at sea level (Amann, 2012; Powers et al., 1993).

What Causes EIAH?

With EIAH, the (A-a)PO₂diff curve increases more than normal as shown by the dotted line increase to 30–50 mmHg in **Figure 10.5F**. This excessively widened (A-a)PO₂diff is the most consistent marker and cause of EIAH. It is a definite indication of lack of efficiency in respiration (Romer et al., 2012; Rowell et al., 1964; Shapiro et al., 1964). The widening of (A-a)PO₂diff during exercise has been attributed to a combination of (1)

nonuniformity in alveolar ventilation (\dot{V}_A) and cardiac output (amount of blood ejected per minute) (Q) distribution; (2) extrapulmonary and intrapulmonary shunts where red blood cells do not come in contact with oxygen or deoxygenated blood is dumped into the left side of the heart; (3) mechanical constraints on airflow or insensitivity to the ventilatory stimuli associated with exercise; and (4) an alveolar-capillary diffusion disequilibrium as determined by the alveolar-capillary surface area, the diffusion gradient from alveolar to capillary PO₂, and red blood cells moving too quickly for equilibrium in the pulmonary capillary.

Why EIAH occurs in some individuals and not others has not been fully determined. Preventive techniques are largely

unknown. Like so many areas of exercise physiology, this is an active area of research.

Respiratory Muscle Fatigue

Both respiratory muscle fatigue and excessive fluctuations in intrathoracic pressures can limit cardiac output. These are most evident in sustained high-intensity endurance exercise performance ($>85\% \dot{V}O_2 \text{ max}$).

High-intensity endurance exercise requires large increases in both inspiratory and expiratory muscle work, often leading to respiratory muscle fatigue. Fatigued respiratory muscles may affect exercise performance via the *metaboreflex*. That is, metabolites accumulate in the respiratory muscles and activate afferent neurons that transport signals to the brain, which in turn increases sympathetic vasoconstrictor activity in the vasculature of the exercising limbs. This vasoconstriction leads to decreased blood flow and oxygen delivery, peripheral muscle fatigue, increased perception of effort, and ultimately to a decrease in exercise performance.

Excessive Fluctuations in Intrathoracic Pressures

The ventilatory response during high-intensity exercise is associated with substantial changes in both negative and positive intrathoracic pressures. The normally occurring negative inspiratory intrathoracic pressures associated with high-intensity exercise increase stroke volume (amount of blood ejected from the heart with each beat). However, when negative pressures are increased beyond these normal levels, no additional increase occurs in stroke volume. Conversely, even small increases in positive intrathoracic pressures with expiration result in a reduced stroke volume. Increases in expiratory positive pressures occur during the transition from moderate to intense exercise in well-trained individuals and/or individuals who develop mechanical expiratory flow limitations during exercise. Since

stroke volume times heart rate equals cardiac output, it is possible that the positive pressure changes will outweigh the negative changes and decrease cardiac output. Any limitation in cardiac output contributes to a decrease in blood flow to the exercising limbs and brings about less oxygen delivery. During sustained exercise greater than 85% $\dot{V}O_2 \text{ max}$ in a highly trained endurance athlete, the respiratory muscles require up to 15–16% of $\dot{V}O_2 \text{ max}$ and cardiac output versus $\leq 10\%$ in the untrained individual (Amann, 2012; Dempsey et al., 2008).

The Influence of Sex and Age on Respiration at Rest and during Exercise

Male-Female Respiratory Differences

Lung Volumes and Capacities

Values for total lung capacity (TLC) and each of its subdivisions are, on the average, lower for females than males across the entire age span, with the possible exception of around 12–13 years of age, when most girls have had their pubertal growth spurt but boys have not. These differences carry over into the dynamic measurements of maximal voluntary ventilation (MVV) and forced expiratory volume in one second (FEV₁). Part of these differences can be attributed to the smaller size of females. Males, for example, have larger-diameter airways, more alveoli, and larger diffusion surfaces than females. However, even when values are expressed relative to height, weight, or surface area, some differences in lung capacities remain (Åstrand, 1952; Comroe, 1965; Ferris et al., 1965; Harms, 2006; Loe et al., 2014).

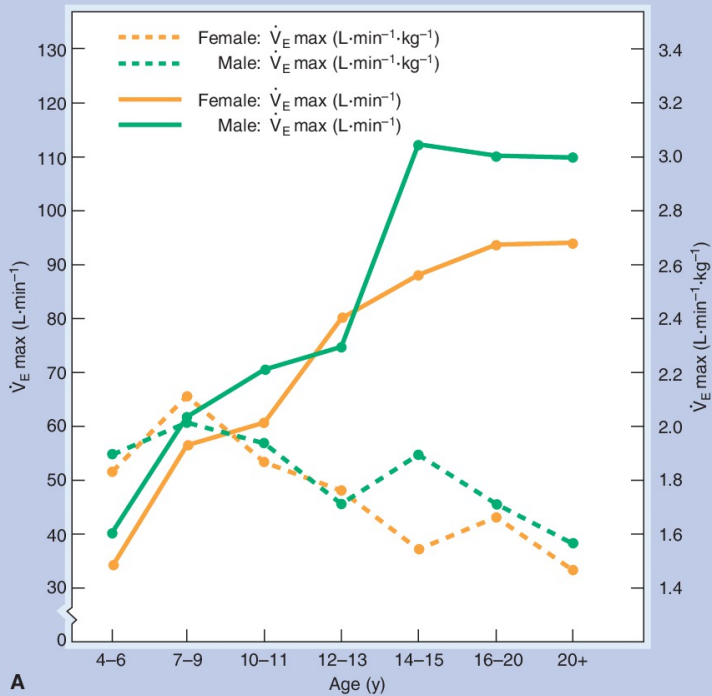
Pulmonary Ventilation

At rest, there is no consistent difference in breathing frequency between males and females (Malina et al., 2004). However, at the same submaximal ventilation, females typically display a higher frequency and a lower V_T than males. This pattern is maintained at maximal exercise (Saris et al., 1985) (**Figure 10.6B**).

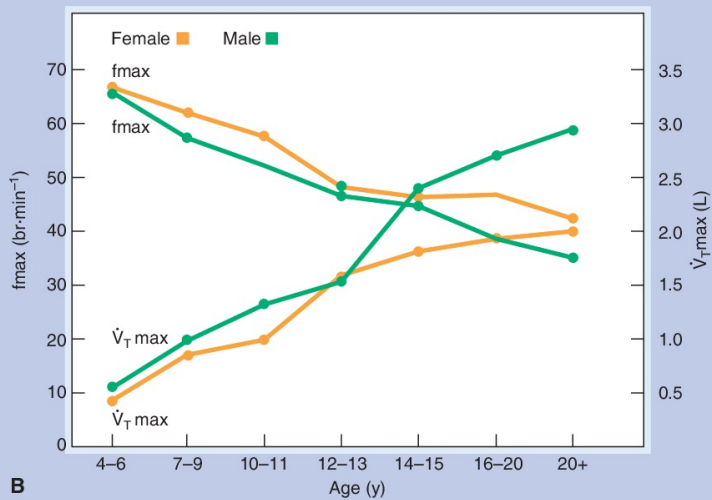
Ventilatory responsiveness during exercise may be influenced in females by levels of circulating estrogen and progesterone

(Harms, 2006). Males also exhibit higher \dot{V}_E at most ages than females at maximal exercise, although these differences are narrowed considerably when expressed relative to body weight (**Figure 10.6A**) (Åstrand, 1952, 1960). Because females have airways that are smaller relative to lung size than males, they are more likely to develop expiratory flow limitations, exhibit an increased elastic load on the inspiratory muscles, and experience more flow-resistive work of breathing than males at increasing

exercise intensities. Thus, at a given \dot{V}_E L·min⁻¹, both the ventilatory work and oxygen cost of breathing are significantly higher for the same-age female as male. Surprisingly, experiments have shown that the inspiratory muscles including the diaphragm of females may fatigue at a slower rate than those of males (Dominelli et al., 2015; Gonzales and Scheuermann, 2006; Guenette et al., 2010; Loe et al., 2014; Romer et al., 2012).



A



B

Figure 10.6 Pulmonary Ventilation Responses to Maximal Treadmill Exercise as Children Age to Adulthood for Males and Females.

A. Maximal minute ventilation, absolute and prorated to body weight. B. Maximal frequency of breathing and tidal volume. **Source:** Based on data from Åstrand (1952).

External and Internal Respiration

Data to compare males and females are unavailable for most external and internal respiratory measures (Harms, 2006). The $a\text{-}v\text{O}_2\text{diff}$ has been measured at rest and during submaximal and maximal exercise, but the results show little consistency (Åstrand et al., 1964; Becklake et al., 1965; Zwiren et al., 1983). The $(A\text{-}a)\text{PO}_2\text{diff}$ is higher and the PaO_2 is lower in females compared to males at any given level of oxygen utilization. An excessive widening of the $(A\text{-}a)\text{PO}_2\text{diff}$ occurs in EIAH, and females exhibit this condition at least as often as males (Dominelli et al., 2013;

Harms, 2006). \dot{V}_A is equal in males and females. The PaCO_2 is slightly lower in females than males at any given $\dot{V}\text{O}_2$ (Hopkins and Harms, 2004; Romer et al., 2012).

Children and Adolescents

Lung Volumes and Capacities

In general, the TLC and each of its subdivisions increase in a mostly rectilinear pattern for both boys and girls as they progress from about 6 years of age into the late teens or early twenties. The FEV₁ and MVV follow essentially the same incremental pattern in children (Åstrand, 1952; Åstrand et al., 1963; Bjure, 1963; Koyal et al., 1976; Malina et al., 2004). From birth to approximately age 10, these changes depend largely on the growth and development of the respiratory system. After that, cell proliferation ceases and hypertrophy of existing structures occurs until maturity. Thus, these changes result primarily but not exclusively from structural enlargement and are strongly related to body height (Bjure, 1963; Johnson and Dempsey, 1991). When the subdivisions of TLC are expressed as a percentage of the V_T , the proportions remain the same from about age 8 to age 20. The anatomical dead space increases in proportion to maturity (Ashley et al., 1975; Robinson, 1938) and,

as in adults, is approximately $1 \text{ mL} \cdot \text{lb}^{-1}$ of body weight (Rowland, 2005).

Pulmonary Ventilation

The control of ventilation is similar in children and adults, except that there is a lower set point of PCO_2 in children than adults (Rowland, 2005), that is, the respiratory centers of children are more sensitive to CO_2 (Romer et al., 2012). However, there are

some minor differences in \dot{V}_E and its components across the age span (Zauner et al., 1989).

REST The \dot{V}_E at rest is surprisingly consistent regardless of age (Robinson, 1938). Figure 10.7 shows that \dot{V}_E in males varies less than $2 \text{ L} \cdot \text{min}^{-1}$ from ages 6 to 76. Comparable data are not

available for females. When \dot{V}_E is expressed relative to body weight, younger boys have a higher \dot{V}_E than older adolescents or adults, but from adolescence to old age, there is little change in \dot{V}_E .

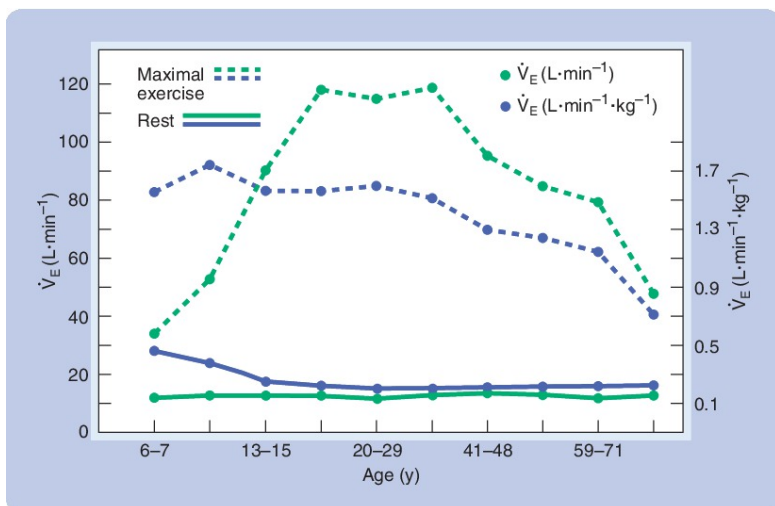


Figure 10.7 Minute Ventilation at Rest and during

Maximal Exercise for Males Aged 6–76.

Source: Based on data from [Robinson \(1938\)](#).

The remarkably consistent \dot{V}_E is achieved differently by children, however, than by older adolescents and adults. **Figure 10.8A** shows that breathing frequency is higher and V_T lower in youngsters than in older adolescents and adults. From approximately the midteen years until old age, frequency stabilizes at $10 \text{ br}\cdot\text{min}^{-1}$, and V_T stabilizes at about 500 mL in males ([Malina et al., 2004](#); [Robinson, 1938](#)). Younger children use a higher portion of their VC as V_T than older adolescents and young adults (**Figure 10.8B**).

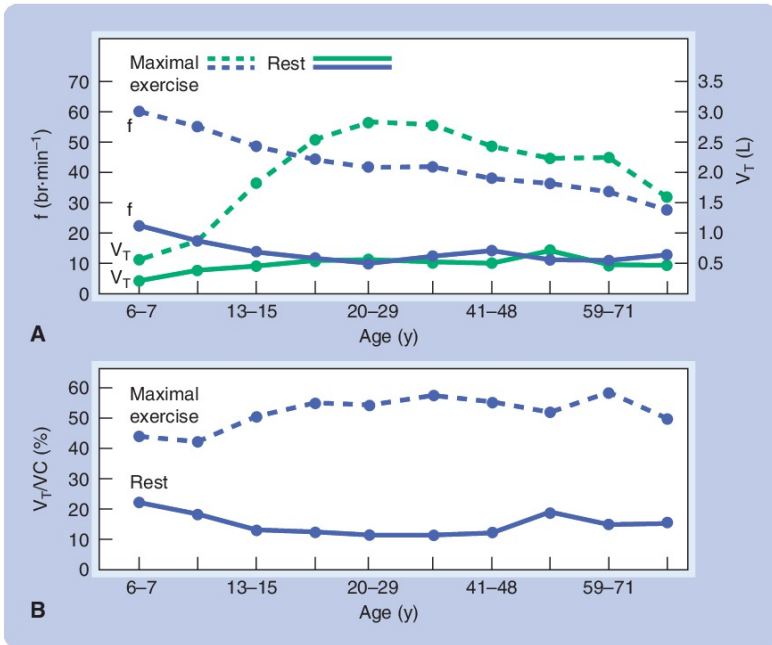


Figure 10.8 Pulmonary Ventilation at Rest and at Maximal Exercise in Males Aged 6–76 y.

A. Frequency of breathing (f) and tidal volume (V_T). **B.** The percentage of vital capacity used as tidal volume (V_T/VC).

Source: Based on data from [Robinson \(1938\)](#).

SUBMAXIMAL EXERCISE Children's and adolescents' respiratory

responses to exercise are similar to those of adults. \dot{V}_E rises in response to greater oxygen needs at all ages, but it does so faster at the onset of exercise in younger individuals than adults

(Rowland, 2005). The higher \dot{V}_E in relation to body weight at

rest is maintained, as is the variation in how \dot{V}_E is obtained. That is, children exhibit a higher frequency and lower V_T at any given submaximal load than adults. They also respond with a

higher \dot{V}_E in relation to body weight at an equal work rate. The

ratio of liters of air processed per one liter of oxygen used ($\dot{V}_E / \dot{V}O_2$) is called the **ventilatory equivalent**. Children and adolescents have a higher ventilatory equivalent at all exercise intensities than adults, indicating that they are hyperventilating. Girls hyperventilate more than boys (Rowland, 2005). These differences are considered negative, indicating a wasteful ventilation, and gradually disappear by late adolescence (Bar-Or, 1983; Do Prado et al., 2010; Robinson, 1938; Rowland, 2005; Rowland and Green, 1988; Rowland et al., 1987). **Figure 10.9A**

and **B** shows differences in frequency (f), V_T , and \dot{V}_E for 11-year-old girls and boys in comparison to 29-year-old adults (Rowland and Green, 1988; Rowland et al., 1987). The speeds are different for males and females, so direct comparison between the sexes cannot be made, but within the sexes, the age differences are similar. In addition to the differences shown in **Figure 10.9**, younger children use a marginally lower proportion of their VC during submaximal exercise than adults.

Ventilatory Equivalent The ratio of liters of air processed per liter of oxygen used ($\dot{V}_E / \dot{V}O_2$).

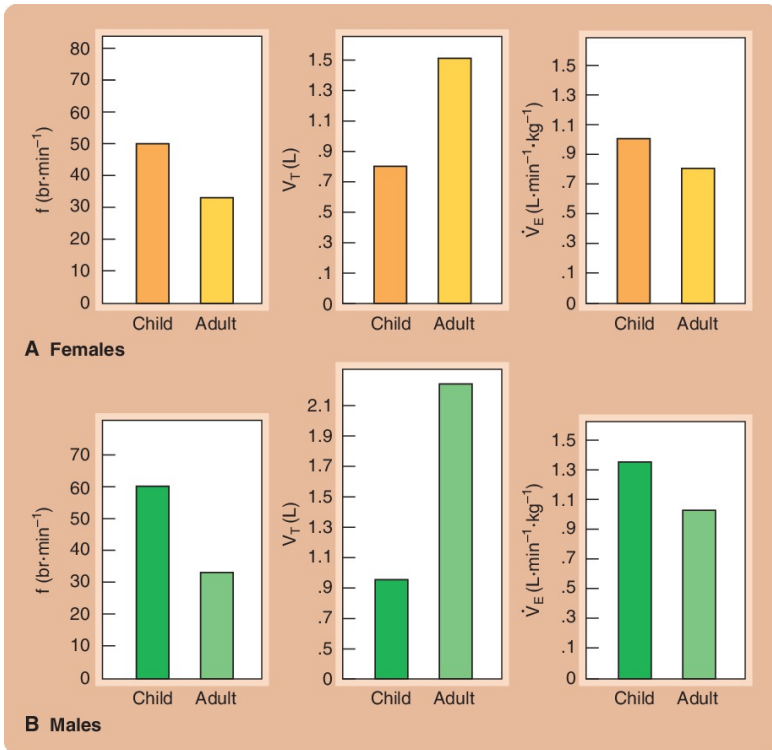


Figure 10.9 Pulmonary Ventilation Responses to Submaximal Exercise in Male and Female Children and Adults.

A. Females: Mean age of child = 11.3 years, adult = 28.7 years; treadmill speed = 7.3 kph (122 m·min⁻¹). **B. Males:** Mean age of child = 11.6 years, adult = 29.2 years; treadmill speed = 9.6 kph (160 m·min⁻¹). **Sources:** Based on data from Rowland and Green (1988); Rowland et al. (1987).

During prolonged submaximal exercise, children and adolescents have the same ventilatory drift as adults, probably in response to a rising core temperature. Nothing in the respiratory response to prolonged exercise would indicate that children are not suited for such activity (Malina et al., 2004; Rowland, 2005).

MAXIMAL EXERCISE In general, children's responses to maximal

exercise parallel the differences seen at rest and during submaximal work rates. The older the child, the higher the \dot{V}_E that can be achieved in absolute terms. The \dot{V}_E in relation to body weight gradually declines from about age 7 until adulthood is achieved at about 20 years of age, although in 4- to 6-year-olds, this value is comparable to that of young adults (**Figure 10.6A**) (Åstrand, 1952; Åstrand et al., 1963; Fahey et al., 1979; Krahenbuhl et al., 1985; Robinson, 1938; Rowland, 1990; Rowland and Green, 1988). Breathing frequency decreases consistently from the youngest children tested to approximately age 20, while V_T shows a steady rise over the same time span (**Figure 10.6B**). The percentage of VC used as V_T rises slightly from childhood to young adulthood. The work of breathing is higher in children than in adults and children probably reach respiratory constraints during maximal exercise at lower relative $\dot{V}O_2$ than adults do (Romer et al., 2012). The $\dot{V}_E / \dot{V}O_2$ gradually declines at maximal work in both boys and girls (Rowland, 2005). Children also show ventilatory breakpoints during incremental exercise to maximum. Some inconclusive evidence suggests that $VT1$ and $VT2$ (expressed as a percentage of $\dot{V}O_{2\max}$) are higher in younger children than adults and gradually decline as children mature into adults (Do Prado et al., 2010; Mahon and Cheatham, 2002). The physiological mechanisms responsible for the $VT1$ and $VT2$ in children are as unclear as they are for adults (Bar-Or, 1983; Rowland and Green, 1988).

External and Internal Respiration

Little is known about the changes in gas exchange and transport that occur during normal growth and maturation. The higher frequency and lower V_T of children in relation to adolescents and adults, at rest and during exercise, seem to be offset by their smaller anatomical dead space. Consequently, \dot{V}_A is more than adequate at all values of \dot{V}_E (Bar-Or, 1983; Malina et al., 2004; Zauner et al., 1989).

Pulmonary diffusion during exercise does not appear to differ by age. Likewise, no meaningful aging trends are apparent for PAO_2 , PaO_2 , or $(\text{A-a})\text{PO}_2\text{diff}$ at rest or during exercise. However, PaO_2 decreases slightly and the $(\text{A-a})\text{PO}_2\text{diff}$ increases slightly as children mature to adulthood (Bar-Or, 1983; Eriksson, 1972; Robinson, 1938). As a consequence, $\text{SaO}_2\%$ is also relatively constant across the age span (Robinson, 1938). EIAH is evident in some trained youth as in some adults, and the causes are undoubtedly the same in this age group (Nourry et al., 2004; Prefaut et al., 2000).

The $\text{a-vO}_2\text{diff}$ is also very similar at both rest and maximal exercise levels from childhood to maturity. If anything, children may be able to extract about 5% more oxygen during maximal exercise than adults (Eriksson, 1973).

Older Adults

Normal, healthy aging causes progressive deterioration in both the structure and function of the respiratory system. The changes begin as young as 20 years and accelerate in the 50s and 60s.

Lung Volumes and Capacities

The effect of aging on TLC is controversial. Inconsistent evidence shows that TLC may decrease or stay the same in individuals over the age of 50 years (Berglund et al., 1963; Jain and Gupta, 1974a, b; Johnson and Dempsey, 1991; Kenney, 1982; Stanescu et al., 1974; Storstein and Voll, 1974). However, research has firmly established that VC and inspiratory capacity (IC) decrease with age and that residual volume (RV) and functional residual capacity (FRC) increase, thus changing the percentage of total volume that each occupies (Åstrand, 1952, 1960; Ericsson and Irnell, 1974; Slonim and Hamilton, 1976; Stanescu et al., 1974; Turner et al., 1968). For example, the ratio of RV/TLC doubles from about 15–30% in the elderly (Comroe, 1965; Johnson and Dempsey, 1991).

FOCUS ON APPLICATION

Side Stitches

The “side stitch,” known medically as *exercise-related transient abdominal pain (ETAP)*, is a common, poorly understood, generally self-limiting acute abdominal pain that is difficult to treat ([Waterman and Kapur, 2012](#)). Mild ETAP is generally described as cramping, aching, or pulling, whereas more severe pain is described as sharp or stabbing. Most stitches occur in the right or left lumbar regions of the abdomen, but occasionally the pain radiates to the shoulder. Although most frequently associated with runners, a high incidence of ETAP has also been reported in other athletes whose sport involves repetitive torso movement and/or jolting such as swimmers and endurance equestrians. Cyclists seem to have a low incidence of ETAP ([Morton and Callister, 2000, 2015](#)).

Stitches seem to occur more frequently in younger individuals and in individuals with kyphotic or lordotic spinal misalignment ([Morton and Callister, 2010](#)). Some studies report more incidences of stitch in females than in males, but others do not. ETAP is experienced more commonly when starting a new or increased exercise routine, but training/fitness level is not totally protective ([Morton and Callister, 2002, 2015](#); [Waterman and Kapur, 2012](#)). The consumption of large amounts of food before or fruit juice that is high in carbohydrate and osmolality both shortly before and during exercise tends to increase the incidence of stitches in individuals susceptible to ETAP ([Morton and Callister, 2000, 2015](#); [Morton et al., 2004](#)).

Several theories have been postulated to explain the causes of ETAP, but there is no consensus to date ([Waterman and Kapur, 2012](#)). Theories include (1) a lack of blood flow to the diaphragm during exercise leading to pain from ischemia, (2) subdiaphragmatic visceral ligament stress, (3) skeletal muscle cramps, and (4) irritation of the parietal peritoneum. Given that the muscles involved in respiration may deprive the rest of the body of blood flow under circumstances of deficit cardiac output and that ETAP is not associated with reduced inspiratory performance, it is

unlikely that the diaphragm is directly responsible ([Morton and Callister, 2000, 2006](#)). Electromyogram (EMG) activity is not elevated during ETAP; therefore, it is also highly unlikely that muscle cramps are responsible despite the subjective feeling of cramping ([Morton and Callister, 2008](#); [Romer et al., 2012](#)). The visceral ligament stress theory is consistent with the influence of increased mass in the gut but does not explain the shoulder-referred pain. The primary function of the parietal peritoneum is to lubricate mobile viscera. Irritation could arise as a result of friction and that in turn could lead to pain. However promising this theory, it has yet to be fully substantiated ([Morton and Callister, 2000, 2015](#); [Plunkett and Hopkins, 1999](#); [Waterman and Kapur, 2012](#)).

Several practical applications of the collective research have been suggested. To avoid a side stitch, an exerciser can try these recommendations:

1. Do not exercise immediately after eating a big meal or ingesting a large amount of fluid (≥ 1 L). Wait 2–3 hours.
2. When drinking during exercise, take small amounts (200–400 mL) frequently (at 15- to 30-minute intervals) rather than a single large drink at a rest stop or aid station.
3. When running downhill, try to keep your breathing regular and your footfalls light.

To deal with a stitch when it occurs:

1. Push in at the location of the pain with your hand, bend forward, and tighten the abdominal muscles.
2. Breathe more deeply to move more air into the lungs at the beginning of each breath, but do not try to force more air out at the end of each breath.
3. Breathe out through pursed lips. This contracts the abdominals. At the same time, if you are an entrainment breather, try to exhale on the opposite foot/side from normal.
4. If you experience side stitches frequently, try wearing a

light, wide belt around your waist that can be tightened when necessary or better yet improve your core strength.



Sources: Morton and Callister (2000, 2002, 2006, 2008, 2010, 2015); Morton et al. (2004); Plunkett and Hopkins (1999); Romer et al. (2012); Waterman and Kapur (2012).

FEV₁ and MVV decline steadily after approximately age 35 in both males and females (Ashley et al., 1975; Ericsson and Irnell, 1974; Quanjer et al., 2012; Shepard, 1978; Slonim and Hamilton, 1976; Stanescu et al., 1974). These declines result from a combination of structural and mechanical changes in the respiratory system. These changes include (1) decreased elastic recoil of lung tissue; (2) stiffening of the thoracic cage, which decreases chest mobility and creates a greater reliance on the diaphragm; (3) a decrease in intervertebral spaces, which in turn decreases height and changes the shape of the thoracic cavity; and (4) losses in respiratory muscle force and velocity of contraction. Of all these changes, the loss of elastic recoil appears to be the most important (Johnson and Dempsey, 1991; Romer et al., 2012; Turner et al., 1968).

Pulmonary Ventilation

REST Resting \dot{V}_E (Figure 10.7) and its components V_T and frequency (Figure 10.10A) are remarkably consistent across the entire age span. However, at rest, the percentage of VC used as V_T does show a very slight U-shaped curve (Figure 10.8B). Both young children and older adults use slightly more of their VC for V_T at rest than young adults (Robinson, 1938).

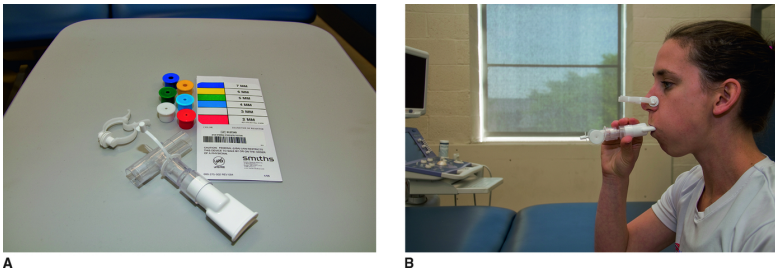


Figure 10.10 Respiratory Muscle Training.

A. This is an example of the specialized equipment used in respiratory muscle training. **B.** An individual is using the specialized equipment for respiratory muscle training.

SUBMAXIMAL EXERCISE \dot{V}_E rises in older adults in response to increased energy needs. As in young adults, this increase is accomplished mainly by an increase in V_T at lighter work rates and then by an increased frequency, if needed. Like children,

older adults seem to have an exaggerated response in \dot{V}_E compared with younger adults. That is, the absolute \dot{V}_E is higher at any given work rate in older than in younger adults. Because the VC decreases with age and V_T remains fairly stable, V_T represents a higher percentage of the older adult's VC (Åstrand, 1952, 1960; Davies, 1972; DeVries and Adams, 1972; Robinson, 1938; Shepard, 1978).

MAXIMAL EXERCISE With aging, both the ability to exercise maximally and the ability to process air decline. The decrement in pulmonary function contributes to the decline in work capacity but probably simply parallels the changes in circulation,

metabolism, and muscle function that are occurring.

The highest \dot{V}_E values are typically seen in young adults, and these may decline by almost half by the seventh decade of life (**Figure 10.9**). This decline is evident both in absolute values (\dot{V}_E in liters per minute) and in values adjusted for body weight (\dot{V}_E in liters per kilogram per minute). Most of this decline is brought about by a reduction in V_T , although maximal frequency does decrease slightly.

The percentage of VC used during maximal work is relatively stable with age from young adulthood on. However, the dead space to tidal volume ratio is consistently 15–20% higher in older adults than in younger adults (Robinson, 1938; Romer et al., 2012) because dead space increases both at rest and during exercise. Older adults begin to exhibit meaningful expiratory flow limitations at ventilatory rates that may be only half of where a younger individual would. This limitation worsens as the exercise intensity increases. At any given exercise ventilation, the work of breathing is higher in the older individual than the younger adult. This is often reflected in higher perceptions of dyspnea as one ages (Romer et al., 2012). The ventilatory breakpoints occur at lower absolute and relative workloads in older adults than in younger adults (Shepard, 1978).

External Respiration

REST The loss of elastic recoil in the lungs not only affects static and dynamic lung volumes but also influences the distribution of air in the lungs. Thus, ventilation may not be preferentially directed to the base of the lung in the upright posture at rest, although most blood flow is still directed there. As a result, there may be an imbalance between alveolar ventilation and pulmonary perfusion (Johnson and Dempsey, 1991).

In addition, structural changes in aging lung tissue decrease the alveolar-capillary surface area, which in turn means a decrease in diffusion capacity (Donevan et al., 1959; Johnson and Dempsey, 1991). Furthermore, pulmonary capillary blood volume decreases because of a stiffening of both pulmonary arteries and

capillaries. The cumulative effects of these changes are a decrease in PaO_2 , but not PAO_2 , and a widening of the $(\text{A-a})\text{PO}_2\text{diff}$ —although these changes are neither inevitable nor large. The saturation of hemoglobin with oxygen in arterial blood declines about 2–3% from age 10 to 70 (Robinson, 1938; Shepard, 1978).

EXERCISE During exercise, the increased ventilation that is required results in a more homogeneous distribution of ventilation in the lungs. Although the decreases noted at rest in diffusion surface and pulmonary capillary blood volume remain, the matching of ventilation and perfusion improves during exercise as more of the lung is used. The available reserve is sufficient to meet the demands for oxygen transport even to maximal exercise levels.

Nevertheless, \dot{V}_A is a smaller portion of minute ventilation in older adults than in younger adults. This change indicates a slightly decreased efficiency of respiration, but general arterial hypoxemia is prevented, and carbon dioxide elimination is adequate. That is, the PaO_2 and PaCO_2 are maintained within narrow limits and are similar to those for younger adults. The $(\text{A-a})\text{PO}_2\text{diff}$ is more variable in older than in younger adults but, on average, is only slightly wider than the usual mean values for younger individuals (Johnson and Dempsey, 1991; Robinson, 1938; Shepard, 1978). Highly fit older adults can, however, exhibit EIAH, as noted previously (Prefaut et al., 2000; Romer et al., 2012).

Internal Respiration

At rest and at any given submaximal level of exercise, the $\text{a-vO}_2\text{diff}$ is greater in older adults than in younger adults. Thus, $\text{SvO}_2\%$ is lower. Conversely, at maximal exercise, the $\text{a-vO}_2\text{diff}$ is lower in older individuals than in younger ones. Maximal values average $14\text{--}15\text{ mL}\cdot\text{dL}^{-1}$ in older adults but average $15\text{--}20\text{ mL}\cdot\text{dL}^{-1}$ in younger adults. Some of these changes in the $\text{a-vO}_2\text{diff}$ can be attributed to a shift in the oxygen dissociation curve to the left, which makes the release of oxygen to the tissues more difficult (Kenney, 1982; Shepard, 1978). Refer to **Figure 10.2** to see this effect.

Respiratory Muscle Training Principles and Adaptations

Extensive training principles or guidelines are not included here for the respiratory system because respiratory training for healthy individuals is still rare and requires specialized equipment. Yet, the respiratory muscles are muscles and as such are subject to fatigue, especially the diaphragm, and as mentioned previously, this may cause vasoconstriction in the working musculature. During both short- and long-duration incremental or constant-load exercise $\leq 80\% \dot{V}O_2 \text{ max}$, the diaphragm does not fatigue. However, at more than 80–85% $\dot{V}O_2 \text{ max}$ intensity continued to exhaustion, the diaphragm does fatigue. The consequence of this fatigue is a decrease in exercise tolerance possibly due to the aforementioned metaboreflex (Sheel, 2002). One study (Enright et al., 2006) documented increased diaphragm thickness with increased performance and power output after high-intensity inspiratory muscle training. There is a growing interest in specific training for the respiratory muscles.

As with any type of muscular training, either strength or endurance may be emphasized in respiratory muscle training (RMT). There are two major types of respiratory muscle strength training (RMST): (1) inspiratory flow-resistive loading (IFRL) and (2) inspiratory pressure-threshold loading (IPTL). Respiratory muscle endurance training (RMET) consists primarily of voluntary isocapnic hyperpnea training (VIHT). Only occasionally has expiratory strength muscle training been undertaken alone. Most strength training involves only the inspiratory muscles, whereas endurance training typically stresses both inspiratory and expiratory muscles.

Two techniques for IFRL training are used. The first technique utilizes equipment that requires the individual to inspire using a variable diameter opening (**Figure 10.10A and B**). At any given flow, the smaller the opening, the greater the resistance. The second technique first tests individuals by having them forcefully exhale to residual volume followed immediately by maximally inhaling against resistance to TLC. An image representing typically 80% of this pressure-time profile effort is presented on

the computer screen. The training regime requires the participant to match (or exceed) this onscreen template within a progressively increasing work-rest ratio. For example, the maneuver is initially performed six times with a 60-second recovery. Only 45 seconds of recovery is permitted following the second set of six efforts. This pattern continues for six sets during which recovery is gradually reduced to 5 seconds.

IPTL involves inspiration against a resistance at a set percentage of peak inspiratory mouth pressure using a specific muscle trainer. This device requires continuous application of inspiratory pressure during inspiration for the valve to remain open while allowing for unrestricted expiration. Typically 30 inspiratory efforts are performed twice a day.

VIHT is performed with a device that uses partial rebreathing and ensures normal CO₂ values. Tidal volume is controlled by feedback and breathing frequency is paced. Training sessions consist of low-force, high-velocity contractions and are typically conducted for 30 min·d⁻¹, 3–5 d·wk⁻¹ at 60–90% of MVV.

In general, regardless of the specific type of inspiratory muscle training employed (either RMST or RMET), functional measures of inspiratory muscle strength, endurance, maximal rate of shortening, maximal sustainable ventilatory capacity, and power have been increased. The importance of these changes is that they likely prevent or delay exercise-induced diaphragmatic fatigue. Delaying or diminishing respiratory muscle fatigue may attenuate the reflex vasoconstrictor activity in the limbs and help preserve limb blood flow. Whole body training does not provide resistance to inspiratory muscle fatigue. Structurally, significant increases in the proportion of type I (slow-twitch, oxidative) fibers and the size of type II (fast-twitch, oxidative glycolytic) fibers have been observed with RMT. Often, but not always, the perception of dyspnea is decreased. Respiratory values for minute ventilation, alveolar ventilation, breathing frequency, and tidal volume as well as oxygen consumption and lactate concentration are typically lowered and actual performance increased during constant submaximal performances or time trials even at

intensities less than 80–85% $\dot{V}O_{2\text{ max}}$, whereas no improvement is seen at maximal exercise or in incremental tests to maximum. The magnitude of performance improvements when

compared to control groups ranges from approximately 3–36%. Less fit individuals benefit more from RMT than highly trained athletes and improvements are greater in long-duration exercise. The inability of RMT to bring about significant changes during incremental tests and in $\dot{V}O_2 \text{ max}$ is not really surprising as central circulatory adaptations require sustained and direct cardiovascular stimulation and this does not occur during RMT.

In addition, $\dot{V}O_2 \text{ max}$ tests only require high respiratory volumes for a brief period at the end of the test. Conversely, endurance time trial performances require high ventilatory volumes for a long duration placing a greater demand directly on respiratory muscles. These positive changes have been seen in rowers, cyclists, soccer players, swimmers, and runners as well as nonathletes such as the elderly (Forbes et al., 2011; Illi et al., 2012; Inbar et al., 2000; Kilding et al., 2010; Leddy et al., 2007; Nicks et al., 2009; Ray et al., 2010; Romer et al., 2002; Sales et al., 2016; Verges et al., 2007; Volianitis et al., 2001; Watsford and Murphy, 2008).

Controlled-Frequency Breathing Training

In addition to RMT, swimmers occasionally use a technique called controlled-frequency breathing (CFB) as a training technique. As opposed to RMT, which makes the work of breathing harder, CFB restricts the number of breaths from the normal breathing pattern. The original assumption was that if a swimmer inhaled once every third, fifth, or eighth arm stroke, instead of every other arm stroke, then the reduced volume of air taken in would decrease oxygen partial pressure and create hypoxia, a decrease in available oxygen. According to this idea, hypoxia should bring about the same beneficial effects for swimmers as altitude training. Now, it is accepted that this does not occur. However, it is theorized that by restricting air exchange at the lungs, oxygen concentrations in pulmonary circulation and at the muscle tissue level allow the swimmer to train at moderate intensity but to mimic the limited oxygen levels characteristic of high-intensity exercise, thus stimulating adaptations to improve oxygen delivery. The long-term benefits of CFB on either sprint or endurance swim performance have yet to be evaluated.

Controlled-frequency breathing does reduce $\dot{V}O_2$ somewhat despite compensatory increases in VT and oxygen extraction. At the same time, CFB as low as every fourth stroke increases the severity of inspiratory muscle fatigue that occurs during high-intensity front crawl swimming. Most importantly, it produces not hypoxia but hypercapnia, an increase in the partial pressure of carbon dioxide. The hypercapnia in turn has been shown to alter the heart rate response to exercise by lowering it. This reduction in exercise HR means that if workouts are monitored by HR intensity, adjustment needs to be made in target values. In addition, hypercapnia often causes headaches that last for 30 minutes or more after the workout ceases. These headaches are painful and may interfere with training. On the positive side, CFB may increase buoyancy and improve body position, enabling the swimmer to concentrate on the biomechanics of the stroke. Despite this, CFB should be used sparingly and be closely monitored (Dicker et al., 1980; Holmer and Gullstrand, 1980; Jakovljevic and McConnell, 2009; Lavoie and Montpetit, 1986; West et al., 2005; Zempel, 1989).

Whole Body Respiratory Training Principles and Adaptations

Other than the specific responses to inspiratory muscle training indicated above, training adaptations that have been documented in the respiratory system occur as a by-product of whole body training for cardiovascular, neuromuscular, and/or metabolic improvement. Applications of the training principles are presented for these systems in their respective units. The few training adaptations that do occur in the respiratory system are documented in the following section.

Lung Volumes and Capacities

Studies of traditional land training sports (running, cycling, wrestling, and the like) have found no consistent significant changes in TLC, VC, RV, FRC, or IC in adult males or females of

any age, nor have they found any differences favoring athletes over nonathletes (Bachman and Horvath, 1968; Cordain et al., 1990; Dempsey and Fregosi, 1985; Eriksson, 1972; Kaufmann et al., 1974; Niinimaa and Shepard, 1978; Saltin et al., 1968). One exception appears to be yoga. Regular yoga training, which involves specific breathing exercises, has been shown consistently to improve respiratory muscle strength, MVV, FEV₁, VC, and peak expiratory flow rates (Abel et al., 2013).

Evidence in children suggests that prepubertal exercise training may accelerate lung growth as an adaptive response. Moreover, prepubescent children may increase lung volumes and expiratory flow rates with long-term endurance training (Romer et al., 2012).

Studies of water-based activities (swimming and scuba diving) have shown that swimmers have higher volumes and capacities than both land-based athletes and nonathletes (Cordain et al., 1990; Leith and Bradley, 1976). Whether the high values in swimmers result from intensive training or genetic endowment has been widely debated. Although some swim training studies have demonstrated increases in TLC and VC in both children and young adults, it may be that swimmers bring these high values to the sport rather than the sport stimulating above normal growth (Bovard et al., 2018). Similar generalizations can probably be made for the dynamic measures of FEV₁ and MVV (Andrew et al., 1972; Bachman and Horvath, 1968; Clanton et al., 1987; Vaccaro and Clarke, 1978; Walsh and Banister, 1988).

Pulmonary Ventilation

Changes in \dot{V}_E are the primary and most consistent adaptations seen in the respiratory system as a result of endurance training.

Although \dot{V}_E itself does not change at rest, a shift occurs in its components: V_T increases, and frequency decreases. This shift is maintained during submaximal work, but overall V_T is lower during submaximal exercise as a result of the training. At

maximal work, \dot{V}_E is higher after training than before, accompanying the ability to do more work. The major component

that changes is frequency, but VT increases as well (Dempsey et al., 1977; Mahler et al., 1991; Rasmussen et al., 1975; Reid and Thomson, 1985; Whipp, 1977; Wilmore et al., 1970). In addition, the capacity for sustaining high levels of voluntary ventilation is improved, reflecting increased strength and endurance of the respiratory muscles (Krahenbuhl et al., 1985; Robinson and Kjeldgaard, 1992).

These adaptations occur within the first 6–10 weeks of a training program (Reid and Thomson, 1985). They result from both land- and water-based activities across the entire age span (Bar-Or, 1983; Fringer and Stull, 1974; Nourry et al., 2004; Pollock et al., 1969; Seals et al., 1984; Zauner and Benson, 1981; Zauner et al., 1989). The ventilatory thresholds shift to a higher workload and oxygen consumption as a result of training both in children and in adults indicating that a greater intensity of exercise can be maintained during endurance exercise performance across this age span (Haffor et al., 1990; Laursen et al., 2005; Loat and Rhodes, 1993; Mahon and Cheatham, 2002; Paterson et al., 1987; Pogliaghi et al., 2006; Poole and Gaesser, 1985).

External and Internal Respiration

In a healthy individual of any age and either sex, gas exchange varies little as a result of training (Reid and Thomson, 1985; Romer et al., 2012). Diffusion capacity has been reported to be higher in elite swimmers (Comroe, 1965; Magel and Andersen, 1969; Mostyn et al., 1963; Vaccaro et al., 1977) and runners (Kaufmann et al., 1974), but it does not consistently increase at either submaximal or maximal work as a result of training (Saltin et al., 1968). Even in studies where diffusing capacity did increase with training, it was most likely due to circulatory changes (an increase in pulmonary capillary volume) rather than any pulmonary membrane change per se. Higher values in diffusion capacity may be an example of genetic selection for specific athletes (Comroe, 1965; Dempsey, 1986; Dempsey et al., 1977; Niinimaa and Shepard, 1978; Vaccaro and Clarke, 1978; Wagner, 1991).

Arterial values of pH and PCO₂ do not change with training,

but venous pH levels increase (become less acidic) and PCO₂ values decrease (Rasmussen et al., 1975) during the same submaximal exercise. The (A-a)PO₂diff decreases at submaximal workloads as a result of training, indicating greater efficiency (Saltin et al., 1968). Training may also cause the oxygen dissociation curve to shift to the right, facilitating the release of oxygen from the blood into the muscle tissue (Rasmussen et al., 1975).

In children, neither the submaximal nor the maximal a-vO₂diff adapts as a result of training (Bar-Or, 1983; Eriksson, 1973). In young adults, the a-vO₂diff increases at rest (Clausen, 1977; Saltin et al., 1968) and at maximal exercise as a result of training (Blomqvist and Saltin, 1983; Coyle et al., 1984; Saltin et al., 1968). Both increases and decreases in the a-vO₂diff have been found during submaximal exercise as a result of endurance training (Clausen, 1977; Ekelund, 1967; Ekelund and Holmgren, 1967; Saltin et al., 1968). Changes in middle-aged and elderly adults are less likely than in younger adults (Green and Crouse, 1993; Saltin, 1969). Exercise training does not appear to protect against the normal age-related deterioration in lung function (Romer et al., 2012). None of the other partial pressure or saturation variables changes significantly and/or consistently with training.

FOCUS ON RESEARCH

Respiratory Training Changes in Older Adults

This study exemplifies several important concepts discussed in this text. Eighteen healthy sedentary males, aged 65–75 years, were divided into three equal groups. One group performed leg training (LT) on a leg ergometer (LE); the second group performed arm training (AT) on an arm ergometer (AE); the third group served as a control (C) and remained sedentary. Pre (before) and post (after) training, all participants performed two incremental exercise tests to

maximum: one on the cycle ergometer and one on the arm ergometer. Twelve weeks of training occurred between tests. Training intensity was based on heart rate at the first ventilatory threshold (HRVT₁): 7 minutes at 90%; 10 minutes at 100%; 5 minutes at 110%; and 5 minutes at 90%. This is an example of how VT₁ can be used to prescribe exercise. Approximately every 2 weeks, the training load (in watts) was adjusted to maintain the HRVT₁ percentages.

The results are presented in the accompanying graphs. Panel A presents the results in terms of power output (W). Four conclusions can be drawn:

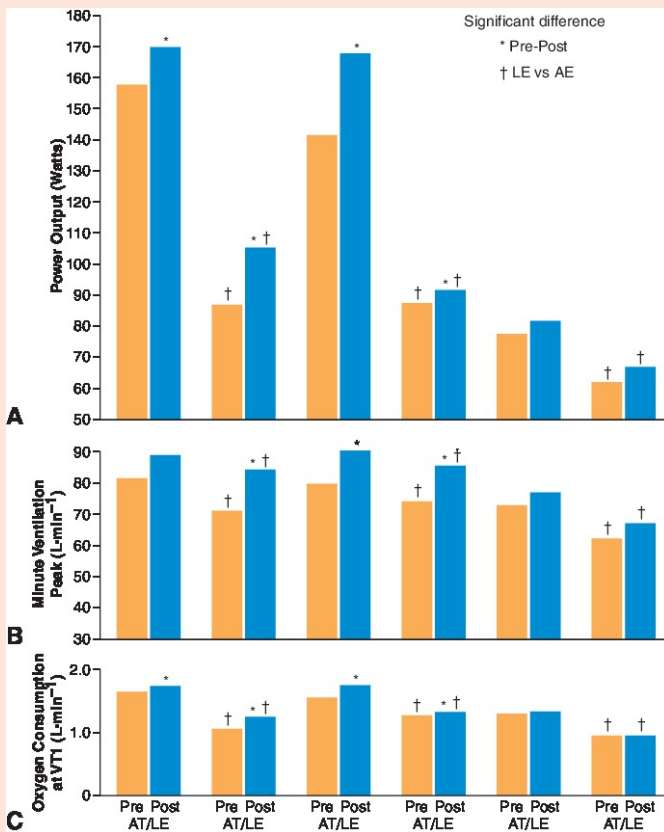
1. The control group (data not shown) did not change either its maximal power output or the wattage at which the VT₁ occurred. Thus, changes in the training groups can be interpreted as being the result of the training.
2. The AT and LT groups significantly improved post training in both modalities of testing and had significantly higher values than the controls. Thus, elderly individuals were trainable.
3. In all three groups, the power outputs were higher at both peak exercise and VT₁ for the LE than the AE. This is a good example of large muscle versus small muscle mass differences.
4. The LT group improved more on the LE than the AE; the AT group improved more on the AE than LE. This exemplifies the principle of specificity. Both groups, however, improved in both modalities, indicating a degree of cross-training.

Panels B and C present the results for \dot{V}_{E} at peak exercise and VT₁ during submaximal exercise. Parallel conclusions can be drawn for these variables:

1. Again, the control group (data not presented) did not show any changes.
2. Posttraining increases in both respiratory variables showed significant increases over pretraining values,

except for the AT group on \dot{V}_E peak during LE testing. Maximal (or peak) minute ventilation increases are the most consistent pulmonary ventilation adaptation to training. Ventilatory thresholds were also expected to increase and did so consistently.

3. In all three groups, both \dot{V}_E and VT1 were significantly higher for LE than for AE, paralleling the differences in power output.
4. Specificity was again evident for both and VT1; the LT group improved more on the LE than the AE, and the AT group improved more on the AE than the LE.



Source: Pogliaghi, S., P. Terziotti, A. Cevese, F. Balestreri, &

F. Schena: Adaptations to endurance training in the healthy elderly: Arm cranking versus leg cycling. *European Journal of Applied Physiology*. 97(6):723–731 (2006).

Table 10.3 summarizes the respiratory training adaptations discussed above.

TABLE 10.3 Respiratory Training Adaptations

| | Rest | Submaximal Exercise | Maximal Exercise |
|-----------------------------|--|---|--|
| Lung volumes and capacities | Show no changes from land-based activities; swimming and diving show increases, especially in total lung capacity and vital capacity | — | — |
| Pulmonary ventilation | | | |
| \dot{V}_E | Shows no change | Decreases | Increases |
| V_T | Increases | Increases | Increases |
| f | Decreases | Decreases | Increases |
| VT1 and VT2 | — | Increase to higher workload/oxygen consumption | — |
| External respiration | | | |
| (A-a)PO ₂ ,diff | Shows no change | Decreases | Shows no change |
| Internal respiration | | | |
| PvCO ₂ | — | Decreases | — |
| Oxygen dissociation curve | — | Curve shifts to the right | Curve shifts to the right |
| a-vO ₂ ,diff | Shows no change in children; increases in young adults | Shows no change in children; inconsistent changes in adults | Shows no change in children; increases in young adults |

Why Are There So Few Respiratory Adaptations to Whole Body Exercise Training?

The most commonly accepted answer to the question of why there are so few respiratory adaptations to whole body exercise training is that the pulmonary system is endowed with a tremendous reserve capacity that is more than sufficient to meet the demands of heavy physical exercise. Thus, the various structural and functional components of the respiratory system (lungs and airways) are not stressed to any significant limits during physical training and so do not need to change. At the same time, the cardiovascular and metabolic capacities of muscle are being stressed and do respond by adapting. The adaptations in these systems may ultimately exceed the capability of the respiratory system, as seen with EIAH and diaphragmatic fatigue, and the pulmonary system can become a limiting factor in elite

athletes. So, the generalization that the respiratory system is “overbuilt” is accurate but only to a point. There is no need for great changes in the respiratory system in the normal healthy, moderately trained individual (Dempsey, 1986; Dempsey et al., 1977; McKenzie, 2012; Sheel, 2002), but highly trained elite athletes could use some adaptation.

Special Considerations

Altitude

The effects of altitude were introduced in [Chapter 9](#). This section details the effects of both acute and chronic exposure to altitude, the exercise response at altitude, and discusses the impact of altitude training on athletic performance. Refer back to [Table 9.1](#) and [Equation 9.4](#) as background for the following discussion.

The Acute Impact of Altitude

The percentage of oxygen in air remains constant at 20.93% to an altitude of 100,000 m (328,083 ft) (Clausen, 1977). However, the barometric pressure (PB) decreases exponentially with increasing altitude. Thus, air has a lower density (fewer molecules per volume) at higher altitudes because the gas has expanded. For instance, at Denver (1,600 m, or 5,280 ft), PB is 630 mmHg, and at Colorado Springs (2,300 m, or 7,590 ft), PB is 586 mmHg ([Figure 10.11](#)). Therefore, the oxygen partial pressure (PO₂) in atmospheric air is reduced at altitude. At Denver, the PO₂ is 132 mmHg (630 mmHg × 0.2093), and at Colorado Springs, it is 123 mmHg (586 mmHg × 0.2093), compared to 159 mmHg at sea level (760 mmHg × 0.2093). Because the partial pressure of the inspired oxygen decreases with altitude, the PAO₂ also decreases to 80 and 74 mmHg, respectively, at Denver and Colorado Springs:

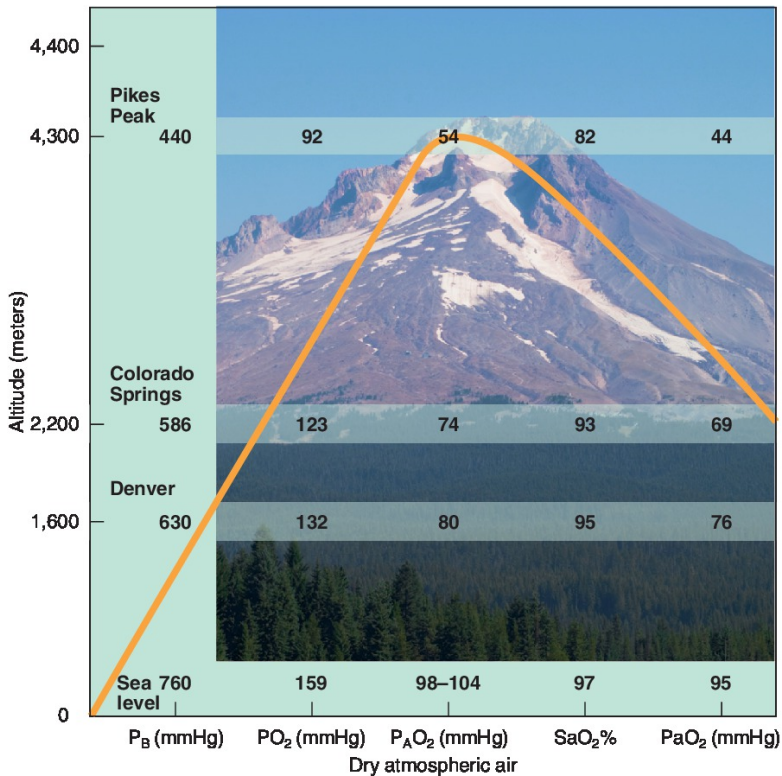


Figure 10.11 The Impact of Altitude on External Respiration.

$$\begin{aligned} [(630 \text{ mmHg}) - (47 \text{ mmHg})] \times (0.137O_2) &= 80 \text{ mmHg;} \\ [(586 \text{ mmHg}) - (47 \text{ mmHg})] \times (0.137O_2) &= 74 \text{ mmHg.} \end{aligned}$$

This decrease in PAO_2 reduces the pressure gradient between the returning venous blood (PvO_2) and the alveoli, resulting in a lower percent saturation ($SaO_2\%$) and arterial partial pressure (PaO_2):

$$\begin{aligned} 80 \text{ mmHg} \times 0.95 &= PaO_2 \text{ of } 76 \text{ mmHg at Denver;} \\ 74 \text{ mmHg} \times 0.93 &= PaO_2 \text{ of } 69 \text{ mmHg at Colorado Springs} \end{aligned}$$

Figure 10.11 summarizes these changes.

Acute Altitude Compensations

The decrease in PaO₂ stimulates an increase in \dot{V}_E via the aortic and carotid body chemoreceptors as compensation to provide more oxygen for alveolar ventilation. The initial increase in \dot{V}_E is hyperventilation (an increase in pulmonary ventilation that exceeds metabolic requirements) and is achieved primarily by an increase in respiratory frequency. Climbers on Mount Everest reportedly averaged 62 br·min⁻¹ and a \dot{V}_E of 207 L·min⁻¹, obviously very high values (Armstrong, 2000). Altitude-induced hyperventilation increases the amount of carbon dioxide exhaled. In turn, the increased exhalation of carbon dioxide decreases alveolar and arterial carbon dioxide partial pressure and increases pH. The decrease in SaO₂% and the other resultant changes at the altitudes of Denver and Colorado Springs are minimal, perhaps 2–5% because of the flatness of the oxygen dissociation curve (Figure 10.2) in that range. However, at the top of Pikes Peak (4,300 m, or 14,100 ft), SaO₂% is 82%, and the effect of altitude is very evident (Hannon, 1978; Haymes and Wells, 1986; Ratzin Jackson and Sharkey, 1988). Table 9.1 and Figure 10.1 show the external respiration values. The SaO₂% is approximately 15% less at Pikes Peak than at sea level. Table 10.4, first column, summarizes these changes.

TABLE 10.4 Pulmonary Ventilation and External Respiration Responses to Altitude

| Physiological Sequence | Acute Compensation* | Chronic Adaptation* |
|---------------------------------------|---|---|
| ↑Altitude ↓ | ↑ (↑f) → ↑CO ₂ blown off ↓ | ↑ \dot{V}_E (↑V _T) (>SL, >acute) ↓ |
| ↓P _B ↓ | ↓P _A CO ₂ , ↓PaCO ₂ , ↑pH + | ↑P _A O ₂ (<SL, >acute) |
| ↓PO ₂ ↓ | ↑2,3-DPG → O ₂ dissociation curve shifts to the right | ↑RBC (Hb) + ↑BV [†] ↓ |
| ↓P _A O ₂ ↓ | ↑ [Hb] + ↓BV + ↑HR ↓ | ↑PaO ₂ (~SL, >acute) + |
| ↓PaO ₂ ↓SaO ₂ % | ↑O ₂ mL·dL ⁻¹ blood | ↑SaO ₂ % (<SL, >acute) |

*At rest and during submaximal aerobic exercise.

†Decrease continues for at least several weeks; by 2 months, blood volume is increasing.

PB, barometric pressure; PO₂, partial pressure of oxygen; PAO₂, partial pressure of oxygen at the alveoli; PaO₂, partial pressure of oxygen in arterial blood; SaO₂%, percent saturation of

arterial blood with oxygen; \dot{V}_E , minute ventilation; f, frequency of breathing; PACO₂, partial pressure of carbon dioxide at the alveoli; PaCO₂, partial pressure of carbon dioxide in arterial blood; pH, negative logarithm of the hydrogen ion concentration; 2,3-DPG, 2,3-diphosphoglycerate, chemical within red blood cells; Hb, hemoglobin; concentration of hemoglobin; BV, blood volume; HR, heart rate; O₂, oxygen; VT, tidal volume; SL, sea level; RBC, red blood cells.

Within the red blood cells, a chemical called 2,3-diphosphoglycerate (2,3-DPG) increases at altitude. The effect of increased 2,3-DPG is the same as increased carbon dioxide partial pressure, hydrogen ion concentration, and body temperature on the oxygen dissociation curve (**Figure 10.2**). That is, it shifts the oxygen dissociation curve to the right, offsetting the initial shift to the left (and decreased release of oxygen) that occurs because of the decreased PCO₂ and increased pH brought about by hyperventilation. The result is a diminished ability of O₂ to combine with Hb in the pulmonary circuit. However, at low and moderate altitudes, the increased dissociation at the tissue level is considered more important and beneficial.

Within 2 days of altitude exposure, hemoglobin concentration increases, which in turn increases the amount of oxygen transported per deciliter of blood. Unfortunately, the hemoglobin concentration increases only because total blood volume initially decreases due to mild dehydration. That is, the same number of red blood cells is distributed in a smaller volume of blood, a process called *hemoconcentration*. The blood volume loss reflects a total body water loss through the kidneys and through increased respiratory evaporation because of the compensatory hyperventilation. Such hemoconcentration increases the viscosity of the blood, resulting in greater resistance to blood flow. In an attempt to maintain blood flow, heart rate increases ([Haymes and Wells, 1986](#); [Ratzin Jackson and Sharkey, 1988](#)). **Table 10.4**, second column, summarizes these acute responses.

These altitude-induced acute responses are compounded during exercise. The normal exercise increase in diffusion capacity does not exceed that at sea level because PaO_2 is decreased and PvO_2 remains the same. As a result, the normal exercise increase in $\text{a-vO}_{2\text{diff}}$ is not as great at altitude as it is at sea level.

Cardiovascular changes occur as well. Heart rate and cardiac output increase in an attempt to compensate for the reduced oxygen content of the blood. Stroke volume is only marginally reduced initially but progressively declines for 1–2 weeks before stabilizing (Mazzeo, 2008).

Although the individual probably does not perceive any of the changes described above, except possibly hyperventilation, alterations can also occur in sensory acuity (vision and hearing), motor skills, memory, and mood (increased irritability). Dehydration and weight loss (in part due to the dehydrations and in part to an elevated resting metabolic rate) are common, as is sleep disturbance. In addition, some unpleasant sensations including a loss of appetite, dizziness, fatigue, nausea, vomiting, and weakness may mark the onset of altitude sickness (Hall and Hall, 2021; Haymes and Wells, 1986).

Exercise Responses at Altitude

The acute effects of altitude are experienced most directly in terms of the intensity at which an aerobic endurance activity can be maintained. The higher the altitude and exercise intensity, the greater the impact and the sooner fatigue is felt. Although oxygen diffusion capacity is not a limiting factor, oxygen delivery cannot keep pace with muscle demand. Maximal aerobic power ($\dot{\text{V}}\text{O}_{2\text{max}}$) declines (Mazzeo, 2008; Wehrlin and Hallén, 2006). Thus, any submaximal absolute load represents a higher relative load. The intensity at which any endurance event can be performed is lower, as is the ability to sustain that activity. The blood lactate response for a given power output is greater compared with sea level. These responses are true for all individuals at all ages, although high-altitude mountain climbers are impacted the most simply by virtue of the extreme altitudes at which they perform. Sprints and other muscular strength,

muscular endurance, and power events do not appear to be physiologically disadvantaged, and they may even be enhanced because of lower air resistance at higher elevations (Burtscher et al., 2006; Haymes and Wells, 1986; Mazzeo, 2008; Ratzin Jackson and Sharkey, 1988).

Acclimation to Altitude

Acclimation to altitude begins quickly. It involves subtle but important shifts caused by the initial attempts to adjust to the lower oxygen partial pressure. Hemoconcentration continues, but the cause shifts from being a decrease in plasma volume with no change in red blood cell count to an increase in red blood cells under the influence of the hormone erythropoietin (Hall and Hall, 2021). Blood volume remains depressed for several weeks but then begins to increase (Haymes and Wells, 1986). Likewise, hyperventilation continues, but instead of an increased frequency, an increase in V_T occurs. Increased V_T allows a more effective pulmonary gas exchange. P_aCO_2 remains depressed, but P_aO_2 increases somewhat as acclimation proceeds (Adams et al., 1975; Grover, 1978; Hall and Hall, 2021; Hannon, 1978; Haymes and Wells, 1986; Ratzin Jackson and Sharkey, 1988). Cardiac output is lower despite the continued elevation in heart rate thus reflecting primarily a reduction in stroke volume with acclimation linked to the slow (>2 months) recovery of blood volume to sea-level values (Lundby, 2012). Muscle blood flow and hence oxygen delivery to the muscles remain unchanged, but to an extent, these are compensated for by increased extraction of

oxygen $[(a-v)O_2\text{diff}]$. $\dot{V}O_2 \text{ max}$ remains reduced. Upon altitude exposure, lactate values are higher for any given workload. After years of debate, it now appears that this response remains despite overall acclimatization (Lundby, 2012). Females seem to acclimatize to altitude more readily than males (Grover, 1978). **Table 10.4**, third column, summarizes the respiratory chronic adaptations.

Training and Competing at Altitude and Sea Level

Athletes who wish to compete at altitude have two choices in

attempting to minimize the adverse environmental effects. The first is to arrive at the altitude site 12–18 hours before the competition. Many U.S. collegiate and some professional teams do this, even at the moderate altitudes where they compete. Although this strategy does not prevent the effects of altitude, it does minimize the chances that acute altitude sickness will impair performance ([Haymes and Wells, 1986](#); [Ratzin Jackson and Sharkey, 1988](#); [Weston et al., 2001](#)).

The second choice is to train at the same altitude as the eventual competition. During training, athletes can acclimate somewhat to the altitude. However, even with extensive acclimation, a former sea-level resident will not be as physiologically adapted as the individual who was born and has always lived at altitude.

In 2007, the [International Federation of Association Football](#) (Fédération Internationale de Football Association or FIFA) passed a regulation regarding the matches in FIFA competitions at high altitude for both players and officials. The guidelines are as follows:

1. Above 2,500 m (~8,200 ft), an acclimation period of 3 days is strongly recommended.
2. Above 2,750 m (~9,000 ft), there is a mandatory acclimation period of 1 week.
3. Above 3,000 m (~9,800 ft), games are generally not permitted except with a minimum acclimation period of 2 weeks.

Although most altitude training studies have been done on individual sport endurance athletes (runners, cyclists, swimmers), the regulations just stated point out the importance of altitude on performance in endurance-based team sports such as soccer and rugby. In particular, the concern in these sports is the capacity to perform consecutive accelerations or sprint endurance. Data exhibit a greater probability of a loss for a sea-level team playing against a moderate-/high-level altitude team and consistently show less distance covered during games played at altitude. Intermittent hypoxic training (IHT), that is, interval training under hypoxic conditions, sometimes known as repeated sprint

training in hypoxia (RSH), may be particularly beneficial for these team sport athletes (Billaut and Aughey, 2013; McLean et al., 2014).

Unfortunately, the processes of acclimation do not completely counteract the fundamental hypoxic stress of altitude. Endurance athletes training at altitude cannot train at the same level of intensity for as long as they could at sea level. A reduction of training intensity to 40% of that at sea level may be necessary initially, and intensity can usually be increased only up to 75% of sea-level intensity (Armstrong, 2000). Highly trained athletes (especially those prone to exercise-induced arterial hypoxemia) are likely to benefit the least from altitude training (Sinex and Chapman, 2015). Untrained or minimally trained individuals, however, may still be doing more training than they did at sea level and reap the benefits of altitude training.

Athletes who train at altitude usually see positive results from that training if the competition is at the same altitude and occurs directly after the training period. The effect of altitude training on later sea-level maximal performance is highly dependent upon the type of altitude training undertaken, the fitness and performance status of the athletes (subelite or elite), the adaptation response of the individual athlete, the event, and the timing of the competition. Results show large variations in response and among individuals and techniques (Mazzeo, 2008; Sinex and Chapman, 2015). And techniques vary widely. For example, both natural/terrestrial and artificial altitudes may be utilized. Natural altitude training varies by elevation and where the athlete lives and/or exercises. Artificial altitudes can use either normobaric hypoxia (atmospheric pressure is unchanged but the fraction of inspired O₂ is decreased) or hypobaric hypoxia (atmospheric pressure is decreased while the fraction of inspired O₂ is maintained at sea-level value).

One group of athletes who must always perform at natural altitude is mountaineers. They typically follow a pattern of training and acclimation called “work (climb) high, sleep low,” although, of course, at some point, the sleep low is actually at a high altitude. Climbers traditionally establish a series of camp sites, each at a higher altitude than the previous one. During the day, they and their porters (Figure 10.12) carry supplies up to

the higher camp (camp 2) and work at that altitude, and then they descend at night to camp 1 to sleep. This pattern continues for days or weeks, at which time the climbers reestablish themselves between the old high camp (camp 2) and a new higher location (camp 3, progressing then from 3 to 4, 4 to 5) until they are in position for the final ascent of the summit.



Figure 10.12 Mountain Climbers Acclimate to Altitude by Establishing a Series of Camps in Which They Progressively Work High and Sleep Low until Attempting the Summit.

The most popular current strategy for nonmountaineer athletes who wish to improve competitive performance at sea level is really a variation of what mountaineers do called “live high, train low” ([Brugniaux et al., 2006a,b](#); [Koistinen et al., 2000](#); [Levine and Stray-Gundersen, 1997](#); [Wehrlin et al., 2006](#)). The goal is to avoid the detrimental effects of altitude (having to train at greatly reduced exercise intensity, with the concomitant detraining effects), while at the same time reaping the benefits of altitude acclimation, which can improve oxygen-carrying capacity and dissociation. The Focus on Research box describes the

definitive classic study that evaluated this approach. As is often the case with any training technique, individual reactions to “living high, training low” can vary widely, and some individuals will not respond at all.

Although “living high and training low” might seem to simply require a mountain with a nearby accessible valley, in practice, such geographical features can be difficult to find and access. Another option now being used experimentally is artificial altitude in a nitrogen house; an airtight dwelling flushed with air diluted with nitrogen. This reduces the oxygen content from 20.93% to approximately 15% and simulates being at altitude. To train low, athletes simply leave the house ([Koistinen et al., 2000](#)). A much less costly option requiring further research is the use of a nitrogen tent. The tent can be set up on any bed or floor and used by an individual when resting and sleeping in his or her own home. Additional simulated situations include the use of hypobaric chambers or hypoxic inhalers.

FOCUS ON APPLICATION | *Clinically Relevant*

Live High, Train Low

This study was designed to test the hypothesis that acclimation to living at moderate altitude (2,500 m, 8,260 ft) combined with training at low altitude (1,250 m, 4,125 ft) (high-low condition) would improve sea-level (5,000 m, 3.1 mi) performance in already well-conditioned athletes more than both living and training either at high altitude (2,500–2,700 m, 8,260–8,900 ft), called high-high condition, or at sea level, called low-low control condition.

Thirty-nine athletes completed 2 weeks of sea-level familiarization and 4 weeks of sea-level training before being randomized into three groups of 13 athletes (nine males and four females in each group) for 4 weeks of high- or low-altitude or sea-level training and living. Training was periodized and tapered before all tests for all groups. As expected, those training at high altitude did so at a slower

speed and at a lower percentage of maximal oxygen uptake than those at sea level. However, the training intensity of the low-altitude training group was reduced by only 6% compared with the sea-level group, whereas that of the high-altitude training group was reduced by 18.5%. Both groups that lived at moderate altitude significantly increased red

blood cell mass by 9% and $\dot{V}O_2 \max$ by 5%, while the sea-level group showed neither change. Arteriovenous oxygen difference was significantly higher for both altitude groups at velocities near 5,000-m run time trial speeds after altitude

training. Velocity at $\dot{V}O_2 \max$ increased only for the high-low group. The only group that significantly improved its 5,000-m time was the high-low group, by an average of 13.4 ± 10 seconds. This improvement persisted for at least 3 weeks after the return from altitude, at which point testing stopped.

These results suggest that an improvement in sea-level performance from altitude training is possible if athletes live at moderate altitude but train at lower levels that permit maintaining a high training intensity.



Source: Levine, B. D., & J. Stray-Gundersen: “Living high—training low”: Effect of moderate-altitude acclimatization with low-altitude training on performance. *Journal of Applied*

A meta-analysis (Bonetti and Hopkins, 2009) investigated the effects on performance measured at or near sea level and related physiological measures of adaptation to six different variations of altitude training. These were (1) natural live high, train high (LHTH); (2) natural live high, train low (LHTL); (3) artificial (simulated) LHTL with long (8–18 hours) continuous exposure to “altitude”; (4) artificial LHTL with brief (1.5–5 hours) continuous exposure to “altitude”; (5) brief (<1.5 hours) intermittent periods of artificial hypoxia; and (6) artificial live low, train high (LLTH). Subelite athletes were found to achieve substantial enhancement of maximal endurance power output with #2 (natural live high, train low [4.2%]), #5 (artificial brief intermittent LHTL [2.6%]), and #3 (long continuous artificial LHTL [1.4%]). Elite athletes benefited only from #2 natural LHTL (4.0%). Thus, LHTL seems best for both nonelite and elite athletes.

If the “live high, train low” approach is taken, the following guidelines are important (Brugniaux et al., 2006a,b; Lundby, 2012; Rusko et al., 2004; Sinex and Chapman, 2015):

1. For those with sufficient time and resources, the LHTL model with natural altitude exposure is recommended. The altitude should be at least between 2,100 and 2,500 m (~6,700–8,200 ft) but less than 3,000 m (~9,800 ft) while training should take place at $\leq 1,250$ m (~4,100 ft or less). If circumstances require it, simulated altitude can be used.
2. Hypoxic exposure should be greater than 12 hr·d⁻¹, and preferably 16 hr·d⁻¹.
3. Training should last at least 3–4 weeks.
4. High-intensity training must be maintained as if altitude were not involved.

The best time to return from altitude training prior to a major competition for peak performance remains to be determined. Conventional wisdom includes several phases relative to performance expectations: (1) return days 1–7, an initial improvement; (2) days 3–14, decrements in performance and

reduced training capability; (3) days 14–20+, a high plateau in performance; and (4) days 36–46, possible benefits. Even given the overlapping days, the best timing may ultimately depend upon individual responses. Athletes who exhibit a high level of ventilatory acclimatization or mechanical limitations to ventilatory flow may need a period of sea-level training before competition; athletes with a faster than normal decline in RBC mass may need to compete as soon as possible upon return from altitude. Individual variation in the response to altitude training is not yet fully understood (Chapman et al., 2013; Sinex and Chapman, 2015). Assess your comprehension of the effects of altitude by completing the [Check Your Comprehension 1—Case Study 1](#).

CHECK YOUR COMPREHENSION 1—CASE STUDY 1

Twins Carter (M) and Sydney (F), 16-years-old, are moving with their family from Virginia Beach, VA (altitude ~10 ft/3 m) to Taos Ski Valley, NM (altitude 9,321 ft/2,841 m) in late Aug. They have registered for school and know that they will be taking the FitnessGram® physical fitness test after their arrival. Their previous scores for each item are listed below.

| | Carter | Sydney |
|---|--------|--------|
| 1-Mile run | 8:08 | 10:04 |
| $\dot{V}O_{2\max}$ mL·kg ⁻¹ ·min ⁻¹ | 43 | 36.5 |
| Curl-ups | 32 | 26 |
| 90-Degree push-ups | 28 | 12 |
| Back saver sit-and-reach | 8 | 12 |
| BMI kg·m ⁻² | 21.5 | 23.4 |
| Trunk lift | 12 | 12 |

Assuming that they have maintained their activity level since this last fitness test, would you expect their score to be the same (=), higher (>), or lower (<) for each item in the upcoming Taos Ski Valley test? Explain your answers.

Check your answer in Appendix C.

Physical Activity and Pollution

Air pollution consists of a mixture of many different chemicals. The major components of automotive pollution include sulfur dioxide (SO₂), nitrogen oxides (NO_x), ozone (O₃), particulate matter (PM), and carbon monoxide (CO). Cigarette smoke, fires, and by-products of the combustion of other fuels also contribute to air pollution. Although the primary concern in this text are those who purposely exercise to achieve fitness or in preparation for athletic competition, it should be remembered that all individuals who must travel to work or whose primary place of employment is outdoors (city street workers, construction workers, bicycle messengers, active military personnel deployed in difficult environments) or indoors with poor air quality are also vulnerable to the physiological effects of pollution (Choudhary and Tarlo, 2014; Korzeniewski et al., 2013; Nyhan et al., 2014; Quin et al., 2019; Tainio et al., 2021).

The impairment of cilia function mentioned in Chapter 9 is not the only respiratory effect of inhaling these pollutants. Sulfur dioxide is absorbed by the moist surfaces of the upper airways and can cause bronchospasm. Sulfur dioxide peaks at midday but is rarely a major problem. Normal values approximate 0.005 ppm and sulfur dioxide does not negatively affect exercise until levels of 0.2 ppm (Marr and Ely, 2010). Nitrogen oxides are absorbed by the mucosal lining of the nose and pharynx and lead to irritation, cough, dyspnea, and diminished resistance to respiratory infection. Levels of NO_x are usually relatively low (~0.001 ppm), but values above 1 ppm would detrimentally affect exercise (Marr and Ely, 2010). Ozone is formed naturally by the action of ultraviolet radiation (UVR) on oxygen as UVR enters the earth's atmosphere and by the action of sunlight UVR on automobile exhaust (Armstrong, 2000). O₃ levels are typically around 0.03 ppm and become a problem for exercise at 0.05 ppm (Marr and Ely, 2010). As a respiratory irritant, ozone impairs pulmonary function, causes lung inflammation, and may interfere with lung defense mechanisms (Giles and Koehle, 2014). Ozone causes a decrease in tidal volume, forced vital capacity (FVC), and FEV₁ and an increase in breathing frequency and airway resistance. Postexercise pulmonary symptoms of ozone exposure include chest tightness, wheezing, and shortness of breath. In

communities with high concentrations of ozone, the risk of children developing asthma is related to the time they spend outside and the number of outdoor sports they play. Acute ozone exposure reduces maximal exercise time, workload, oxygen consumption, and maximal performance (Giles and Koehle, 2014). Ozone levels are higher in summer than in winter because of the greater sunlight and elevated temperatures, and in rural rather than urban areas.

PM is solid or liquid materials produced from fuel combustion that remain suspended in air for long periods of time. Sources of PM include wood fires, fossil fuel combustion from lawn mowers, snowblowers, string trimmers, restaurant stoves, incense and candle burning, vehicular traffic, power plants, laser printers, and windblown dust. PM is categorized by size into coarse, fine, and ultrafine. Particulates greater than 10 μm in diameter are not considered harmful to airways since they are generally filtered by the nasal passageways. Coarse particulates are usually between 10 and 2.5 μm in diameter and are labeled as PM₁₀. Fine particulates are between 0.1 and 2.5 μm in diameter (PM_{2.5}) and ultrafine particles are less than 0.1 μm (PM_{0.1}). All three of these sizes are smaller than the diameter of a human hair (Giles and Koehle, 2014). The smaller the particles, the deeper they can penetrate into the lungs. Experiments have shown that the deposit of ultrafine particles in the airways is high during mouth breathing in healthy individuals at rest and increases more than 4.5 times as much during moderate exercise (Daigle et al., 2003). As the intensity of exercise increases, the volume, rate, and depth of breathing also increase. An athlete running at 70%

$\dot{V}\text{O}_2 \text{ max}$ for approximately 3 hours during a marathon inhales the same volume of air as a sedentary person would in 2 days! When the ventilatory rate exceeds 30 $\text{L}\cdot\text{min}^{-1}$ (which it does easily even in low-intensity submaximal exercise), a combination of nasal and mouth breathing begins. This is important because a portion of the air then bypasses the nasal filtration, warming, and humidifying system (Sacha and Quinn, 2011). Additionally, as a result, a greater percentage of inhaled pollutants penetrate more deeply into the respiratory tract than at rest. Particulate pollution is highest in heavy smog, which can also include ozone. Lead is associated with particulates, and a significant relationship has

been shown between training duration and blood lead accumulation. The effects of particulates include systemic oxidative stress, airway inflammation, vascular dysfunction, increased airway resistance, and decreased capacity for oxygen exchange. PM increases breathing frequency and decreases tidal volume (Carlisle and Sharp, 2001; Giles and Koehle, 2014; Rundell, 2012). There is an inverse dose-response relationship between air pollutants and lung function at constant exercise workloads (Marr and Ely, 2010). Concerns about air pollution are not limited to those who exercise out of doors. In fact, indoor air quality can have important effects on physiological function (Andrade and Dominski, 2018). The prevalence of exercise-induced bronchoconstriction (EIB), asthma, and low resting lung function is high for individuals who train and compete in high PM environments. Indoor winter athletes, especially ice hockey players and figure and speed skaters, can be particularly vulnerable. Ice resurfacing by internal combustion fossil-fueled machines (gas and/or propane) produces levels of CO, NO_x, and PM 20–30 times greater than outside air. The use of electric-powered ice resurfacers maintains air quality. In addition to these health aspects, inhalation of high levels of PM during exercise has been shown to result in decreased performance in the range of 3–5%. In elite competitions, this could be enough to separate the winner and last-place finisher. Even a light 20-minute warm-up or one 6-minute bout of exercise in high-pollution conditions may have a detrimental carryover effect that could last for 3 days (Haymes and Wells, 1986; McCafferty, 1981; Rundell, 2012).

If the pollutant is CO, it reduces both the ability to carry oxygen and the ability to release oxygen already bound to red blood cells. The affinity of hemoglobin for CO is 210–240 times greater than its affinity for oxygen, and CO binds at the same site where oxygen would. Thus, when carboxyhemoglobin (COHb) is formed, the arterial percent saturation of oxygen decreases. The release of oxygen from hemoglobin is impaired by a shift in the oxygen dissociation curve to the left (see **Figure 10.2**). Myoglobin (Mb, the oxygen transporting and storage protein of muscles) and its role in assisting oxygen diffusion through the sarcoplasm to the mitochondria are also affected. First, the decreased release of oxygen from the red blood cells reduces the efficiency of Mb for attracting and holding oxygen within the

muscle cells. Second, CO binds directly to Mb with approximately the same affinity as to Hb, thereby reducing Mb's ability to combine with whatever oxygen is available. The combined result of the effects of elevated CO levels on Hb and Mb is an earlier and possibly greater dependence on anaerobic metabolism.

This is manifested by a lower exercise intensity at which anaerobic metabolism becomes important, a shorter endurance time at submaximal loads, a lower maximal exercise performance, a lower maximal a-vO₂diff, and a lower maximal oxygen

consumption ($\dot{V}O_{2\text{ max}}$) (McDonough and Moffatt, 1999). As little as a 4% COHb level will have detrimental effects on exercise time and intensity. This level may result if training is done near heavy traffic (**Figure 10.13**). CO inhaled by smoking has additional respiratory impact, including increased pulmonary airway resistance, increased oxygen cost of ventilation, and an increased diffusion distance for oxygen and carbon dioxide across the alveolar walls because of mucosal swelling and bronchial constriction (McDonough and Moffatt, 1999). The higher the

%COHb, the higher the \dot{V}_E for the same work rate (Giles and Koehle, 2014). COHb levels of 5% can increase heart rate during exercise. Individuals smoking 10 or fewer cigarettes per day average approximately 4% COHb, and a two-pack-a-day habit almost doubles this value. A nonsmoker riding in a car for 1 hour with a smoker can reach 3% COHb level (Haymes and Wells, 1986). Strenuous exercise near heavy traffic for 30 minutes can increase the level of COHb as much as smoking 10 cigarettes (Carlisle and Sharp, 2001; Giles and Koehle, 2014). The half-life of COHb is 3–4 hours, meaning that it takes that long for one half of the CO to become unbound to hemoglobin and be removed.



Figure 10.13 Training Near Traffic Will Increase COHb and Particulate Matter Levels.

Swimmers have unique pollutants to deal with. The reaction of chlorine-containing agents with organic nitrogen-containing compounds (e.g., dirt, sweat, and urine) brought into the pool water by users leads to the formation of various chloramine by-products. Some of these are transferred to the atmosphere as gases or droplets just above the water as the surface of the water is churned up. These are inhaled by swimmers. One of them, nitrogen trichloride (NCl_3), is known to cause acute disruption of airway epithelium. NCl_3 concentration becomes a problem at levels above 0.3 mg.m^{-3} . A flow rate of fresh air into a swimming pool of at least $60 \text{ m}^3.\text{h}^{-1}$ will reduce the concentration of chloramines that accumulate in the air above pool water. Breathing air containing chloramines during the many hours of training in indoor pools appears to be the reason for the high prevalence of airway hyperresponsiveness, asthma, and exercise-induced bronchoconstriction in swimmers (Kippelen et al., 2012; Nicholas, 2015).

Interaction between Physical Activity and Pollutants

Physical activity/exercise leads to improved health outcomes, as described throughout this text. Air pollution leads to impaired health as briefly described above. But, there are multiple ways that physical activity and pollution interact. These interactions are of concern to individual exercisers, coaches and fitness leaders, and policy makers. An increase in physical activity or sporting events may increase the inhalation of pollutants, which can lead to respiratory or cardiovascular irritation (as detailed above). On the other hand, a change in commuting patterns with more people walking or biking to work has the potential to decrease air pollution due to a decrease in vehicle traffic. An increase in air pollution, such as heavy smog or smoke from large forest fires, can also lead to a decrease in physical activity ([An et al., 2018](#)). These interactions warrant additional research. See the Literature Search box at the end of the chapter to further explore the research that has been done in this area.

Athletes are often affected by pollution levels that do not bother spectators. Individuals with cardiovascular and respiratory diseases and children are also particularly vulnerable ([McCafferty, 1981](#)). The following recommendations are suggested to minimize the impact of pollutants on an exercise training session or competition ([Armstrong, 2000](#); [Campbell et al., 2005](#); [Cutrufello et al., 2012](#); [Giles and Koehle, 2014](#); [Kippelen et al., 2012](#)):

1. Individuals with health problems that make them particularly susceptible to the effects of pollution should not exercise outside during air quality warnings. They should seek sites where the air is filtered.
2. Everyone should avoid prolonged heavy exercise when hazardous air warnings are in effect. Individuals can adapt to breathing pollutants, but in the long term, adaptation is harmful because it suppresses normal defense mechanisms. Therefore, adaptation should not be attempted. Anyone experiencing symptoms such as coughing, wheezing, chest tightness, pain with breathing deeply, or difficulty breathing should reduce their activity level and seek medical attention.
3. Atmospheric ozone levels peak at around 1–3 PM and are much higher during most of the daylight hours in summer

than in winter. CO peaks at approximately 7 AM and 8 PM and is higher in winter than in summer. Thus, heavy outdoor workouts might be best early in the morning and late evening during the summer and around noontime in the winter.

4. Runners, cyclists, and in-line skaters should seek locations away from heavy vehicular traffic. Exercise should not be performed within 250 m of major roadways and should be avoided during times of high vehicular congestion. Trailing closer to a pace car than 50 ft or waiting at stoplights behind cars' exhaust pipes should be avoided. At the very least, athletic venues, parks, and exercise areas near high-traffic roads should have evergreen trees planted between the road and the facility.
5. Smoking should be banned from all indoor training and competition sites. Smoking should be discouraged at all times. Individuals need to be mindful that environments in night clubs and/or casinos are likely to be cigarette smoke-filled locations.
6. Arenas that use fossil-fueled ice-resurfacing machines and have poor ventilation should be avoided. Skiers should be deterred from spending long hours in the hot waxing room and should make sure the area is always well ventilated. Cold weather exercisers can employ strategies that increase temperature and water content of the inhaled air that may include face masks at subfreezing temperatures. However, these devices make it difficult to train and/or compete at high intensity.
7. To avoid the production of as many chloramines as possible, swimmers should shower with soap before entering the pool, wear a bathing cap, use a swimsuit reserved exclusively for swimming, remove makeup, and respect bare-feet zones.

To evaluate your understanding of respiratory factors that may impact maximal exercise performance, complete the [Check Your Comprehension 2 box](#).

CHECK YOUR COMPREHENSION 2

Which two of the following conditions/situations that can decrease maximal exercise performance share the same basic physiological causes? Explain your choices.

1. Altitude ($> \sim 2,500$ ft)
2. Breathing polluted air
3. Entrainment
4. Hypoxic swimming (controlled-frequency breathing)
5. EIAH

Check your answer in Appendix C.

Summary

1. During short-term, light to moderate aerobic exercise, minute ventilation, alveolar ventilation, and the arteriovenous oxygen difference ($a-vO_{2diff}$) increase rapidly and reach a steady state within approximately 2–3 minutes. The partial pressure of oxygen at the alveolar and arterial levels does not change, and, as a result, the alveolar to arterial oxygen pressure gradient and percent saturation of arterial hemoglobin with oxygen are maintained. The percent saturation of oxygen in venous blood and the partial pressure of oxygen in venous blood decrease rapidly and reach a steady state within approximately 2–3 minutes.
2. During the first 30 minutes of long-term, moderate to heavy aerobic exercise, the only meaningful differences in respiratory responses from short-term, light exercise are in magnitude and a slight drop in the arterial partial pressure of oxygen; this in turn widens the alveolar to arterial oxygen pressure gradient. After approximately 30 minutes, minute ventilation, alveolar ventilation, and the $a-vO_{2diff}$ exhibit a slight upward drift. This respiratory drift is associated with a rising body temperature.
3. During incremental aerobic exercise to maximum, both minute ventilation and alveolar ventilation exhibit a rectilinear rise interrupted by two breakpoints that change

the slope upward. The $a-vO_2\text{diff}$ rises rectilinearly until approximately 60% of maximal work, where it levels off. The partial pressures of both alveolar and arterial oxygen and the resultant alveolar to arterial oxygen pressure gradient and percent saturation of arterial blood remain constant until approximately 75% of maximal work. At this point, the first three exhibit a slight exponential rise, and the arterial oxygen saturation percent decreases slightly as a result. Both the percent saturation of oxygen in venous blood and the venous oxygen partial pressure decrease rectilinearly until approximately 60% of maximal work, where they level off. As the oxygen is extracted and used, carbon dioxide is produced so that the partial pressure of carbon dioxide in venous blood rises.

4. All of the respiratory responses to static exercise are the same as for short-term, light to moderate submaximal aerobic exercise except that oxygen extraction, as denoted by the $a-vO_2\text{diff}$, either shows no change or decreases slightly and minute ventilation and $a-vO_2\text{diff}$ exhibit little, if any, change during exercise, with a rebound rise in recovery.
5. In most healthy sedentary or moderately trained individuals, the respiratory system has more than enough reserve for the ventilatory and gas-exchange demands for exercise of any intensity. However, there are three situations in which the respiratory system may limit high-intensity exercise: (1) exercise-induced arterial hypoxemia (EIAH), (2) respiratory muscle fatigue, and (3) excessive fluctuation in intrathoracic pressure. A primary cause of EIAH is a widening of $(A-a)PO_2\text{diff}$, the failure to maintain arterial oxygen saturation.
6. During exercise, oxygen dissociation is increased by a widening of the oxygen pressure gradient, an increase in partial pressure of carbon dioxide, a decrease in pH, and an increase in body temperature. The changes in PCO_2 , pH, and body temperature increase oxygen dissociation by causing a rightward shift of the oxygen dissociation curve. At altitude, the tendency for the decrease in PCO_2 , caused by the compensatory hyperventilation, to shift the curve to the left and impair oxygen dissociation is counteracted by an increase in 2,3-diphosphoglycerate activity.

7. Locomotor-respiratory coupling (entrainment) occurs when limb movement is synchronized with breathing frequency. Exercising individuals should use either an entrainment or a spontaneous breathing pattern, whichever comes more naturally to them.
8. In general, exercise responses are in the same direction but may vary in magnitude across the age span for both males and females.
9. Many of the differences in respiratory measures when comparing children and adolescents with young adults or males with females are at least partially related to size. Smaller individuals have smaller values.
10. Specific respiratory muscle training results in increases in functional measures of inspiratory muscle strength, endurance, maximal rate of shortening, maximal sustainable ventilatory capacity, and power. Structurally, significant increases in the proportion of type I (slow-twitch, oxidative) fibers and the size of type II (fast-twitch, oxidative glycolytic) fibers have been observed. Often, but not always, the perception of dyspnea is decreased. Respiratory values for minute ventilation, alveolar ventilation, breathing frequency, and tidal volume as well as oxygen consumption and lactate concentration are typically lowered and actual performance increased during constant submaximal performances or time trials, whereas no improvement is seen at maximal exercise.
11. Whole body training respiratory adaptations are minimal in land-based athletes or fitness participants. The most consistent change is in minute ventilation, which goes down during submaximal work and increases at maximum. Yoga, swimming, and diving show increases in most static and dynamic lung volumes and capacities, particularly in total lung capacity and vital capacity.
12. The relative percentages of oxygen, carbon dioxide, and nitrogen remain the same to an altitude of 100,000 m (328,083 ft). However, because barometric pressure goes down exponentially as altitude increases, the partial pressures exerted by oxygen and carbon dioxide also decrease with altitude. The result is a lower percent

saturation of oxygen in the arterial blood and a decreased ability to maintain high-intensity aerobic exercise.

13. Individuals who must compete at altitude should arrive at the location either 12–18 hours or 3 days to 4 weeks before the event. Acclimation and training at altitude appear to be beneficial for competing at that same altitude. Evidence supports the use of a “live high, train low” regimen for improvement in sea-level performance for endurance athletes. Not everyone will be a positive responder to altitude training.
14. Exercise in highly polluted air should be avoided. Any adaptation to such conditions is done at the expense of natural defense mechanisms.

Review Questions

1. List and explain the four factors that increase oxygen dissociation during exercise and how these are related. Describe the additional factor that influences oxygen dissociation at altitude.
2. Compare and contrast the pulmonary ventilation, external respiration, and internal respiration responses to short-term, light to moderate submaximal aerobic exercise; long-term, moderate to heavy submaximal aerobic exercise; incremental aerobic exercise to maximum; and static exercise. Where known, explain the mechanisms for each response.
3. Should fitness participants and athletes be encouraged to practice locomotor-respiratory coupling (entrainment) rather than spontaneous breathing? Why or why not?
4. Explain the three situations in which the respiratory system may limit exercise.
5. Compare the procedures and adaptations of specific respiratory muscle training and whole body training for respiratory variables.
6. Defend or refute this statement: “Hypoxic training, whether achieved by training at altitude or by breath holding, is beneficial to an athlete.”

7. Describe the major chemical air pollutants and their environmental sources. Explain the impact of these pollutants on exercise training. List recommendations for minimizing the impact of pollutants for those working or exercising.

Literature Search

1. Air pollution can place athletes, exercisers, and workers at increased risk of pulmonary and cardiovascular disease issues. To better understand the research that has been done on this important issue, do a literature search using a search engine such as pubmed, Google scholar, or Web of Science.
 - a. Search Exercise and Air Pollution. This search will yield many articles.
 - b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Health risks and exercise and air pollution
 - ii. Exercise duration and health risk of elevated CO
 - iii. Cardiovascular risks associated with increased PM during indoor sporting events
 - iv. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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11 The Cardiovascular System



Chapter Outline

Introduction

Overview of the Cardiovascular System

The Heart

The Vascular System

Blood

Cardiovascular Dynamics

Cardiac Output (\dot{Q})

Mean Arterial Pressure

Total Peripheral Resistance

Principles of Blood Flow

Regulation of the Cardiovascular System

Neural Control

Anatomical Sensors and Factors Affecting Control of the Cardiovascular System

Neurohormonal Control

Measurement of Cardiovascular Variables

Cardiac Output

Stroke Volume

Heart Rate

Maximal Oxygen Consumption

Blood Pressure

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Explain the functions of the cardiovascular system.
- Identify the various components of the cardiovascular system.
- Distinguish among the vessels that comprise the vascular system and compare the pressure, velocity, and resistance in each type of vessel.
- Describe the roles of the vascular endothelium.
- Explain how electrical excitation spreads through the conduction system of the heart.
- Explain the relationships among the electrical, pressure, contractile, and volume changes throughout the cardiac cycle.
- Calculate mean arterial pressure, total peripheral resistance, and cardiac output.
- Describe the hormonal mechanisms by which blood volume is maintained.
- Explain how the cardiovascular system is regulated.
- Describe how these variables are measured: maximal oxygen consumption, cardiac output, stroke volume, heart rate, and

blood pressure.

Introduction

Chapter 9 described how oxygen is taken into the body for delivery to body cells. The ability to deliver oxygen (and other substances) depends on the proper functioning of the cardiovascular system. In many ways, the cardiovascular system and the respiratory system operate together to accomplish a common mission—to deliver oxygen to working muscles—and they are driven by similar mechanisms. This chapter provides an overview of the cardiovascular system, discusses basic principles of cardiovascular dynamics, and outlines techniques typically used to assess cardiovascular function at rest, during, and following exercise.

Overview of the Cardiovascular System

The *cardiovascular system* includes the heart, blood vessels, and blood. Its primary functions are as follows:

1. To transport oxygen and nutrients to the cells of the body and to transport carbon dioxide and waste products from the cells
2. To regulate body temperature, pH levels, and fluid balance
3. To protect the body from blood loss and infection

The heart is a double pump that provides the force to circulate the blood throughout the vessels of the circulatory system. The blood vessels serve as sophisticated, dynamic conduits for the blood as it is distributed through the body. The blood is the medium that transports gases and nutrients within the cardiovascular system.

Figure 11.1 is a schematic overview of the cardiovascular system. *Arteries* carry blood away from the heart, and *veins* return blood to the heart. The *capillary beds* serve as the site of exchange for gases and nutrients between the blood and body tissues. Blood

is ejected from the ventricles on both sides of the heart simultaneously. The right ventricle pumps blood through the pulmonary arteries to the lungs, where it is oxygenated and then returned to the left atrium via the pulmonary veins; this is called the *pulmonary circulation*. The left ventricle pumps oxygenated blood through the aorta, which then branches extensively into arteries to carry the blood to body cells through numerous specific circulations. The partially deoxygenated blood returns to the right atrium. Collectively, this route from the left ventricle to the right atrium is known as the *systemic circulation*.

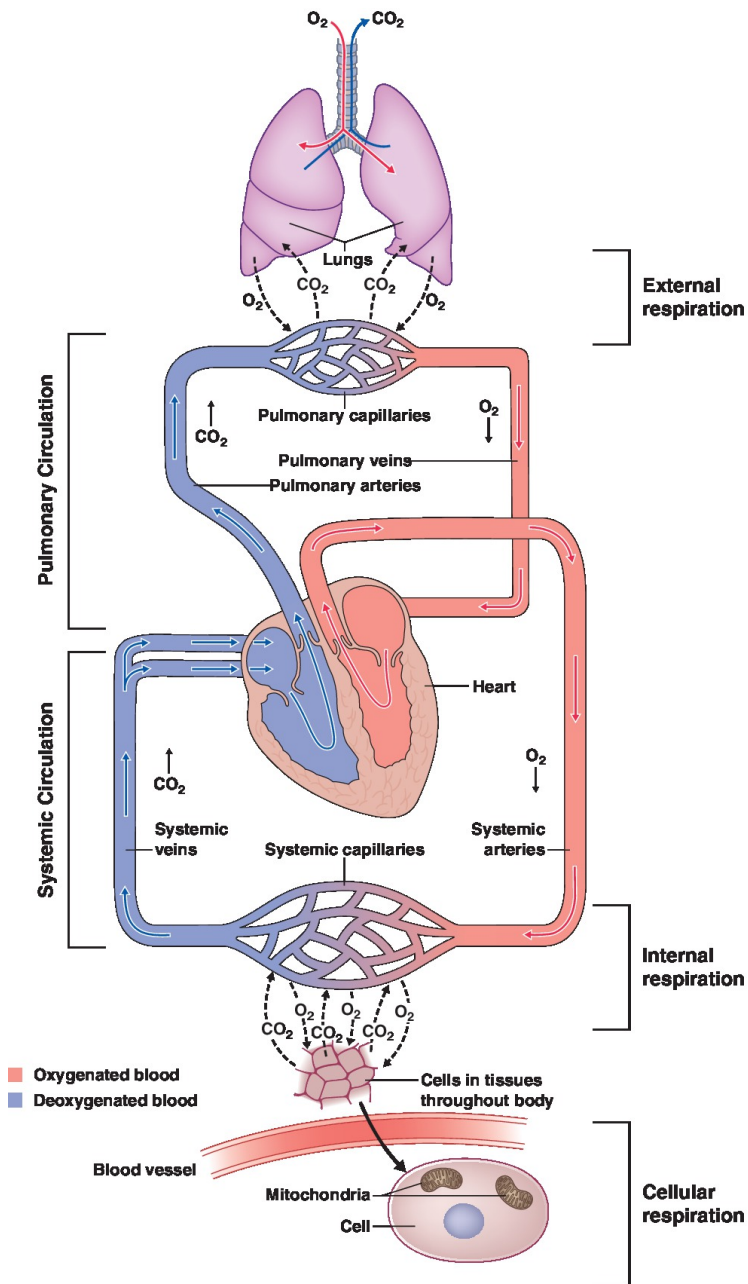


Figure 11.1 Schematic Overview of the Cardiovascular System and Interaction with the Respiratory System.

As described in the respiration chapters, *external respiration* is the exchange of gases (O₂, CO₂) between the lungs and blood. *Internal respiration* is the exchange of gases (O₂, CO₂) at the cellular level. The cardiovascular system functions primarily to move the gases (as well as nutrients from the digestive tract to the tissues) between these two exchange sites so that energy can be produced by cellular respiration. *Cellular respiration* is described completely in the metabolic unit.

The Heart

The heart is a hollow muscular organ located in the thoracic cavity. It weighs approximately 250–350 g and is 12–14 cm long, about the size of a clenched fist. The heart beats approximately 70 times per minute in a resting adult—or over 100,000 times per day!

Macroanatomy of the Heart

The heart has four chambers and is functionally separated into the right and left heart. The right side pumps blood to the lungs (pulmonary circulation), and the left side pumps blood to the entire body (systemic circulation). Heart muscle is called **myocardium**. The two sides of the heart are separated by the interventricular septum. The upper chambers, called *atria* (*atrium* is the singular), receive the blood into the heart. The lower chambers, called *ventricles*, eject blood from the heart (**Figure 11.2A**). Blood is ejected from the right ventricle to the pulmonary artery and from the left ventricle to the aorta.

Myocardium The heart muscle.

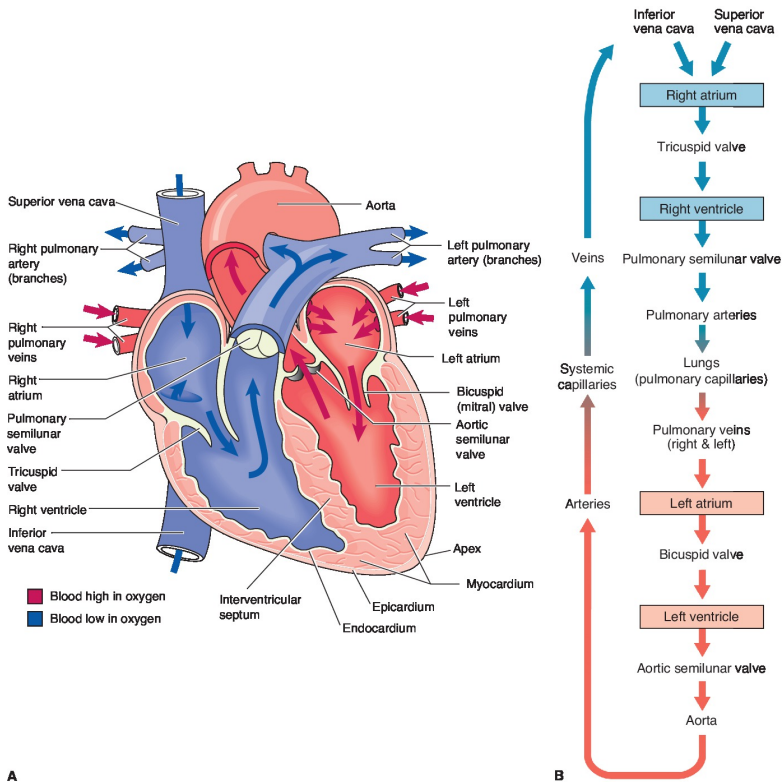


Figure 11.2 Blood Flow through the Heart.

A. Schematic of the heart. B. Summary of blood flow through the heart.

One-way valves control blood flow through the heart. The *atrioventricular (AV) valves* separate the atrium and ventricle on each side of the heart. Specifically, the tricuspid valve separates the atrium and ventricle on the right side of the heart, and the bicuspid (or mitral) valve separates the atrium and ventricle on the left side of the heart. The *semilunar valves* control blood flow from the ventricles. Specifically, the aortic semilunar valve allows blood to flow from the left ventricle into the aorta, and the pulmonary semilunar valve allows blood to flow from the right ventricle into the pulmonary artery.

Connective tissue plays a critical role in providing structure to the heart, anchoring it to surrounding structures, and preventing

overfilling. The heart is enclosed by a double-walled sac of connective tissue called the *pericardium* that is filled with pericardial fluid. The *epicardium* is the inner layer of the double-walled layer and is the layer of connective tissue that directly covers the myocardium. The *endocardium* is a single layer of endothelium (connective tissue) that lines the cavities of the heart. The heart contains a dense network of connective tissue fibers, including collagen and elastic fibers, that form a fibrous skeleton of the heart and reinforces the myocardial structure and provides an anchor for cardiac muscle cells. **Figure 11.2A** shows the major structures of the heart and the flow of blood. **Figure 11.2B** summarizes the blood flow through the heart and body.

See animation, Blood Circulation, on Lippincott Connect.



Microanatomy of the Heart

Cardiac muscle cells, called **myocytes**, are the contractile cells that produce the force that ejects blood from the ventricles. Cardiac muscle cells are both similar to and different from skeletal muscle cells. Both are striated in appearance because they contain the contractile proteins actin and myosin. The primary difference between cardiac and skeletal muscle cells is that cardiac muscle cells are highly interconnected, that is, the cell membranes of adjacent cardiac cells are structurally and functionally linked by **intercalated discs** (**Figure 11.3**). The intercalated discs contain specialized intracellular junctions (gap junctions) that allow the electrical activity in one myocyte to pass to adjacent myocytes. Thus, the individual cells of the myocardium function collectively: when one cell is stimulated electrically, the stimulation spreads from cell to cell over the entire area. This electrical coupling allows the myocardium to function as a single coordinated unit or a functional **syncytium**. Each of the two functional syncytia, the atrial and ventricular, contracts as a unit.

Myocytes The contractile cells of the heart (cardiac muscle cells).

Intercalated Discs The junction between adjacent cardiac muscle cells that forms a mechanical and electrical connection between cells.

Syncytium A group of cells of the myocardium that function collectively as a unit.

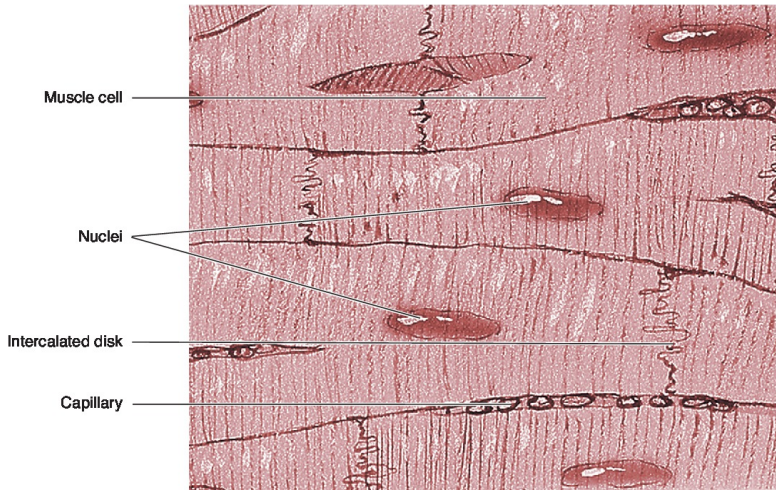


Figure 11.3 Cardiac Myocyte.

The Heart as Excitable Tissue

Cardiac muscle cells are excitable cells that are polarized (have an electrical charge with the inside being negative relative to the outside of the cell) in the resting state and contract when they become depolarized (the charges reverse). Repolarization (the electrical charge returns to resting value) results in relaxation of the myocytes. With each contraction, blood is ejected from the chambers. Individual myocardial cells function together to produce a coordinated contraction of the entire syncytium. Generally, when contraction of the heart is referred to, unless specified otherwise, it means contraction of the ventricles.

In addition to contractile muscle cells, the heart contains specialized conducting cells (**Figure 11.4A**). Although there are

far fewer conducting cells than contractile muscle cells, they are essential because they spread the electrical signal quickly throughout the myocardium. The conduction system cells with the fastest spontaneous rate of depolarization are called the *pacemaker* cells. These are located in the *sinoatrial (SA) node* in the right atria. As shown in **Figure 11.4A**, the excitation spreads from the SA node throughout the right atria by internodal tracts and to the left atria by *Bachmann's bundle*. Because the atrial and ventricular syncytia contract separately, excitation in the atria does not lead directly to the contraction of cardiac cells in the ventricles. The electrical signal is spread from the atria to the ventricles via the *atrioventricular (AV) node*. Once the AV node is depolarized, the electrical signal continues down the specialized conduction system consisting of the *bundle of His*, the *left and right bundle branches*, and the *Purkinje fibers*. The electrical excitation then spreads out from the conducting system to excite all of the cardiac muscle cells in the ventricles. Thus, the excitation is spread first by the conduction system and then by cell-to-cell contact: The excitation must be passed from muscle cell to muscle cell within the ventricles since the conduction system does not reach each individual myocyte.

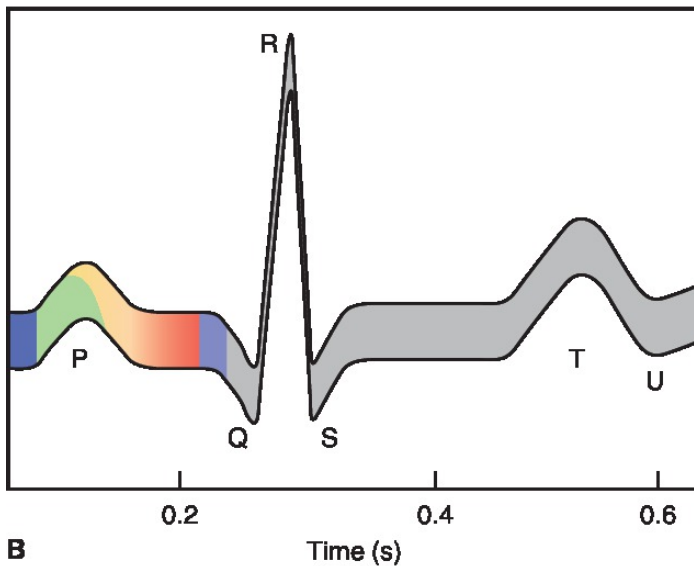
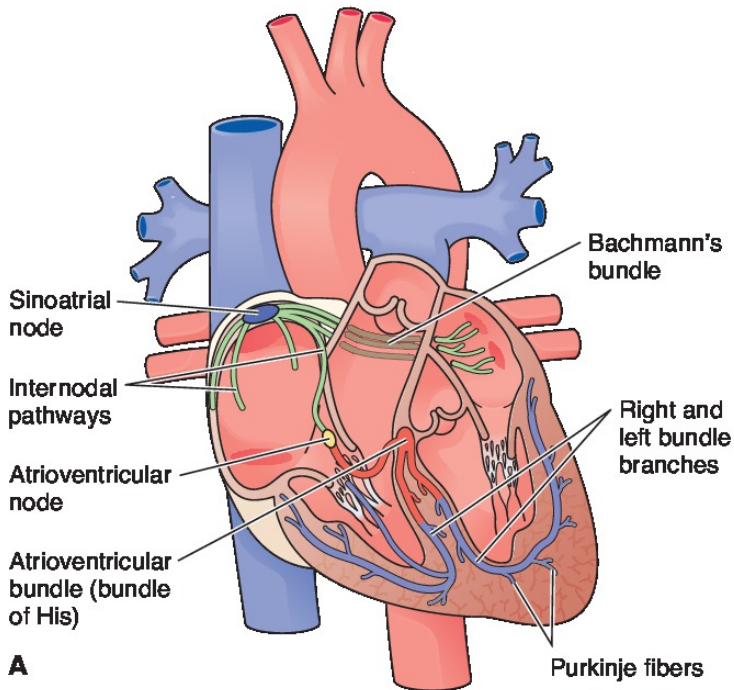


Figure 11.4 Conduction System of the Heart.

A. Conduction pathway of the heart. B. ECG tracing that is color coded to show movement of the electrical signal

through the conduction system and then through the heart.

As mentioned earlier, the cells of the SA node are considered the pacemaker cells of the heart because they normally have the fastest rate of depolarization. Cells in each area of the conduction system have their own inherent rates of depolarization. For the SA node, the intrinsic rate of depolarization is 60–100 times·min⁻¹. The AV node discharges at an intrinsic rate of 40–60 times·min⁻¹, and the Purkinje fibers at a rate of 15–40 times·min⁻¹ (Hall and Hall, 2021). If the SA node is diseased, the AV node may take over the pacemaking. While it is generally known that endurance training leads to a lower resting heart rate, if the only thing you knew about an individual was that he or she had a resting heart rate of 40 b·min⁻¹, you could not tell whether the person was a highly trained endurance athlete or someone in need of an artificial pacemaker implant because the SA node was not functioning properly.

Electrocardiogram

An **electrocardiogram (ECG)** provides a graphic illustration of the electrical current generated by excitation of the heart muscle. **Figure 11.4B** presents an ECG tracing that is color coded to match the movement of the electrical current through the conduction system in **Figure 11.4A**. The spread of the electrical signal through the conduction system of the atria is shown in green. The P wave represents atrial depolarization, which causes atrial contraction. Repolarization of the atria, which results in a T_a wave, is normally not detectable on a resting ECG but occurs during the time period concurrent with the QRS complex and may be evident during exercise. The electrical signal reaches the AV node at the end of the P wave (shown in *yellow*). Excitation of the bundle of His and bundle branches (shown in *red*) occurs in the middle of the PR interval, followed by excitation of the Purkinje fibers (shown in *purple*). Note that excitation of the various portions of the conduction system happens very quickly and that activation of the entire conduction system precedes the QRS complex. The QRS complex reflects depolarization of the myocytes in the ventricles. It occurs after the electrical signal has traveled through the specialized conduction system in the

ventricles and is occurring simultaneously with atrial repolarization. The T wave reflects repolarization of the muscle cells in the ventricles and is followed by relaxation in preparation to start the cycle all over again. The U wave may or may not be seen in a normal ECG but is often present in the slower cardiac cycle of trained individuals. When present, it probably represents the final phase of ventricular repolarization during which the Purkinje system recovers.

Electrocardiogram (ECG) Tracing that provides a graphic illustration of the electrical current generated by excitation of the heart muscle.

Although the SA node can depolarize spontaneously, the firing of the SA node is influenced by neural and hormonal factors (discussed later in the chapter). Additionally, heart rate varies with age. **Table 11.1** presents typical resting heart rate (HR) values in healthy individuals of various ages. While the resting heart rate values may not seem impressive when reported on a per minute basis, it is remarkable to consider how many times the heart beats per hour, or per day. Other factors that affect heart rate are discussed later in this chapter.

TABLE 11.1 Typical Resting Cardiovascular Values for Males and Females of Various Ages

| Cardiovascular Value | Age of Males (y) | | | Age of Females (y) | | |
|---------------------------------------|------------------|-------|-------|--------------------|-------|-------|
| | 10–15 | 20–30 | 50–60 | 10–15 | 20–30 | 50–60 |
| HR (b·min ⁻¹) | 82 | 72 | 80 | 85 | 76 | 82 |
| Stroke volume (mL·b ⁻¹) | 50 | 90 | 70 | 40 | 75 | 62 |
| Cardiac output (L·min ⁻¹) | 4.0 | 6.5 | 5.5 | 3.4 | 5.5 | 5.0 |

Sources: Åstrand (1952); Fleg et al. (1995); Ogawa et al. (1992); Spina et al. (1992, 1993a, 1993b).

Cardiac Cycle

To function successfully as a pump, the heart must have alternating times of relaxation and contraction. The relaxation phase, called **diastole**, is the period when the heart fills with

blood. The contraction phase, called **systole**, is the period when blood is ejected from the heart. The **cardiac cycle**—one complete sequence of contraction and relaxation of the heart—includes all events associated with the flow of blood through the heart. During the cardiac cycle, there are dramatic changes in pressure and blood volume. **Figure 11.5** summarizes the flow of blood in the heart and the position of the heart valves throughout the periods of the cardiac cycle. A key to understanding the periods of the heart is the fact that valves control the flow of blood through the heart. If the AV valves are closed, blood volume in the ventricles cannot increase; if the semilunar valves are closed, then blood cannot be ejected and volume does not decrease. Furthermore, it is pressure differences that control the valves. When pressure is greater in the ventricle than the aorta, it forces the one-way valve to open.

Diastole The relaxation phase of the cardiac cycle.

Systole The contraction phase of the cardiac cycle.

Cardiac Cycle One complete sequence of contraction and relaxation of the heart.

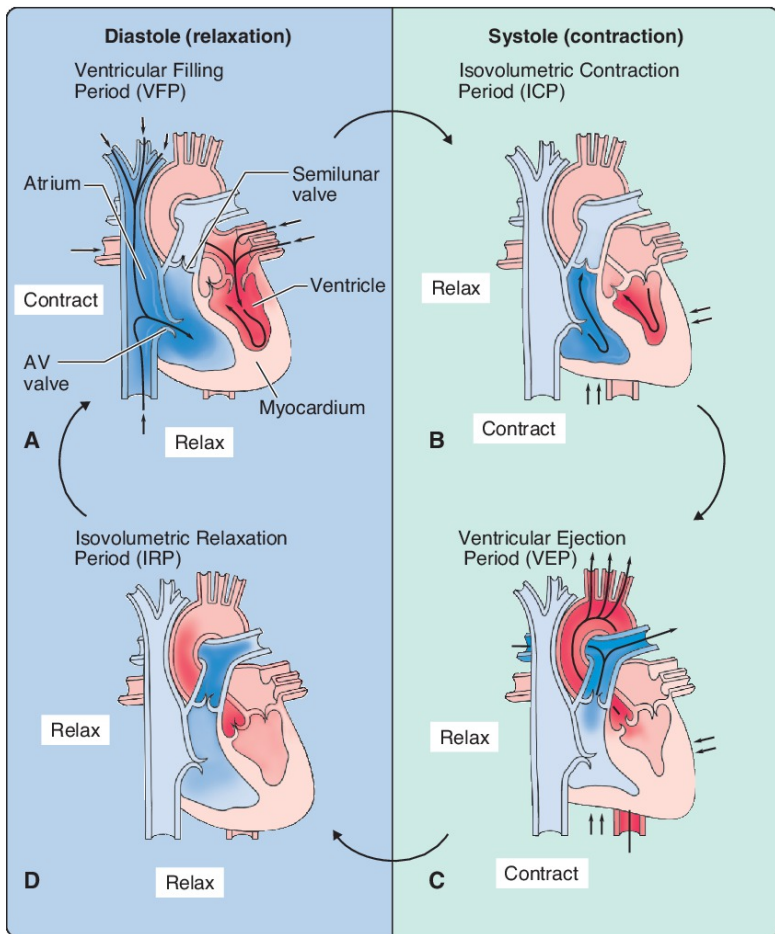


Figure 11.5 Periods of the Cardiac Cycle.

A. Ventricular filling period (VFP). The ventricles are relaxed. The AV valves are open, and venous blood is filling the ventricles. **B.** Isovolumetric contraction period (ICP). The contraction of the myocardium increases intraventricular pressure, but all the valves are closed and blood volume in the ventricles remains constant. **C.** Ventricular ejection period (VEP). Increasing ventricular pressure forces the semilunar valves to open, and blood is ejected from the ventricles. **D.** Isovolumetric relaxation period (IRP). The ventricles are relaxed, and all the valves are closed, so blood volume in the ventricles remains

constant.

The *ventricular-filling period (VFP)* (**Figure 11.5A**) occurs when the ventricles are at rest (ventricular diastole) and the AV valves are open. The ventricles fill as blood is returned to the atria and flows down into the ventricles. Blood flow into the ventricles from the atria is assisted by gravity in an upright person. Atrial contraction also pushes a small volume of additional blood into the ventricles at the end of diastole. Blood volume in the ventricles is greatest at the end of ventricular filling, but pressure remains relatively low because the ventricles are relaxed.

Systole (the contraction phase, shown on the right side of the figure) is divided into two periods, the *isovolumetric contraction period (ICP)* and the *ventricular ejection period (VEP)*. During the ICP (**Figure 11.5B**), both the AV valves and the semilunar valves are closed. Thus, for this very brief period, blood volume in the ventricles remains constant (*isovolumetric*) despite the high pressure generated by the contraction of the ventricular myocardium. Once the pressure in the ventricles exceeds the pressure in the aorta, the semilunar valves are forced open and blood is ejected from the ventricles, initiating the VEP. During VEP, ventricular volume decreases as blood is ejected from the ventricles through the open semilunar valves (**Figure 11.5C**).

During the *isovolumetric relaxation period (IRP)*, both the AV and the semilunar valves are closed (**Figure 11.5D**). Thus, ventricular volume is again unchanged (isovolumetric), but pressure is low because the ventricles are relaxed.

All these events occur within a single cardiac cycle, which repeats with every beat of the heart. **Figure 11.6** summarizes the cardiac cycle graphically, showing concurrent information about the electrocardiogram; the pressure in the left atrium, the left ventricle, and aorta; the left ventricular volume; the heart phase; the period of the cardiac cycle; and the position of the heart valves. As in **Figure 11.5**, diastole is shown in blue, and systole is shown in green. The position of the AV and semilunar valves is shown schematically in **Figure 11.5** and in the boxes at the bottom of **Figure 11.6**. The valves control the one-way flow of blood through the heart, so knowing if the valves are open or closed allows one to know whether blood is filling the ventricles,

leaving the ventricles, or if the volume of blood in the ventricles is the same.

FOCUS ON APPLICATION | *Clinically Relevant*

Are All Elevations in Heart Rate Equal?

Heart rate (HR) can be elevated by a variety of factors mediated by the neural and hormonal systems (see [Figures 11.16 and 11.17](#)). One of these factors is movement (exercise), but others include emotion and environmental temperatures. Does an individual derive the same benefit from HR elevation caused by emotion or heat as from HR elevation caused by exercise? That is, is it possible to improve one's cardiovascular function while sitting in a sauna or hot tub or when frightened, angry, or anxious?

A regular, sustained elevation in HR is recognized as an important factor for improving cardiovascular fitness (techniques of exercise prescription based on HR are described in [Chapter 13](#)). However, the exercise HR responses are primarily an indicator of the training stimulus to the body—the increase in energy expenditure or metabolism (oxygen consumption). Heart rate and oxygen consumption rise in a directly proportional fashion during exercise. When emotion or temperature causes an elevation in HR, however, minimal change occurs in energy expenditure; hence, there is no training stimulus.

The importance of an increase in oxygen consumption has been demonstrated by individuals on medications such as beta-blockers, which markedly suppress HR at rest and during exercise, and by those with constant heart-rate pacemakers. Both of these groups routinely show improvements in exercise capacity and fitness as a result of exercise programs, despite the fact that the exercise-induced increase in HR is dampened. Although HR can be elevated by other factors, you do not derive the health-related benefits unless the elevated HR is accompanied by physical activity.



Source: [Franklin and Munnings \(1998\)](#).

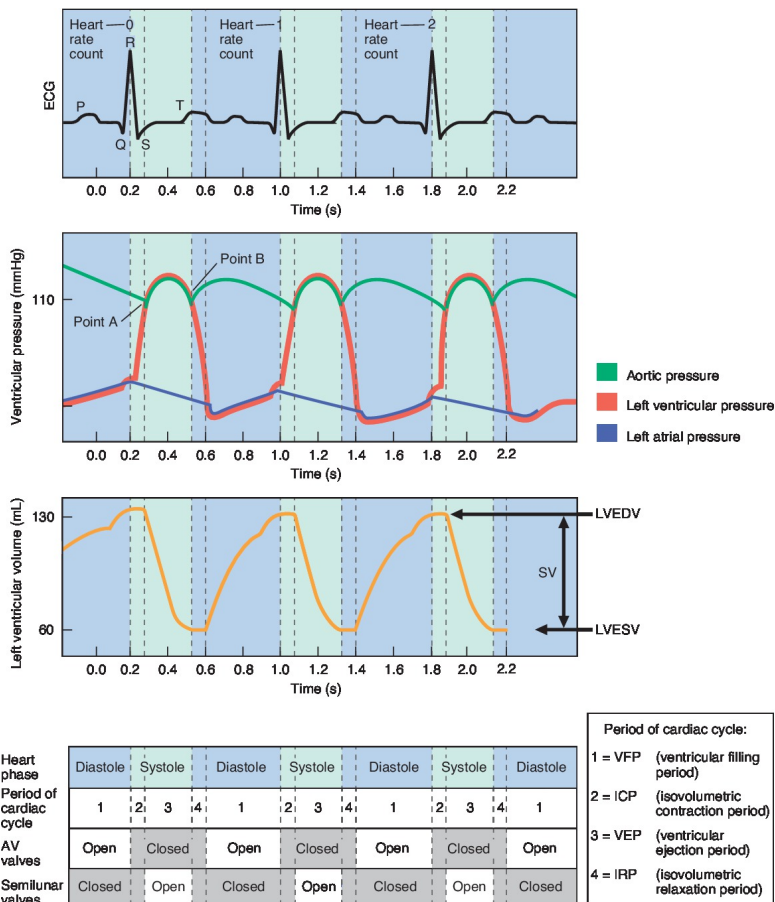


Figure 11.6 Graphic Summary of the Cardiac Cycle.

The periods of the cardiac cycle are labeled as 1, VFP; 2, ICP; 3, VEP; and 4, IRP.

Figure 11.6 begins arbitrarily during the VFP of ventricular diastole (also shown in **Figure 11.5A**). The AV valves are open, allowing blood to flow from the atria into the ventricles; therefore, ventricular volume is increasing. As the atria contract, more blood is forced into the ventricles, causing a small increase in ventricular volume and ventricular pressure.

Following the QRS complex, there is an immediate and dramatic increase in ventricular pressure as the myocardium contracts. Note, however, that the ventricular volume does not

immediately change. This is the ICP. Locate Point A on the graph of ventricular pressure in **Figure 11.6**—this is the point where pressure in the ventricle exceeds pressure in the aorta, and the aortic semilunar valve is forced open. Follow the dashed line downward to the row for the semilunar valves in the chart at the bottom of the figure, and note that these valves are now opened. Also note that this dashed line coincides with the start of a rapid decrease in ventricular volume. Once the valves are open, blood is forced out of the ventricles; thus, blood volume in the ventricles decreases. This is the VEP.

When pressure in the ventricles falls below pressure in the aorta, the semilunar valves close. Refer to Point B on the pressure curve of **Figure 11.6** and again follow the dashed line downward, noting ventricular volume and valve position. Ventricular pressure is decreasing because the myocardium is relaxed (in diastole). Ventricular volume, however, remains constant, because all the valves are closed and no blood can enter or leave the ventricles. This is known as the IRP.

Following the T wave (ventricular repolarization), the ventricles relax and begin to fill with blood: The AV valves are open, and ventricular volume increases. This is the VFP. This cycle of diastole followed by systole followed by diastole continues with each beat of the heart.

Diastole provides time for the cardiac cells to relax and the ventricles to fill. The length of time spent in diastole thereby directly affects the amount of blood that will be present in the ventricles to be pumped during the subsequent systole. Furthermore, it is during diastole that the myocardium is supplied with blood.

Systole is the contraction period of the heart, first isometrically (ICP) and then dynamically (VEP). The volume of blood ejected from the ventricles directly affects the cardiovascular system's ability to meet the demands of the body. The volume of blood in the ventricles at the end of diastole is termed **end-diastolic volume (EDV)** and is labeled in **Figure 11.6**. Similarly, the volume of blood in the ventricles at the end of systole is termed **end-systolic volume (ESV)**. The amount of blood ejected from the ventricles with each beat is called **stroke volume (SV)**, which is equal to $EDV - ESV$. **Figure 11.6** depicts

the volume in the left ventricle (LV), but because both sides of the heart must pump the same amount of blood over any significant period of time, the SV is typically the same for both sides of the heart.

End-Diastolic Volume (EDV) The volume of blood in the ventricle at the end of diastole.

End-Systolic Volume (ESV) The volume of blood in the ventricle at the end of systole.

Stroke Volume (SV) Amount of blood ejected from the ventricles with each beat of the heart.

Before going to the next section, study [Figures 11.5 and 11.6](#). Be sure you understand what is happening with the ECG, with ventricular, atrial, and aortic pressure, with ventricular volume, and with the heart valves at each period of the cardiac cycle. The [Check Your Comprehension 1 box](#) below will help ensure you understand the relationship among these variables. Now, find your radial pulse and begin counting: 0, 1, 2, 3, All the events described in this section occur every time you feel a pulse, which occurs once every 0.8 seconds when the heart rate is 75 b·min⁻¹. **Heart rate (HR)** is thus defined as the number of cardiac cycles per minute, expressed as beats per minute (b·min⁻¹).

Heart Rate (HR) The number of cardiac cycles per minute.

See animation, Cardiac Cycle, on Lippincott Connect



CHECK YOUR COMPREHENSION 1

What heart valves are open during the VFP? Where is the blood flowing from and to?

What heart valves are open during the ICP? Where is the blood flowing from and to?

What heart valves are open during the VEP? Where is the blood flowing from and to?

What heart valves are open during the IRP? Where is the blood flowing from and to?

What produces the force to eject blood from the ventricles during VEP? Why does blood not leave the ventricles as soon as contraction of the ventricles begins?

Check your answer in Appendix C.

Ventricular Pressure-Volume Loop

The relationship between left ventricular pressure and left ventricular volume throughout a cardiac cycle can be shown graphically with the pressure-volume loop (**Figure 11.7**). The pressure-volume loop integrates information that was presented in **Figures 11.5 and 11.6** and allows for the visualization of the amount of work done by the heart. The volume of blood in the ventricles is presented on the x-axis, and the pressure in the ventricle is shown on the y-axis. The four periods of the cardiac cycle are represented by the four sides of the closed pressure-volume loop. The VFP is seen along the bottom of the loop with volume increasing from approximately 60 mL to about 130 mL as blood enters the ventricle through the open A-V valve. During this period, volume increases but pressure does not change much (except for a small increase toward the end of the period reflecting the increase following atrial contraction). The ICP is seen along the right-hand side of the loop. During this period, pressure increases due to contraction of the ventricle but volume does not change because the valves are closed. The VEP is shown along the top of the loop. During this period, pressure continues to increase and then begins to decrease as contraction continues and then ends, and blood volume decreases as blood is ejected from the ventricle through the open aortic valve. The IRP is shown along the left-hand side of the loop. During this period, the pressure decreases as the ventricles relax but volume remains

the same because the valves are closed.

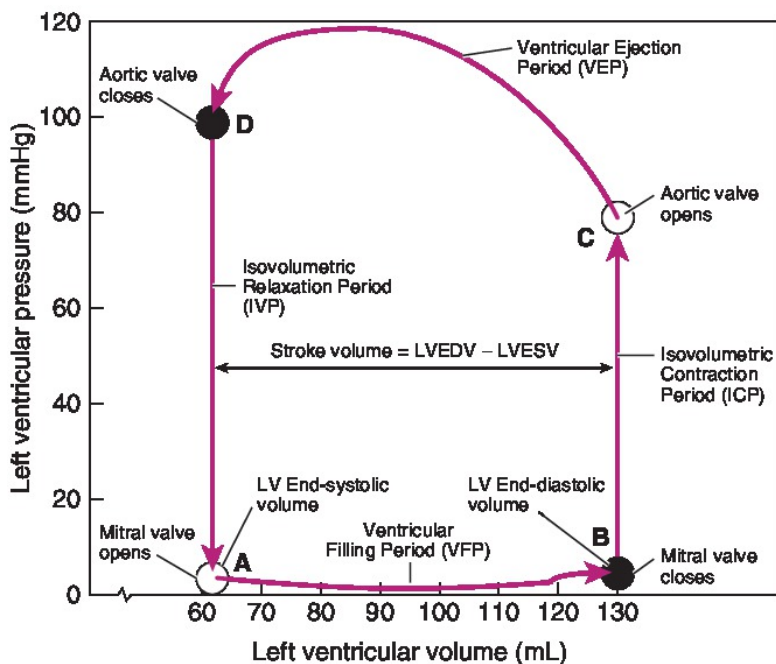


Figure 11.7 Left Ventricular Volume-Pressure Curve.

The pressure-volume loop has the advantage of providing a visual display of the work of the heart. The mechanical energy expended by the myocardium of the heart can be quantified as the area inside the pressure-volume loop. Thus, anything that increases stroke volume (the EDV-ESV) or ventricular pressure, such as exercise, will also increase the work of the myocardium and will be reflected in a larger area inside the pressure-volume loop.

Stroke Volume

As mentioned previously, stroke volume (SV) is the volume of blood ejected from the ventricles with each beat, expressed as milliliters per beat ($\text{mL}\cdot\text{b}^{-1}$) or simply in milliliters (mL). The amount of blood ejected from the heart is determined by three primary factors:

1. Preload—the volume of blood returned to the heart
2. Contractility—the force of myocardial contraction
3. Afterload—the resistance presented to the contracting ventricle

The volume of blood returned to the heart is called **preload** and is critical to the stroke volume because the heart cannot eject blood that is not there. Under resting conditions, the heart ejects approximately 50–60% of the blood that is returned; this is known as the **ejection fraction (EF)**. **Figures 11.6 and 11.7** both depict changes in the volume of blood in the ventricle throughout the cardiac cycle. In these figures, the EDV is approximately 130 mL of blood, which is typical for an adult male under resting conditions. Following systole, the ESV is approximately 60 mL of blood. Stroke volume can then be calculated as

Preload Volume of blood returned to the heart.

Ejection Fraction (EF) The percentage of EDV that is ejected from the heart.

$$\text{stroke volume (mL)} = \text{end-diastolic volume (mL)} - \text{end-systolic volume (mL)}$$

or

$$SV = EDV - ESV$$

$$11.1 \quad 70 \text{ mL} = 130 \text{ mL} - 60 \text{ mL}$$

Thus, 70 mL of blood was ejected from each ventricle during this contraction of the heart.

The ejection fraction (EF) can now be calculated from the information as follows:

ejection fraction (%) = stroke volume (mL) ÷
end-diastolic volume (mL) × 100

or

11.2
$$EF = \frac{SV}{EDV} \times 100$$

Example

Calculate the ejection fraction for the previous example.

$$EF = \left[\frac{70 \text{ mL}}{130 \text{ mL}} \right] \times 100 = 54\%$$

Under normal conditions, as the volume of blood returned to the heart (EDV) increases, SV increases. This relationship is known as the Frank-Starling law of the heart (**Figure 11.8**). Increasing EDV, to a point, increases stroke volume because the increased volume of blood in the ventricles provides a preload that stretches the myocardium and enhances the force of contraction by optimizing the overlap of actin and myosin filaments ([Levick, 2003](#)).

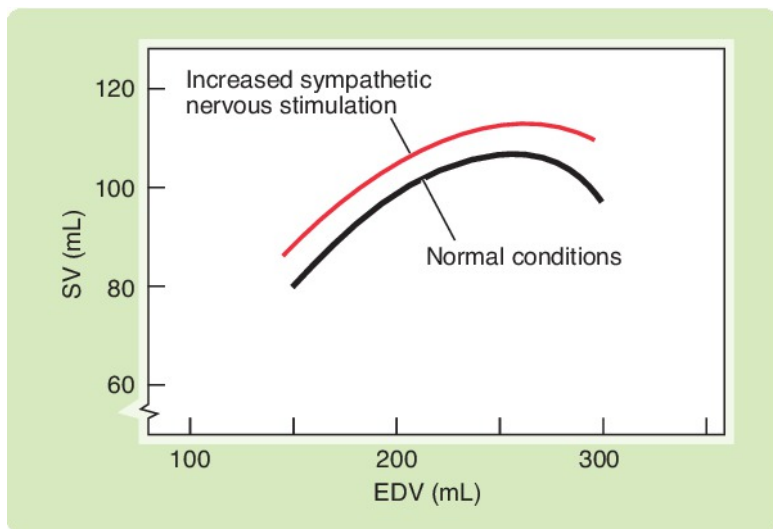


Figure 11.8 Frank-Starling Law of the Heart.

Contractility, the force of contraction of the myocardium, is determined primarily by neural innervation. Sympathetic nervous stimulation causes an increase in the contractility of the myocardium independent of the volume of blood returned to the heart. Circulating catecholamines (hormones) also reinforce the increased contractility caused by sympathetic nerve stimulation. During exercise, the Frank-Starling mechanism (increases in preload) and increased contractility (due to sympathetic nervous stimulation) function together to enhance stroke volume. **Figure 11.8** (upper curve) shows how sympathetic nervous stimulation can increase stroke volume at any given EDV. Answer the [Check Your Comprehension 2](#) box to ensure your understanding of this figure.

Contractility The force of contraction of the heart.

CHECK YOUR COMPREHENSION 2

Using **Figure 11.8**, answer the following questions under normal conditions and increased sympathetic nervous

stimulation:

1. What is the SV associated with an EDV of 150 mL? Of 200 mL? Of 250 mL? Of 300 mL?
2. What is the EF for each SV?

Check your answer in Appendix C.

The **afterload**, or the resistance presented to the contracting ventricle, is determined primarily by the blood pressure in the aorta. As blood pressure increases, opposition to the outward flow of blood increases, and less blood is ejected from the ventricles for any given force of contraction—that is, stroke volume decreases as afterload increases. The stroke volume decreases in this way because the increased pressure in the aorta causes the semilunar valves to remain closed longer and to close sooner. The valve is thus open for less time, thereby causing a decrease in ejection time and a subsequent decrease in stroke volume. **Table 11.1** presents typical values for stroke volume at rest in healthy individuals of various ages.

Afterload Resistance presented to the contracting ventricle.

Cardiac Output

Cardiac output (\dot{Q}) is the amount of blood pumped per unit of time (indicated by the dot over the Q), normally reported in liters per minute. It represents the total blood flow through the entire cardiovascular system and reflects the body's ability to meet changing metabolic needs during rest and exercise. Cardiac output is calculated as follows:

Cardiac Output (\dot{Q}) The amount of blood pumped per unit of time, in liters per minute.

$$\text{cardiac output (mL} \cdot \text{min}^{-1}) = \text{stroke volume (mL} \cdot \text{b}^{-1}) \times \text{heart rate (b} \cdot \text{min}^{-1})$$

or

$$11.3 \quad \dot{Q} = SV \times HR$$

At rest, cardiac output for an adult male is approximately 5 L·min⁻¹. It is interesting to note that resting blood volume, in average-sized adult males, is also approximately 5 L. Thus, at rest, the entire volume of blood is circulated through the body each minute. **Table 11.1** presents typical values for cardiac output at rest in healthy individuals of various ages.

Example

Calculate \dot{Q} for an individual with an HR of 64 b·min⁻¹ and an SV of 100 mL of blood.

$$\begin{aligned} \dot{Q} &= SV \times HR = (100 \text{ mL} \cdot \text{b}^{-1}) \times (64 \text{ b} \cdot \text{min}^{-1}) \\ &= 6,400 \text{ mL} \cdot \text{min}^{-1} \end{aligned}$$

Because \dot{Q} is usually reported in liters, this value is divided by 1,000 and is reported as 6.4 L·min⁻¹. Note that although SV is usually expressed in milliliters (mL), it is actually measured in milliliters per beat (mL·b⁻¹) since by definition SV must be per beat.

The **Check Your Comprehension 3** box asks you to do some calculations to check your understanding of this formula.

CHECK YOUR COMPREHENSION 3

Using your knowledge of the components of cardiac output, perform the necessary calculations to determine the missing values.

| | HR ($\text{b}\cdot\text{min}^{-1}$) | SV ($\text{mL}\cdot\text{b}^{-1}$) | \dot{Q} ($\text{L}\cdot\text{min}^{-1}$) |
|-------|---------------------------------------|--------------------------------------|--|
| Mike | 80 | 90 | |
| Keiko | 60 | 120 | |
| Kirk | 122 | 146.5 | |
| Don | 72 | | 6.34 |
| Nora | | 98 | 5.68 |

Check your answer in Appendix C.

Coronary Circulation

The energy necessary for cardiac function is supplied through aerobic metabolism. Arterial coronary circulation supplies oxygenated blood to the myocardium through two major arteries, the right coronary artery and the left coronary artery (**Figure 11.9**). Both arteries originate at the root of the aorta. The left coronary artery divides into the left circumflex and anterior descending arteries. The right coronary artery divides into the marginal artery and the posterior interventricular artery. The myocardium is supplied with a dense distribution of arterioles and capillaries, approximately 3,000–4,000 capillaries per square millimeter of cardiac muscle (Rowell, 1986). The venous blood from the coronary circulation is returned to the right atrium via the coronary sinus.

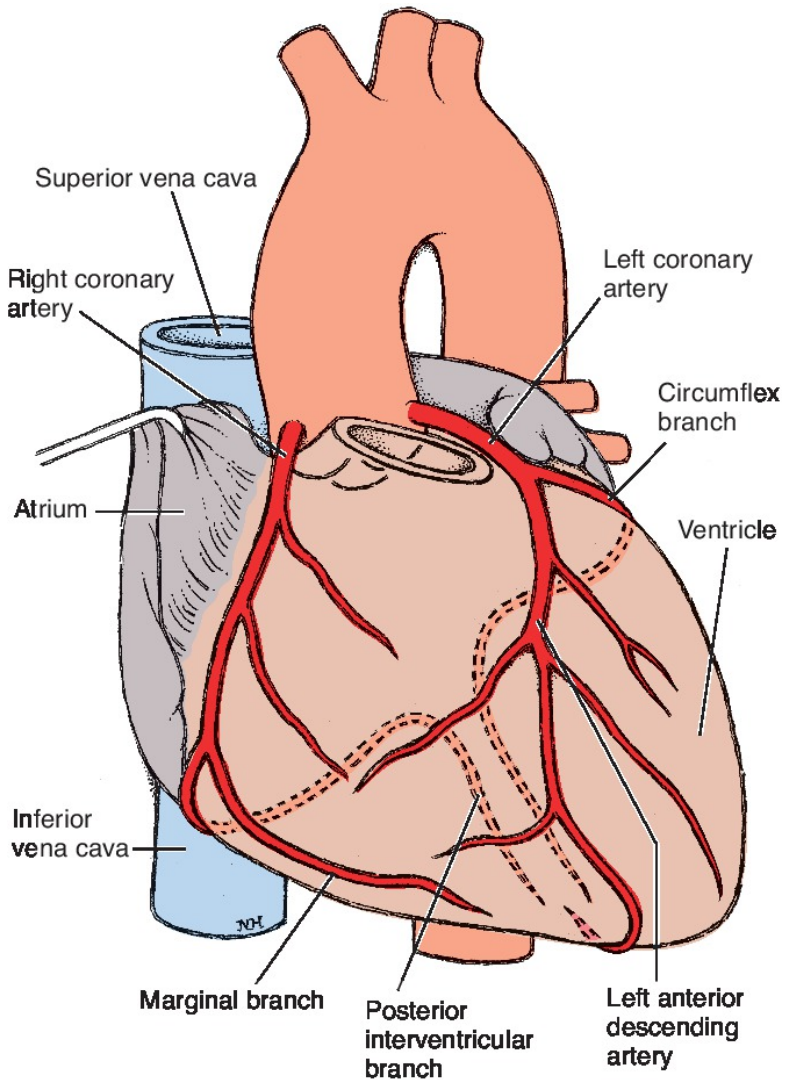


Figure 11.9 Coronary Circulation.

Coronary blood flow is affected greatly by the phase of the cardiac cycle. Because of the high intramyocardial pressure during systole, the coronary arteries are compressed, and blood flow to the myocardium is decreased. Thus, the myocardium receives the largest portion of its blood flow during diastole ([Hall and Hall, 2021](#)). The myocardial blood flow required to provide

necessary oxygen at rest is about $250 \text{ mL}\cdot\text{min}^{-1}$, which represents approximately 4% of the normal resting cardiac output (Rowell, 1986). The coronary circulation very effectively extracts oxygen as the blood flows through the capillary beds. Under resting conditions, 60–70% of the available oxygen is extracted.

Myocardial Oxygen Consumption

Because the work of the myocardium is increased during exercise (reflected in an increased stroke volume and increased ventricular pressure), **myocardial oxygen consumption**—the amount of oxygen used by the heart muscle to produce energy for contraction—increases during exercise. Myocardial oxygen consumption reflects the work of the heart and is determined by

oxygen extraction ($a-vO_2\text{diff}$) and blood flow (\dot{Q}). As mentioned previously, oxygen extraction of the coronary circulation is nearly optimal at rest (60–70%) and increases little if at all during exercise. Thus, the increased myocardial oxygen consumption during exercise occurs almost entirely by increased blood flow (\dot{Q}) to the myocardium.

Myocardial Oxygen Consumption The amount of oxygen used by the heart muscle to produce energy for contraction.

The coronary blood flow must be regulated to meet the demands of the myocardium for oxygen. In addition to a higher heart rate, blood flow is increased by two mechanisms:

1. The greater force of myocardial contraction that results from exercise causes more blood to be forced into the coronary circulation.
2. By-products of cellular work cause vasodilation of the arterioles that supply the myocardium. Thus, as the heart works harder and produces more by-products, the arterioles dilate, which decreases resistance and effectively increases blood flow.

Myocardial oxygen consumption increases as heart rate

increases. Because heart rate increases with the intensity of exercise, so also does myocardial oxygen consumption (Kitamura et al., 1972). Thus, oxygen consumption also increases as myocardial contractility increases. Myocardial oxygen consumption can be estimated from the **rate-pressure product (RPP)**, which is the product of heart rate (HR) and systolic blood pressure (SBP):

Rate-Pressure Product (RPP) An estimate of the myocardial oxygen consumption, calculated as the product of heart (HR) and systolic blood pressure (SBP).

rate-pressure product (units) = [systolic blood pressure (mmHg) × heart rate (b · min⁻¹)] ÷ 100
or

$$11.4 \quad RPP = \frac{SBP \times HR}{100}$$

RPP provides a good estimate of myocardial oxygen consumption under a wide range of conditions, including dynamic and static exercise. RPP is a commonly used clinical measure, especially in cardiac rehabilitation, because it provides an estimate of myocardial work.

The Vascular System

The *vascular system* is composed of vessels that transport the blood throughout the body. Their size and structure vary throughout the vascular tree, with each portion of the vascular system having a specific structure and function related to the overall function of the cardiovascular system. Blood vessels, except for capillaries, have three layers; the *adventitia* (outer layer), *tunica media* (middle layer), and *tunica intima* (innermost layer) (**Figure 11.10**). The *adventitia* is composed of connective tissue and attaches the blood vessel to surrounding tissue. The *tunica media* contains smooth muscle, which is critical for controlling the vessel's diameter, and connective tissue, and gives the vessel elasticity and strength. The *intima* consists of a single

layer of endothelial cells, the endothelium, and a thin layer of connective tissue (basal lamina). The **endothelium** serves as the barrier between blood and underlying tissue and:

Endothelium Single layer of epithelial tissue.

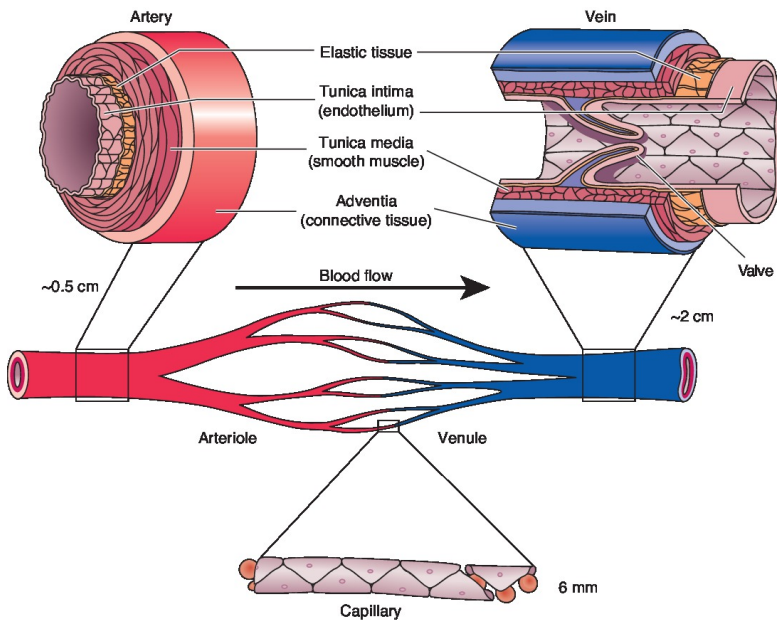


Figure 11.10 Three Layers of Blood Vessel Wall.

1. Plays a critical role in the movement of material out of the blood
2. Releases factors that help regulate the contraction of smooth muscle in the tunica media, thus helping to regulate blood flow
3. Helps prevent unnecessary clot formation
4. Interacts with immune cells in the inflammatory process

The vessels of the vascular system, along with various circulations, are illustrated in **Figure 11.11**.

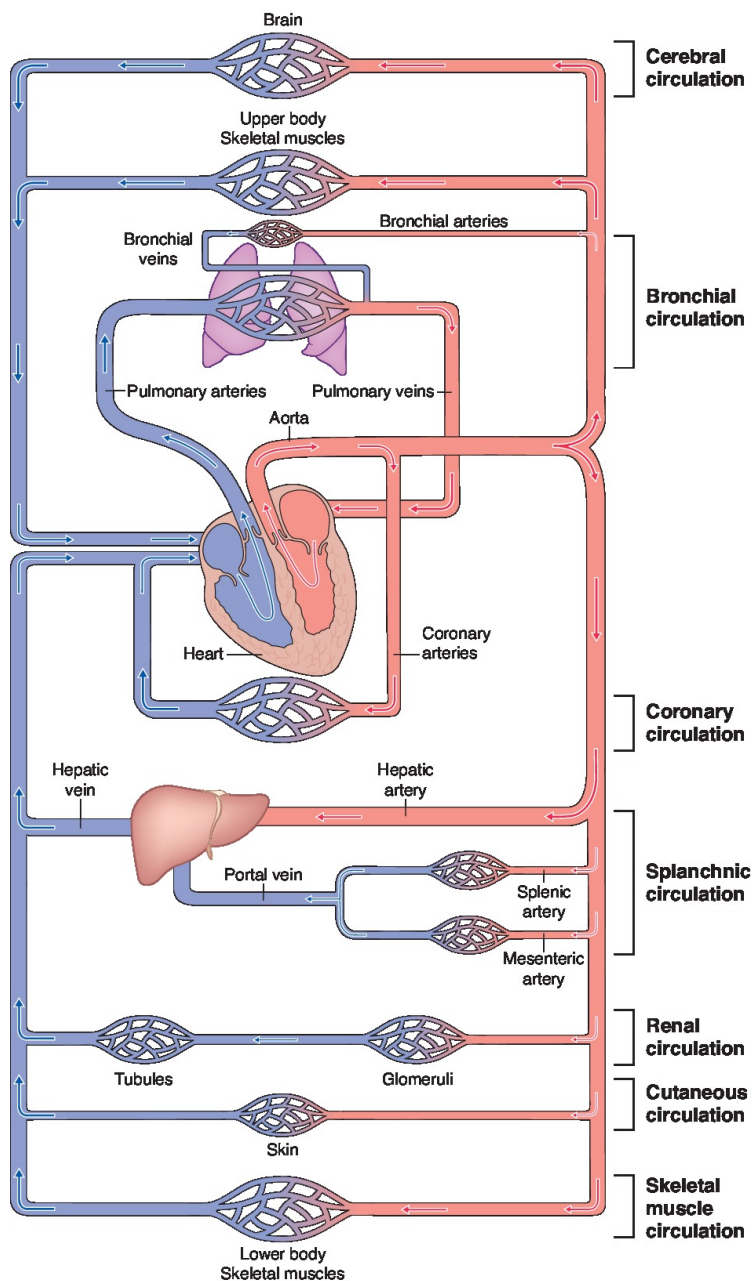


Figure 11.11 Schematic of the Vascular System.

Arteries

The arteries are thick-walled conduit vessels that carry blood from the heart to the body's organs (see **Figure 11.10**). Arteries contain a large amount of elastic connective tissue that allows them to distend when blood is ejected into them during systole and to recoil during diastole. Blood flow in the arteries is pulsatile owing to the pumping action of the heart (see **Figure 11.15C**).

As the left ventricle ejects blood into the aorta, the blood stretches the aorta's elastic walls. Blood pressure (BP) is the force exerted on the wall of the blood vessel by the blood. **Systolic blood pressure (SBP)** is the force exerted on the wall of the blood vessel by blood during systole. During relaxation of the heart (diastole), the arterial walls recoil, maintaining pressure on the blood still in the vessels. As a result, although the blood pressure drops during diastole, there is always some pressure in the arteries. Thus, **diastolic blood pressure (DBP)** is the force exerted on the wall of blood vessels by blood during diastole. **Mean arterial pressure (MAP)** is a weighted average of SBP and DBP, representing the mean driving force of blood throughout the arterial system. Typical resting blood pressure values for males and females at different ages are given in **Table 11.2**.

Systolic Blood Pressure (SBP) The force exerted on the wall of blood vessels by the blood as a result of contraction of the heart (systole).

Diastolic Blood Pressure (DBP) The force exerted on the wall of blood vessels by blood during relaxation of the heart (diastole).

Mean Arterial Pressure (MAP) The weighted average of SBP and DBP, representing the mean driving force of blood throughout the arterial system.

TABLE 11.2 Typical Resting Blood Pressure Values for Males and Females of Various Ages

| Blood Pressure Value | Age of Males (y) | | | Age of Females (y) | | |
|---------------------------------------|------------------|-------|-------|--------------------|-------|-------|
| | 10–15 | 20–30 | 50–60 | 10–15 | 20–30 | 50–60 |
| Systolic blood pressure (SBP) (mmHg) | 100 | 120 | 134 | 84 | 120 | 130 |
| Diastolic blood pressure (DBP) (mmHg) | 60 | 80 | 84 | 40 | 74 | 84 |
| Mean arterial pressure (MAP) (mmHg) | 73 | 93 | 100 | 55 | 88 | 99 |

Sources: Fleg et al. (1995); Ogawa et al. (1992); Spina et al. (1993a, 1993b).

Arterioles

Arterioles, also called **resistance vessels**, are smaller than arteries and are the major site of resistance in the vascular system. Because of their ability to change diameter, these vessels play a critical role in determining the *distribution* of blood to various organs. This is important since we have a limited amount of blood. Blood flow to working tissues needs to be increased while the nonactive tissues may receive less blood flow.

Resistance Vessels Another name for the arterioles because this is the site of greatest resistance to blood flow in the vascular system.

Because of increased resistance in the arterioles, the pulsatile arterial blood flow becomes continuous before it reaches the capillaries (see **Figure 11.15C**). Arterioles absorb the pulsatile force of blood flow because they contain a large amount of elastic tissue. Imagine bouncing a basketball on a gymnasium floor and on a wrestling mat. The ball rebounds from the gym floor at an angle and height proportional to the force imparted by your muscle action. However, the same force will not produce much (if any) rebound from the wrestling mat. The elastic tissue in the walls of the arteries absorbs the energy from the pulsatile blood flow in a similar way that the mat absorbs energy from the basketball. In an individual with reduced elasticity, or arterial stiffening, the arteries, like the gym floor, are not able to distend as readily, resulting in elevated blood pressure.

The smooth muscle surrounding arterioles is able to contract

and relax. Contraction of the smooth muscle around an arteriole results in *vasoconstriction*, a decrease in vessel diameter and therefore a decrease in blood flow to a given region. Relaxation of the smooth muscle results in *vasodilation*, an increased vessel diameter, and therefore an increase in blood flow to a region. The vasoconstriction and vasodilation of the smooth muscles surrounding the arterioles are primarily responsible for determining blood flow distribution to various organs.

The degree to which an arteriole vasodilates or vasoconstricts depends on the balance of *extrinsic* (originating outside the part on which it acts) and *intrinsic* (originating within the part on which it acts) mechanisms. Extrinsic mechanisms are geared toward maintaining mean arterial pressure and include nervous stimulation and circulating hormones. Smooth muscle surrounding terminal arteries and arterioles is innervated by sympathetic neurons. Sympathetic stimulation causes most arterioles to vasoconstrict. However, during exercise, when the sympathetic nervous system is clearly activated, arterioles to the working muscles dilate in order to supply the working muscle with increased blood flow—this occurs primarily because local factors dominate during exercise.

Intrinsic (local) mechanisms that control arteriole diameter include *myogenic* (originating within the muscle) mechanisms, *metabolic* factors, and *shear stress* in the blood vessel that causes the release of local vasoactive substances. Myogenic control is accomplished by mechanisms that cause the vessels to dilate in response to decreased stretch (decreased flow) and vasoconstrict in response to increased stretch (increased flow). This reflex action helps to ensure that changes in blood pressure do not lead to dramatic changes in blood flow to a given vascular bed. The metabolic control of vascular diameter plays a critical role in determining the degree of smooth muscle contraction and hence local blood flow. When tissue is metabolically active, such as skeletal muscle during exercise, it produces metabolic by-products that act locally to cause vasodilation, thereby increasing blood flow to the metabolically active area. Thus, contracting skeletal muscles act locally (intrinsically) on the smooth muscle surrounding the arterioles to increase blood flow in that region during exercise. Shear stress on the wall of the blood vessels causes the endothelium to release vasoactive substances that

regulate vascular tone, and hence vessel diameter. An increase in shear stress during exercise causes the release of nitric oxide (NO) from the endothelium, which in turn causes smooth muscle surrounding the vessel to relax, thus bringing about vasodilation. During exercise, the local vasodilatory effects have a greater impact on vessel diameter in arterioles supplying the skeletal muscle than the sympathetic vasoconstrictor effects, leading to vasodilation in the skeletal muscle. On the other hand, arterioles supplying nonworking muscle and other organs (stomach, kidneys) constrict, resulting in decreased blood flow to these areas during exercise due to sympathetic nerve stimulation. The decrease in blood flow to nonworking areas while increasing blood flow to working muscles reflects a delicate interplay between intrinsic and extrinsic mechanisms to distribute cardiac output (total blood flow) effectively. The balance of vasoconstriction/vasodilatation also helps ensure that blood pressure does not change dramatically.

Capillaries

The *capillaries* perform the ultimate function of the cardiovascular system: transferring gases and nutrients between the blood and tissues. Some exchange of gases occurs in the smallest vessels on both sides of the capillaries (collectively termed the **exchange vessels**), but most of the gas exchange occurs across the capillary wall. The walls of the capillaries are very thin, essentially composed of a single layer of endothelial cells (**Figure 11.10**). Capillaries have a very small diameter, such that red blood cells often must pass through in single file.

Exchange Vessels Another name for capillaries because this is the site of gas and nutrient exchange between the blood and tissues.

Blood flow through capillaries also depends on the other vessels that make up the **microcirculation**. As shown in **Figure 11.12**, the microcirculation includes several vessels: arterioles, venules, arteriovenous anastomoses, metarterioles, and true capillaries. *Anastomoses* are wide, connecting channels that act as

shunts between arterioles and venules. These vessels are not common in most tissue but are abundant in the skin and play an important role in thermoregulation (Levick, 2003). When anastomoses are open, large volumes of blood can be directed to blood vessels close to the surface of the skin, facilitating heat dissipation. A *metarteriole* is a short vessel that connects an arteriole with a venule, creating a direct route through the capillary bed. The metarteriole gives rise to the capillaries. *True capillaries* vary in number depending on the capillary bed. Smooth muscles around these vessels that relax or constrict in response to local chemical conditions control blood flow through the capillaries. Thus, a capillary bed can be perfused with blood or be almost entirely bypassed, depending on the needs of the tissue it supplies.

Microcirculation Smallest vessels of the vascular system, including the arterioles, venules, arteriovenous anastomoses, metarterioles, and true capillaries.

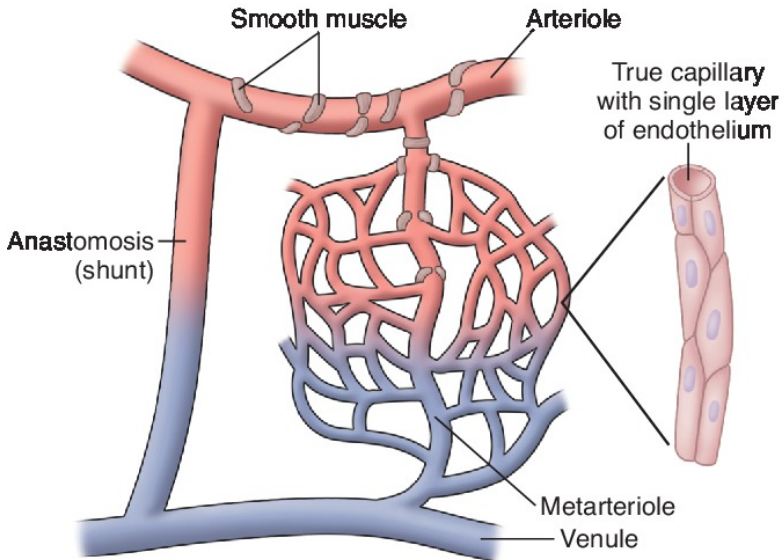


Figure 11.12 Anatomy of the Microcirculation.

GAS EXCHANGE The exchange of gases and nutrients in the capillaries depends on *diffusion*. For a substance to diffuse from a capillary into a cell, it must cross two membranes: the capillary wall (composed primarily of endothelium) and the cell membrane. Substances pass from the capillary to the interstitial space by the process of diffusion. Movement from the interstitial space into the cell may also occur by diffusion or may require carrier-mediated transport. The movement of gases and nutrients into and out of the capillaries depends on the concentration gradient or pressure gradient of the substance or gas that is diffusing.

As discussed in [Chapter 9](#), oxygen and carbon dioxide diffuse down pressure gradients. Oxygen diffuses down its pressure gradient from systemic capillaries into muscle cells. Therefore, there is less oxygen in the veins draining skeletal muscles than in the arteries supplying them. The difference in the oxygen content of the arteries and veins is termed the *a-vO₂ difference*, which reflects the oxygen taken up by the skeletal muscles.

MOVEMENT OF FLUIDS Fluids also pass through the capillary membrane. The movement of fluids is determined by two opposing forces: hydrostatic pressure and osmotic pressure. *Hydrostatic pressure*, created by blood pressure, acts to “push” fluid out of the capillaries. *Osmotic pressure*, caused by the larger concentration of proteins in the capillaries, acts to “pull” water into the capillaries. The net result of these opposing forces is the loss of approximately 3 L of fluid a day from the plasma into interstitial spaces ([Marieb and Hoehn, 2018](#)). This fluid returns to the blood via the lymphatic system. Any change in hydrostatic pressure or osmotic pressure of the blood will alter the fluid exchange between the blood and the interstitial fluid.

Venules

The venules are small vessels on the venous side of the vascular system. These vessels contain some smooth muscle, which can influence capillary pressure. The venules and capillaries constitute the microcirculation where gas and nutrient exchange occurs. Venules empty into veins.

Veins

Veins, also called **capacitance vessels**, are low-resistance conduits that return blood to the heart (**Figure 11.10**). They contain smooth muscle innervated by the sympathetic nervous system, which can change their diameter. Contraction of smooth muscle around the veins is known as *venoconstriction*; relaxation of the veins is known as *venodilation*. Because veins can expand (distensibility), they can pool large volumes of blood—up to 60% of the total blood volume at rest—and therefore are sometimes referred to as a blood reservoir. The amount of blood in all the veins varies with posture and activity. If blood accumulates in the veins and is not returned to the heart, ventricular end-diastolic volume decreases, and hence stroke volume decreases. Conversely, venoconstriction can significantly increase ventricular end-diastolic volume and thereby lead to an increase in stroke volume, according to the Frank-Starling law of the heart.

Capacitance Vessels Another name for veins because of their distensibility, which enables them to pool large volumes of blood and become reservoirs for blood.

The skeletal muscle pump and the respiratory pump help increase venous return by “massaging” blood back toward the heart. The one-way valves in the veins also help regulate venous pressure and are particularly helpful in counteracting the effects of gravity that oppose blood flow back to the heart because they prevent the backward flow of blood. Additionally, the increased sympathetic nervous activity during exercise helps to increase venous return via venoconstriction.

Blood

Blood is the fluid that circulates through the heart and the vasculature to transport nutrients and gases. Blood contains living blood cells suspended in a nonliving fluid matrix called plasma. **Figure 11.13** depicts blood that has been centrifuged to separate the cells and the plasma. Blood cells are classified as erythrocytes

(red blood cells, RBC) or leukocytes (white blood cells, WBC). Blood cells account for 38–45% of the total blood volume in adult females and 43–48% of the total blood volume in adult males. The ratio of blood cells to total blood volume is known as **hematocrit** and is usually expressed as a percentage.

Hematocrit The ratio of blood cells to total blood volume, expressed as a percentage.

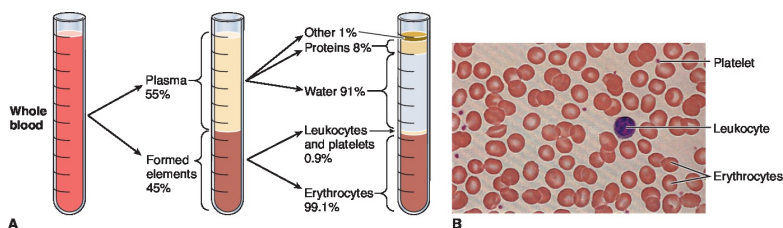


Figure 11.13 Components of Blood.

A. Whole blood is composed of formed elements and plasma, which can be separated by “spinning the blood” in a centrifuge. **B.** Formed elements (platelets, leukocytes, and erythrocytes) as seen through a microscope (400 \times).

As discussed in the respiratory section, the RBCs transport oxygen from the lungs to body cells by binding oxygen to hemoglobin. Leukocytes are less numerous than erythrocytes, accounting for about 1% of total blood volume. Despite their seemingly small number, leukocytes are essential to the body’s defense against disease and play a critical role in inflammation.

Plasma accounts for approximately 55% of the volume of blood. It is composed primarily of water, which accounts for approximately 90% of its volume. It also contains over 100 dissolved solutes, including proteins, nutrients, electrolytes, and respiratory gases. The composition of plasma varies greatly, depending on the needs of the body. Plasma also plays an important role in thermoregulation by helping to distribute heat throughout the body.

Blood Donation and Exercise

When one donates blood, approximately one pint of blood (about 450–500 mL) is typically taken from the body. Because the total blood volume is approximately 5,000 mL (5 L), the donation results in roughly a 10% reduction in blood volume or a greater percentage in small individuals with less blood (typically women). The blood plasma volume is reestablished in approximately 24 hours, and red blood cells are replaced in about 6 weeks. Because of this reduction in plasma volume, it seems prudent for endurance athletes to avoid donating blood during the competition phases of the training cycle. Strenuous activities should be avoided for 24 hours after giving blood to allow the body to replace the majority of the lost fluids. Plenty of water should be ingested following blood donation.

Training intensity may need to be slightly reduced pending complete RBC replacement for fitness participants or athletes who do donate blood.



Hormonal Control of Blood Volume

As is discussed in the accompanying Focus on Application: Clinically Relevant Box, blood volume is decreased by blood donation. Decreased blood volume may also result from profuse sweating and/or dehydration. Blood volume varies considerably among individuals and is affected by fitness status. Healthy adult males have an average blood volume of approximately 75 mL of blood per kg of body weight, or a total of approximately 5–6 L of blood. Healthy adult females have approximately 65 mL of blood per kg of body weight, which equals 4–4.5 L of blood for the average-sized woman. Children typically have about 60 mL of blood per kg of body weight, with total volume varying depending on body size.

Blood volume plays an important role in maintaining stroke volume, cardiac output, and blood pressure. Under normal conditions, blood volume is maintained within physiological

limits by overlapping and coordinating homeostatic mechanisms involving the endocrine system and urinary system. The major hormones involved in maintaining blood volume are antidiuretic hormone (ADH), released from the posterior pituitary gland, and aldosterone, released from the adrenal cortex. **Figure 11.14** outlines the hormonal mechanisms that respond to a reduction in blood volume.

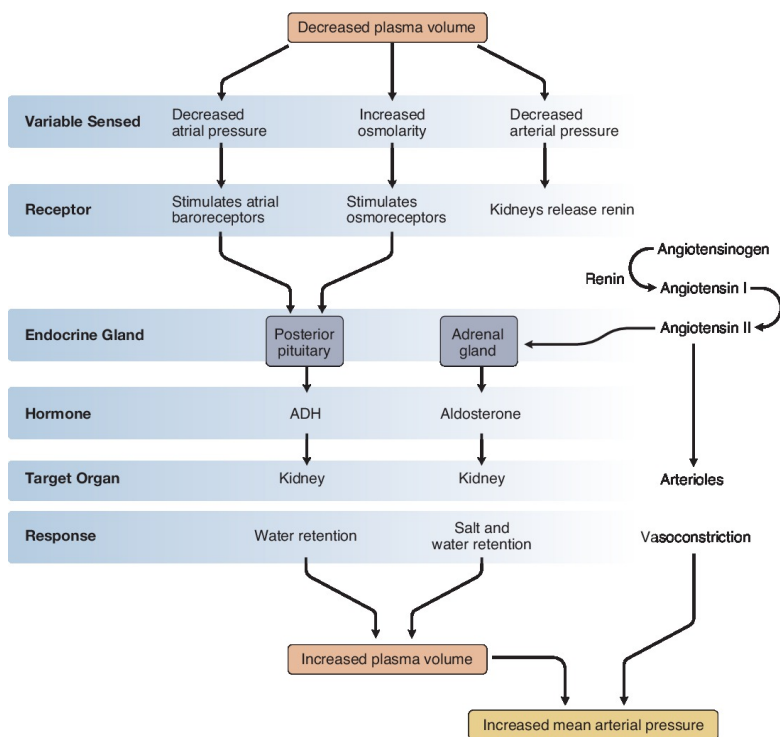


Figure 11.14 Hormonal Control of Blood Volume.

Plasma volume reduction causes a decrease in atrial and arterial pressure. The decrease in pressure is sensed by atrial baroreceptors (*baro* means “pressure”) and arterial receptors in the kidneys. Atrial baroreceptor activation leads to the release of ADH from the posterior pituitary gland, which causes the tubules of the kidneys to reabsorb water, thus increasing plasma volume.

A reduction in blood volume is also associated with an increase in plasma osmolarity (solute concentration). For

example, with profuse sweating, more water than solutes is lost; thus, the osmolarity of the blood increases. An increase in osmolarity of the blood stimulates osmoreceptors in the hypothalamus, which signals the posterior pituitary gland to release ADH. ADH causes the kidneys to retain water, thus leading to an increase in blood volume.

Simultaneously, the receptors in the kidneys respond to decreased arterial pressure by releasing the enzyme renin. Renin is necessary for the conversion of angiotensinogen to angiotensin I, which is then converted to angiotensin II. Angiotensin II signals the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to retain salt and water. Angiotensin II also has a vasoconstrictor effect on arterioles, thus helping to increase blood pressure.

Cardiovascular Dynamics

The different components of the cardiovascular system function together to meet the changing demands of the body. These components are highly integrated and interdependent. Although both the heart and the vasculature respond independently to various conditions, they are interrelated because the response of the heart affects the vessels and vice versa.

To differentiate the responses of the heart and the vessels, we commonly refer to central and peripheral cardiovascular responses. **Central cardiovascular responses** are those directly related to the heart: heart rate, stroke volume, cardiac output, etc. **Peripheral cardiovascular responses** are those occurring in vessels: vasodilation, vasoconstriction, venous return, etc.

Central Cardiovascular Responses Responses directly related to the heart.

Peripheral Cardiovascular Responses Responses directly related to the vessels.

Cardiac Output (Q.)

As described earlier, cardiac output is the amount of blood ejected from the ventricles each minute. This volume of blood changes constantly to meet the body's metabolic demands. The volume of blood flow can be described using the basic formula presented in [Chapter 9](#) to describe air flow ($F = \Delta P/R$). Applied to the cardiovascular system, this equation is

$$\text{cardiac output (L} \cdot \text{min}^{-1}\text{)} = \frac{\text{mean arterial blood pressure (mmHg)}}{\text{total peripheral resistance (mmHg} \cdot \text{mL}^{-1} \cdot \text{min}^{-1}\text{)}}$$

or

$$11.5 \quad \dot{Q} = \frac{\text{MAP}}{\text{TPR}}$$

In this formula, cardiac output represents the blood flow for the entire cardiovascular system, mean arterial pressure reflects the pressure gradient driving blood through the vascular system, and total peripheral resistance refers to the factors that oppose blood flow in the entire system. Technically, the equation should use the difference in pressure (ΔP) between mean arterial pressure and the pressure in the right atrium (where blood is flowing to). However, since pressure in the right atrium is so low (< 4 mmHg), it is considered negligible and is not usually given in the formula. Thus, mean arterial pressure is used alone in the equation.

Mean Arterial Pressure

Mean arterial pressure represents the driving force of blood through the vascular system. Adequate MAP is necessary to ensure adequate perfusion of all the vital organs of the body. On the other hand, excess blood pressure can damage the endothelial lining of the blood vessels. Thus, MAP is a physiological variable that is tightly controlled by homeostatic mechanisms. MAP can be expressed in relation to \dot{Q} and TPR by rearranging the equation above to become

mean arterial pressure (mmHg) = cardiac output
 $(\text{L} \cdot \text{min}^{-1}) \times \text{total peripheral resistance}$
 (TPR units)

$$11.6 \quad \text{MAP} = \dot{Q} \times \text{TPR}$$

Total Peripheral Resistance

Total peripheral resistance (TPR), or simply **resistance (R)**, results from factors that oppose blood flow. It is expressed in millimeters of mercury per milliliter per minute ($\text{mmHg} \cdot \text{mL}^{-1} \cdot \text{min}^{-1}$) or more simply as TPR units. Most of the resistance in the vascular tree results from the friction of blood against vessel walls, and it varies depending on the size of the vessel. The three primary factors that affect resistance and their mathematical relationship are described by Poiseuille's law:

Total Peripheral Resistance (TPR) or Resistance (R) The factors that oppose blood flow.

$$11.7 \quad \text{resistance} = \frac{\text{length} \times \text{viscosity}}{(\text{radius})^4}$$

Thus, the more viscous the blood is, the greater the resistance to flow. The longer the blood vessel, the greater the friction between the vessel walls and the blood. Under normal conditions, however, a vessel's length and blood viscosity do not change substantially. On the other hand, the vessel's radius can change considerably because of vasodilation or vasoconstriction. Furthermore, because the resistance is inversely related to the fourth power of the radius, a small change in vessel diameter can result in a large change in blood flow. Thus, vessel radius is by far the most important factor determining resistance to blood flow. Recall that vessel diameter is affected by local conditions (local control) and neural innervation (extrinsic control).

Total peripheral resistance (TPR) can be calculated by rearranging the formula $\dot{Q} = \text{MAP} \div \text{TPR}$ to solve for TPR,

provided that the flow rate (\dot{Q}) and blood pressure (MAP) are known. The formula for TPR becomes

total peripheral resistance (TPR units) = mean
arterial blood pressure (mmHg) \div cardiac output
($\text{L} \cdot \text{min}^{-1}$)

or

$$\text{TPR} = \frac{\text{MAP}}{\dot{Q}}$$

11.8

Example

Assume that normal resting blood pressure is 110/80 (MAP = 90 mmHg), normal cardiac output = $5.4 \text{ L} \cdot \text{min}^{-1}$, and central venous pressure = zero. Calculate TPR for the entire cardiovascular system.

The calculation is

$$\text{TPR} = \frac{\text{MAP}}{\dot{Q}} = \frac{90 \text{ mmHg}}{5.4 \text{ L} \cdot \text{min}^{-1}} = 16.67 \text{ (TPR units)}$$

In [Check Your Comprehension 4](#), calculate the TPR.

CHECK YOUR COMPREHENSION 4

Given the following information, calculate TPR.

$$\begin{aligned} \text{SBP} &= 150 \text{ mmHg}; & \text{DBP} &= 90 \text{ mmHg}; \\ \dot{Q} &= 5.1 \text{ L} \cdot \text{min}^{-1}. \end{aligned}$$

Check your answer in Appendix C.

Principles of Blood Flow

Figure 11.15 presents the relationships among the cross-sectional area of the blood vessels, blood pressure, and blood velocity throughout the vascular system. The diameter of the various vessels is shown in **Figure 11.15A**, and the total cross-sectional area of the various vessels is shown in **Figure 11.15B**. Thus, although a single capillary is incredibly small, approximately 6 mm, there are so many capillaries that the total cross-sectional area far exceeds that of the other vessels. **Figure 11.15B** also depicts the velocity of blood in the various vessels—that is, the speed at which blood flows. The velocity of a fluid in a closed system varies inversely with the total cross-sectional area at any given point. Therefore, the velocity of blood flow decreases dramatically in the capillaries. This decreased velocity allows adequate time for the exchange of respiratory gases and nutrients.

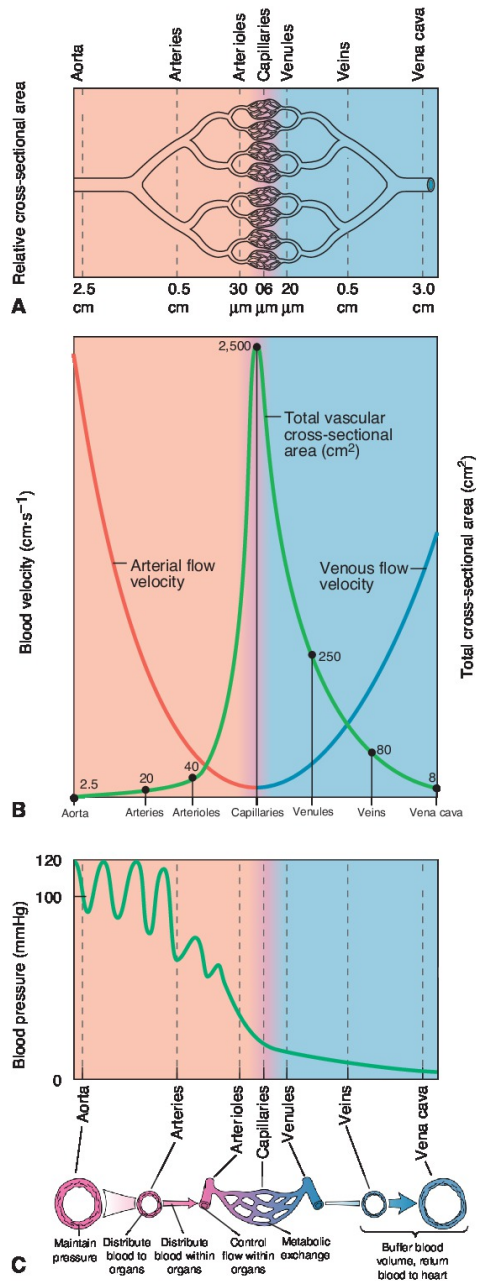


Figure 11.15 Relationships among Cross-Sectional Area (CSA) of Blood Vessels, Blood Pressure, and Resistance.

A. Despite the very small diameter of individual capillaries,

relative CSA of the capillaries is much higher than that of other vessels because they are so numerous. **B.** Blood velocity through the vascular system is inversely related to total CSA. **C.** Blood pressure is pulsatile in the aorta and major arteries but becomes continuous as blood travels through the arterioles. Blood pressure in the venous system is very low compared to the arterial system.

Figure 11.15C depicts the blood pressure throughout the vascular system. The driving force for the blood is the contraction of the myocardium. Blood flows because of a pressure gradient. Thus, blood flows through the vascular tree because pressure is highest in the aorta and major arteries and lowest in the great veins and right atrium of the heart. Pressure continues to decrease as the blood travels further from the heart, reaching a low of approximately 4 mmHg in the right atrium. In fact, one-way venous valves and muscle and respiratory pump activity are needed to help return blood to the heart.

Regulation of the Cardiovascular System

The cardiovascular system is regulated by interrelated and overlapping mechanisms, including mechanical events, neural control, and neurohormonal control. Mechanical events, such as muscle action, influence venous return and thereby help regulate stroke volume and cardiac output. This regulation is particularly important during exercise. Neural and neurohormonal mechanisms of cardiovascular control are more complex and are discussed in detail in the following sections.

Neural Control

Three cardiovascular centers are located within the medulla oblongata of the brainstem (**Figure 11.16**). These regulatory centers play an important role in controlling the output of the heart and the radius of the blood vessels. The cardioaccelerator and cardioinhibitor centers innervate the heart. As the names

imply, the *cardioacceleratory center* sends signals, via sympathetic accelerator nerves, that cause the heart rate to increase and the force of contraction to strengthen. The *cardioinhibitor center*, also called the vagal nucleus, sends signals via the vagus nerve that causes a decreased heart rate and force of contraction. The *vasomotor center* innervates the smooth muscles of the arterioles via sympathetic nerves. Activation of these sympathetic fibers generally causes vasoconstriction (sympathetic fibers to the skin are the only clear exception to this rule in humans).

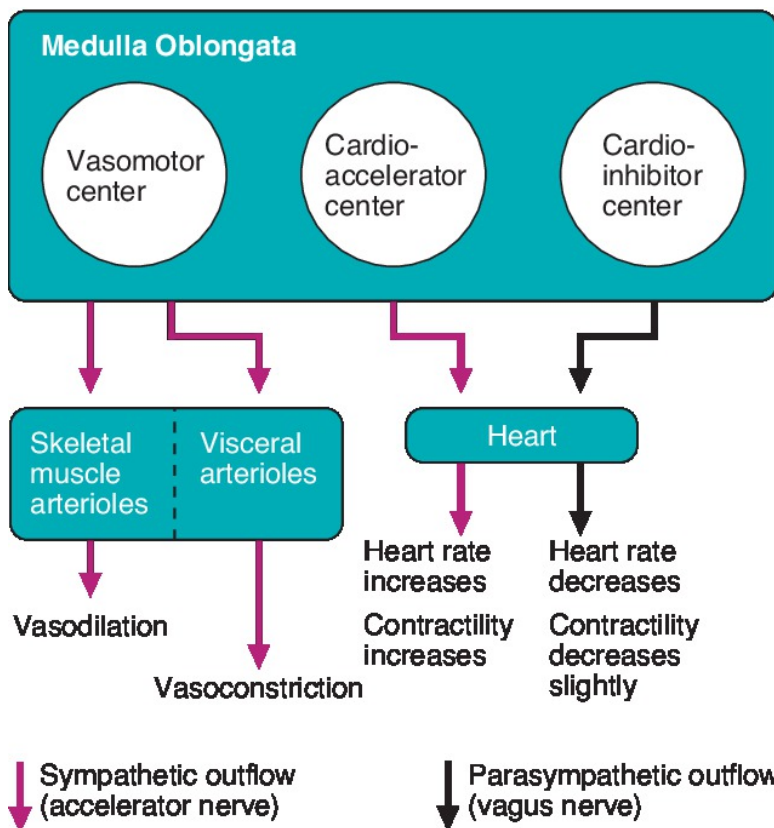


Figure 11.16 Neural Control of Cardiovascular Function.

In summary, activation of sympathetic nervous system leads to an increased heart rate, increased cardiac contractility, and vasoconstriction in most arterioles. During exercise, there is also

vasodilation in the arterioles of skeletal muscle, but the role of the autonomic nervous system in this response is not clear. What is clear is that metabolic (intrinsic) controls lead to a wide spread vasodilation in arterioles supplying skeletal muscle in response to increased metabolic activity during exercise. Activation of the parasympathetic nervous system leads to the opposite responses in each of the above.

See animation, Neural Control of CV System, on Lippincott



Anatomical Sensors and Factors Affecting Control of the Cardiovascular System

The cardiovascular centers—and therefore, the sympathetic and parasympathetic outflow from those centers—are influenced by several factors in a variety of circumstances, including exercise. **Figure 11.17** schematically presents the most important factors influencing the cardiovascular centers. These factors are described in detail in the following sections.

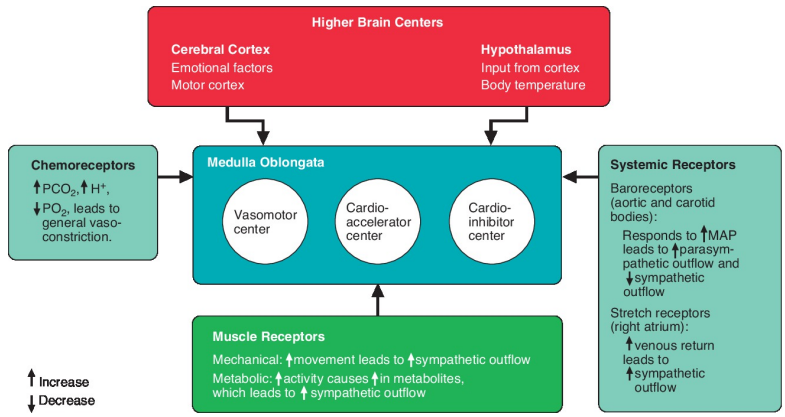


Figure 11.17 Factors Affecting Neural Control of Cardiovascular Function.

Higher Brain Centers

The cardiovascular medullary centers are influenced by several higher brain centers, including the cerebral cortex and the hypothalamus. Emotional influences arising from the cerebral cortex can affect cardiovascular function at rest. Input from the motor cortex, which is relayed through the hypothalamus, can influence cardiovascular function during exercise, leading to an increase in heart rate and vasodilation in active muscle. The influence of the cortex and hypothalamus on the cardiovascular centers during exercise is often termed “central command,” denoting that the signal to alter cardiovascular variables comes from the central nervous system.

Body temperature also affects the cardiovascular centers through the influence of the hypothalamus. An increased body temperature results in an increased heart rate, increased cardiac output, and vasodilation in the arterioles of the active muscles and skin.

Systemic Receptors

Systemic receptors are present in the great veins, the heart, and the arterial system. These receptors provide sensory information to the cardiovascular control centers that leads to reflex action.

BARORECEPTORS Baroreceptors (pressor sensors) are located in the aorta and carotid bodies. With an increase in mean arterial pressure, these receptors cause a reflex decrease in mean arterial pressure through a decreased heart rate (and thus decreased cardiac output). The decrease in heart rate is mediated through an increased parasympathetic outflow and a simultaneous decrease in sympathetic outflow to the heart. This reflex control of blood pressure is called the *baroreceptor reflex*.

Because this reflex functions to maintain mean arterial blood pressure, you may wonder how someone becomes hypertensive (high blood pressure) or why mean arterial blood pressure goes up during exercise. In someone with hypertension, the action of the baroreceptors is mediated by a set point. If something causes the resting blood pressure to be elevated (and no one knows precisely what causes this elevation), the baroreceptors fire for about 24 hours in an effort to bring down the mean arterial pressure. If this is unsuccessful, the baroreceptors appear to

simply reset at a level above the previous value. The baroreceptor reflex is also reset during exercise. The resetting of the baroreflex is in direct proportion to exercise intensity (Raven et al., 2006). The baroreceptor reflex is also very important in achieving recovery to baseline values after exercise.

STRETCH RECEPTORS Stretch receptors, located in the right atrium of the heart, are stimulated by an increase in venous return. The signal is transmitted to the cardiovascular centers in the medulla, where they cause an increase in sympathetic outflow and a decrease in parasympathetic outflow. This results in an increased heart rate and force of contraction, increasing cardiac output. This sequence is called the *Bainbridge reflex*.

Chemoreceptors

Chemoreceptors are located in the aortic and carotid arteries. They are sensitive to arterial blood PO₂, PCO₂, and H⁺. An increase in PCO₂ and H⁺ or a decrease in PO₂ causes a reflex vasoconstriction of arterioles.

Muscle Joint Receptors

Muscle receptors include mechanical (*mechanoreceptors*) and metabolic (*metaboreceptors*) receptors located in the joints and muscles. These receptors send impulses to the brain, where the impulses synapse with the cardiovascular centers. When stimulated by muscle contraction, these receptors lead to an increased rate and force of heart contraction. Vasoconstriction occurs in inactive skeletal muscles.

Neurohormonal Control

The endocrine system also helps regulate the cardiovascular system. Considerable control is exerted by components of the autonomic nervous system and the hormones of the adrenal medulla. The previous section discussed the influence of the sympathetic nervous system on the heart and the blood vessels. The sympathetic nervous system also innervates the adrenal medulla, causing the adrenal glands to release the hormones

epinephrine and norepinephrine. These hormones travel in the bloodstream to the heart and the blood vessels. Generally, epinephrine and norepinephrine have the same effect on these target organs as the sympathetic nerve fibers innervating them.

In addition to the adrenal hormones, aldosterone and ADH help maintain blood volume and blood pressure as described in the section on blood volume.

FOCUS ON RESEARCH

Cardiovascular Responses to Exercise

Researchers interested in quantifying cardiovascular responses to exercise often rely on both easily obtained measurements and measurements requiring sophisticated laboratory equipment. In this study, Doppler echocardiography (an ultrasound system that is typically found only in hospital settings or well-equipped laboratories; see **Figure 11.18**) was used to investigate the stroke volume response to isometric contraction. Investigators had young boys between the ages of 7 and 12 years perform isometric leg extension exercises at 30% of maximal voluntary contraction (MVC) for 3 minutes. Researchers measured systolic blood pressure (SBP) and diastolic blood pressure (DBP) through standard auscultatory methods (see **Figure 11.20**) and then calculated mean arterial pressure (MAP). The results of this study when compared with previous research on young adult males suggest that young boys respond to isometric exercise in much the same way that young adult males do, with an increase in MAP and a small decrease in SV.

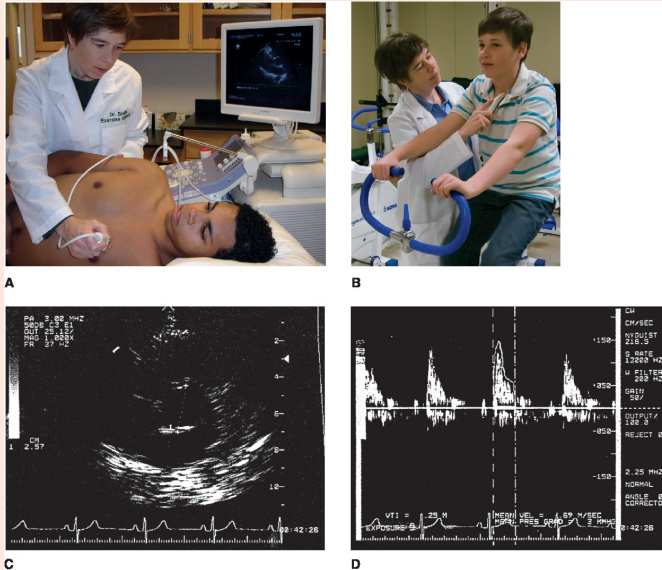
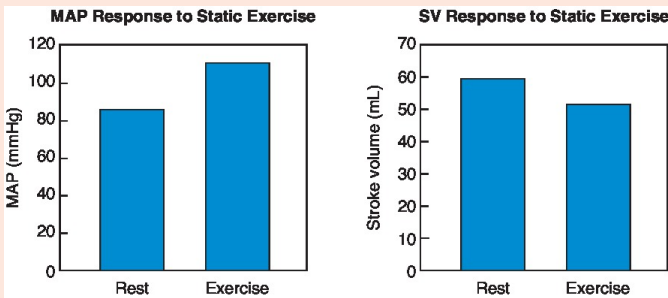


Figure 11.18 Doppler Echocardiography.

Ultrasound technician obtains aortic diameter (A) and Doppler waveforms from the suprasternal notch (B). Printout of aortic diameter (C) and Doppler waveforms (D).



Source: Rowland, T., K. Heffernan, S. Y. Jae, G. Echols, G. Krull, & B. Fernhall: Cardiovascular responses to static exercise in boys: Insights from tissue Doppler imaging. *European Journal of Applied Physiology*. 97(5):637–642 (2006).

Measurement of Cardiovascular Variables

Cardiovascular variables are routinely measured and monitored in sports, fitness, rehabilitation, and research settings. The following variables are measured in order to assess fitness, prescribe exercise, and monitor physiological responses to exercise. Most of these variables can be assessed both at rest and during submaximal or maximal exercise.

Cardiac Output

Recall that cardiac output (\dot{Q}) is equal to the product of stroke volume and heart rate (Equation 11.3). However, because stroke volume has historically been very difficult to measure, cardiac output has been calculated from another known relationship described by the **Fick equation**:

Fick Equation An equation used to calculate cardiac output from oxygen consumption ($\dot{V}O_2$) and arteriovenous oxygen difference (a-vO₂diff).

$$\begin{aligned} \text{cardiac output (L} \cdot \text{min}^{-1}) &= \text{oxygen consumption} \\ &(\text{mL} \cdot \text{min}^{-1}) \div \text{arteriovenous oxygen difference} \\ &(\text{mL} \cdot \text{L}^{-1}) \\ \text{or} \end{aligned}$$

$$11.9 \quad \dot{Q} = \frac{\dot{V}O_2}{a-vO_2\text{diff}}$$

See animation, Fick Principle, on Lippincott Connect



The direct determination of cardiac output via the Fick equation requires measurement of both oxygen consumption and arteriovenous oxygen difference. Many laboratories can directly

measure oxygen consumption (see [Chapter 4](#)). The assessment of arteriovenous oxygen difference (a-vO₂diff), however, is more problematic. This invasive test requires a sample of arterial blood from an artery and a sample of mixed venous blood from the vena cava or right atrium. Given the difficulties of obtaining a direct measurement of \dot{Q} , researchers often take the approach of measuring SV and HR and calculating \dot{Q} ($\dot{Q} = SV \times HR$).

Stroke Volume

Advances in technology have made the assessment of stroke volume (SV) easier, particularly during exercise. Stroke volume can now be measured using **Doppler echocardiography**. This is a technique that calculates stroke volume from noninvasive measurements of aortic cross-sectional area (CSA) and time-velocity integral (TVI) of the blood flow in the ascending aorta ([Figure 11.18](#)) using this formula:

Doppler Echocardiography A technique that calculates stroke volume from measurements of aortic cross-sectional area and time-velocity integrals in the ascending aorta.

$$\text{stroke volume (mL)} = \text{cross-sectional area (cm}^2\text{)} \times \text{time-velocity integrals (cm)}$$

or

$$11.10 \quad SV = CSA \times TVI$$

Note that for conversion purposes, cm³ = mL.

Two-dimensional echocardiography is used to measure aortic diameter ([Figure 11.18](#)). Cross-sectional area is then calculated from a geometric model using the following formula:

$$\text{cross-sectional area (cm}^2\text{)} = \text{diameter (cm)}^2 \times \pi / 4$$

or

$$11.11 \quad CSA = d^2 \times \pi / 4$$

Doppler ultrasound is used to assess blood velocity in the

ascending aorta (**Figure 11.18B**). From the Doppler waveforms, a time-velocity integral is obtained. Once stroke volume is known, cardiac output can be calculated using the formula

$$\dot{Q} = SV \times HR$$

Example

Using the information provided in **Figure 11.18C and D**, calculate SV.

The calculations are as follows:

$$CSA = 2.57^2 \times \pi / 4 = 5.18 \text{ cm}^2$$

$$SV = (5.18 \text{ cm}^2) \times (25 \text{ cm}) = 129.5 \text{ mL}$$

The Doppler method does not require a steady state and thus is a viable option for assessing SV (and \dot{Q}) during maximal exercise. Disadvantages of the Doppler method include the high cost of the equipment and the training required to obtain good time velocities, especially during heavy exercise (**Rowland and Obert, 2002**).

Heart Rate

In a laboratory, heart rate is often obtained by measuring the R-R interval in an ECG recording (**Figure 11.19**). Although the ECG equipment/computer may be programmed to automatically calculate the HR, it is useful to know how this measure is performed. The first step is to calculate the distance the ECG paper travels in 1 minute based on the speed of the paper. The distance between cycles is then measured. Because you now know the number of cycles that occurred within the distance measured, you can solve for the b·min⁻¹ by solving for X in the following equation:

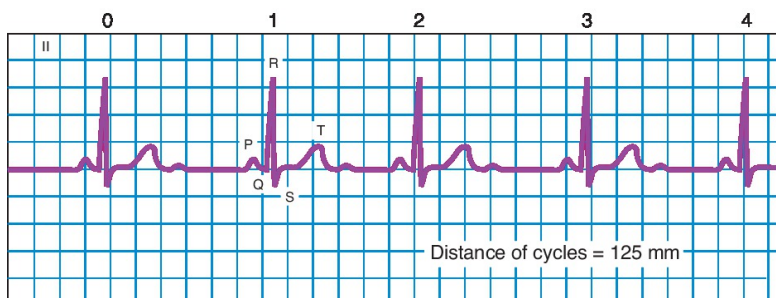


Figure 11.19 ECG Used to Calculate Heart Rate.

$$\text{HR (b} \cdot \text{min}^{-1}\text{)} = [\text{number of beats} \div \text{distance the cycles occupy (mm)}] = [\text{X (b} \cdot \text{min}^{-1}\text{)} \div \text{distance the}$$

$$\text{11.12 paper travels in 1 minute (mm} \cdot \text{min}^{-1}\text{)}]$$

Example

Using the ECG strip in **Figure 11.19**, calculate the heart rate.

Step 1: Using the paper speed, calculate the distance the paper travels in 1 minute. Paper speed equals $25 \text{ mm} \cdot \text{s}^{-1}$. Since there are $60 \text{ s} \cdot \text{min}^{-1}$, $25 \text{ mm} \cdot \text{s}^{-1} \times 60 \text{ s} \cdot \text{min}^{-1} = 1,500 \text{ mm} \cdot \text{min}^{-1}$.

Step 2: Measure the distance between 4 cycles, which in this case is 125 mm.

Step 3: Since 4 cycles (1 box = 5 mm) occurred within the time period required for the ECG paper to travel 125 mm, you calculate how many beats occur in 1 minute by solving for X in the equation:

$$\frac{4}{125} = \frac{X}{1,500}$$

$$125X = 6,000, \quad X = 6,000 / 125 = 48 \text{ b} \cdot \text{min}^{-1}$$

Heart rate can also be recorded by wireless telemetry. Most often, the transmitter is worn around the chest, and the heart rate

signal is transmitted to a small receiver that looks very similar to a watch. Many pieces of fitness equipment also have the ability to pick up the HR signal from the transmitter or to measure heart rate by having the exercisers grasp a sensor built into the exercise equipment.

Heart rate is often measured during exercise to monitor exercise intensity. In some exercise sessions, however, heart rate-measuring devices are not available. Heart rate can then be assessed by counting the pulse—a method called palpation. The pulse can be felt at the carotid or radial artery. This technique requires instruction and practice.

To measure an exercise heart rate, the person usually pauses and finds the pulse as quickly as possible. The pulse count begins with zero and is counted for a set period of time, usually 6, 10, or 15 seconds, using a watch. This pulse count is then multiplied by 10, 6, or 4, respectively, to obtain the per-minute heart rate. A period less than 1 minute is used because the heart rate drops quickly once exercise is paused.

Maximal Oxygen Consumption

Maximal oxygen consumption is the highest amount of oxygen an individual can take in and utilize to produce energy (ATP) aerobically while breathing air during heavy exercise. It is

abbreviated as $\dot{V}O_2 \text{ max}$ to indicate the maximal volume of oxygen consumed. The respiratory system brings in the oxygen from the environment, the cardiovascular system transports the oxygen, and the cells extract the oxygen and use it in the production of energy (ATP). The assessment of maximal oxygen consumption is therefore a means of quantifying the functional

capacity of the entire cardiovascular system. $\dot{V}O_2 \text{ max}$ is often considered the single most important variable in describing an individual's fitness level and is routinely used to describe an individual's cardiorespiratory capacity.

The limit of cardiovascular function is reached at the highest attainable oxygen consumption. By rearranging the Fick equation (Equation 11.9), this is expressed:

$$11.13 \quad \dot{V}O_2 \max = (\dot{Q} \max) \times (a-vO_2 \text{ diff} \max)$$

Although $\dot{V}O_2 \max$ can be calculated from the variables in Equation 11.13, it is typically measured in the laboratory using open-circuit indirect spirometry with the equipment often organized into a metabolic cart. The assessment of oxygen consumption is described in detail in Chapter 4. Although oxygen is utilized only in aerobic metabolic production of ATP, the delivery of oxygen is primarily limited by the cardiovascular system. Thus, $\dot{V}O_2 \max$ is considered to be a cardiovascular variable.

Field Tests of Cardiorespiratory Capacity

Several field techniques may be used to assess cardiorespiratory endurance and estimate cardiorespiratory capacity ($\dot{V}O_2 \max$).

The use of even estimated $\dot{V}O_2 \max$ values optimizes the correct classification of health risk in health-related physical fitness tests (Cureton et al., 2013). Field techniques include submaximal cycle ergometer tests, step tests, and distance walks or runs. By far, the most practical and inexpensive method, especially when large numbers of individuals are tested in a short period of time (such as in a school setting), is the distance run. Walking tests are recommended for testing the elderly, overweight, or very sedentary individuals. Three tests are briefly described here: the 1-mile walk/run, the 20 meter shuttle test (20MST), also known as the progressive aerobic cardiovascular endurance run (PACER) (The Cooper Institute, 2017), and the Rockport Fitness Walking Test (RFWT) (Kline et al., 1987).

The mile run test allows the individual to walk if necessary, but the intent is to cover the mile as quickly as possible, and that goal is best accomplished by running the entire distance. Thus, the individual is asked to perform at a high percentage of his or her maximal capacity for the entire test.

A $\dot{V}O_2 \max$ estimate from the mile run can be calculated as follows (Cureton et al., 1995):

$$\begin{aligned}\dot{V}O_2 \text{ max} &= 0.21 (\text{age} \times \text{sex}) - 0.84 (\text{BMI}) - \\ &8.41 (\text{MT}) + 0.34 (\text{MT}^2) + 108.94 \\ 11.14 \quad \text{SEE} &= 4.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

where age is the age of the individual in years; sex is 0 if female or 1 if male; BMI is the body mass index (weight in kilograms divided by height in meters squared); and MT is the time it takes the individual to run a mile in minutes.

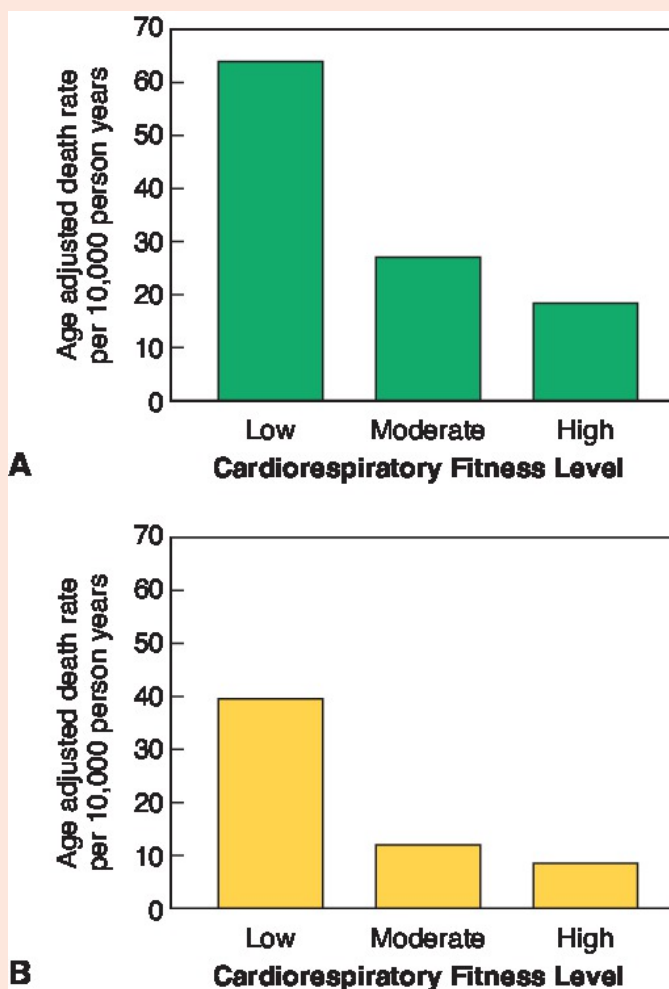
FOCUS ON RESEARCH

Cardiovascular Fitness as a Vital Sign?

Maximal oxygen consumption ($\dot{V}O_2 \text{ max}$) is the criterion measure for aerobic fitness, and it reflects the functional capacity of the cardiovascular system. It is a powerful predictor of overall health outcomes and is the fourth leading risk factor for cardiovascular disease. The American Heart Association (AHA) compiled evidence on the associations between low CRF and cardiovascular disease, all-cause mortality, and cancer-related mortalities with such compelling results that they advocate for the use of $\dot{V}O_2 \text{ max}$ as a “vital sign” similar to the way that clinicians routinely assess other vital signs such as body temperature, heart rate, and respiration rate (Ross et al., 2016).

A landmark study by Blair and colleagues in 1989 reported that for both men and women, the risk of all-cause death rates decreased as CRF increased (Blair et al., 1989). In the study, greater than 13,000 participants were assessed for CRF level at baseline and followed up an average of 8 years later for all-cause mortality. The results showed age-adjusted death rate per 10,000 person-years in males of 64.6, 27.1, and 18.6 for low, moderate, and high CRF, respectively, and in females 39.5, 12.2, and 8.5 for low, moderate, and high CRF, respectively.

Since that time, many authors have reported on the role of CRF in overall health outcomes. As reported by the AHA, even small increases in CRF (1 METs) are associated with 10–25% improvement in survival. What is possibly more shocking is that across studies more than half of the improvements in survival with higher CRF occurred between the least fit and next to least fit groups.



The age-adjusted death rate of males (A) and females (B) with low, moderate, and high cardiorespiratory fitness.

Source (s): Ross, R., S. N. Blair, R. Arena, T. S. Church, J. P. Després, B. A. Franklin, W. L. Haskell, L. A. Kaminsky, B. D. Levine, C. J. Lavie, J. Myers, J. Niebauer, R. Sallis, S. S. Sawada, X. Sui, U. Wisløff; American Heart Association Physical Activity Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Clinical Cardiology; Council on Epidemiology and Prevention; Council on Cardiovascular and Stroke Nursing; Council on Functional Genomics and Translational Biology; Stroke Council. Importance of assessing cardiorespiratory fitness in clinical practice: A case for fitness as a clinical vital sign: A scientific statement from the American Heart Association. *Circulation*. 2016;134(24):e653–e699; Blair, S. N., H. W. Kohl III, R. S. Paffenbarger Jr., D. G. Clark, K. H. Cooper, L. W. Gibbons. Physical fitness and all-cause mortality: A prospective study of healthy men and women. *JAMA*. 1989;262:2395–2401.

Example

Estimate $\dot{V}O_2 \text{ max}$ given the following data:

Age = 15 years; BMI = 24.3 kg·m²

Sex = female; MT = 8.75 minutes

The calculation is

$$\begin{aligned}\dot{V}O_2 \text{ max} &= (0.21)(15 \times 0) - (0.84)(24.3) - (8.41) \\ &\quad (8.75) + 0.34(8.75^2) + 108.94 \\ &= 40.97 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

Note that the equation includes an indication of the standard error of the estimate (SEE). For $\dot{V}O_2 \text{ max}$, acceptable errors generally range from 3 to 5 mL·kg⁻¹·min⁻¹. The 4.8 mL·kg

$-1\cdot\text{min}^{-1}$ for this equation falls within that range. The SEE means that as estimates, not measured values, the calculated results could vary from the measured value by that much. That is, the $40.97\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ calculated in the example might actually be anywhere from 36.17 to $45.77\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

The RFWT also covers a 1-mi distance; however, it is specifically designed for walking, with the fastest sustainable walking speed as the goal. Depending on the age and fitness level of the participant, this speed may or may not represent a high percentage of maximal aerobic capacity (Kline et al., 1987).

Unlike the previous set distance tests, the PACER is a multistage fitness test adapted from the 20-m shuttle run developed in Europe by Léger and his colleagues (1982, 1988). It most closely resembles a graded exercise test that would be performed on a treadmill in a laboratory. It begins with a light workload and progresses, in small increments, until the individual can no longer complete the required number of laps per minute. This test is run between two lines marked 20 m apart on a smooth unobstructed surface. This test does not require a sustained high-intensity effort but a gradual progression from submaximal to maximal effort. Pacing is accomplished by prerecorded music or beeps (The Cooper Institute, 2017).

The equations used to estimate maximal oxygen consumption from the RFWT (Kline et al., 1987; McSwegin et al., 1998) and the 20 MST/PACER test (Cureton et al., 2013; Léger et al., 1988; Plowman and Liu, 1999) are given in Appendix B. Criterion-referenced norms are available for evaluation of the results of all these field tests (The Cooper Institute, 2017).

Blood Pressure

In well-equipped laboratories and hospitals, blood pressure can be measured directly by an intra-arterial transducer. A small transducer is inserted into the artery, and systolic blood pressure and diastolic blood pressure are recorded for every beat of the heart. Although this procedure provides valuable information, it is not practical for routine use because as an invasive procedure it involves risks.

By far, the most common technique for obtaining blood

pressure measurements is the *auscultation* method. The indirect method of auscultation uses a sphygmomanometer and a blood pressure cuff wrapped around the upper arm (**Figure 11.20A**). For a blood pressure measurement to be accurate and meaningful, the proper cuff size must be used to obtain the measurement ([Muntner et al., 2019](#)). The blood pressure cuff is inflated to a pressure greater than systolic pressure (140 mmHg) as shown in **Figure 11.20B**. Note that systolic blood pressure (**Figure 11.20C**) is taken as the First Korotkoff sound (the first loud sound heard through the stethoscope) but that there are two diastolic blood pressures (**Figure 11.20D**). The first diastolic blood pressure (DBP1) occurs when the sound heard through the stethoscope is muffled (the Fourth Korotkoff sound). The second diastolic blood pressure (DBP2) occurs when sound disappears through the stethoscope (the Fifth Korotkoff sound). The pressure at the Fifth Korotkoff sound (DBP2) is considered the best measure of diastolic blood pressure in normal adults at rest. However, DBP1 is recommended for children, for adults during exercise, and for adults if DBP2 is lower than 40 mmHg ([American Society of Hypertension, 1992](#)). This is important for an accurate description of blood pressure and in the calculation of mean arterial pressure (MAP).

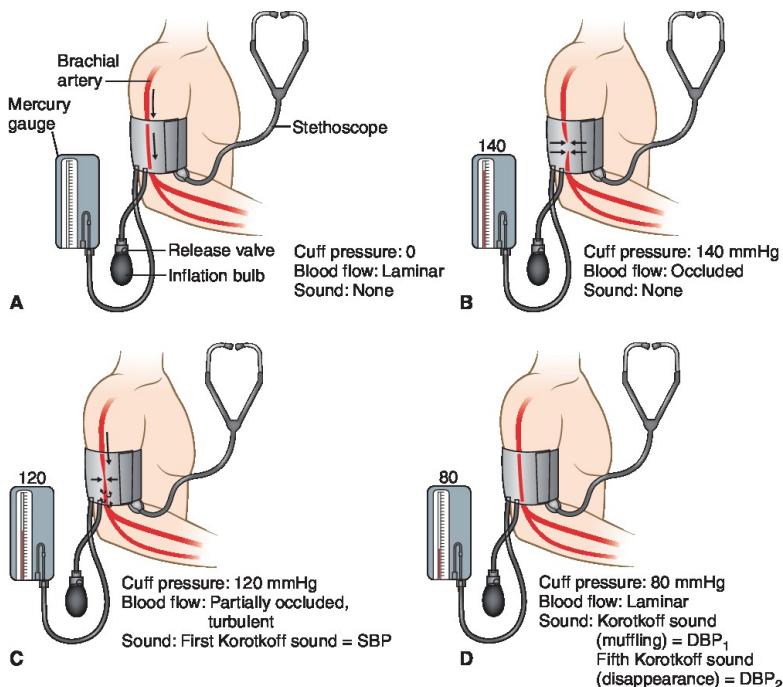


Figure 11.20 Assessment of Blood Pressure.

A. An appropriate-sized blood pressure cuff is placed around the upper arm. B. The blood pressure cuff is inflated to approximately 20 mmHg higher than SBP in order to occlude arterial blood flow. C. Pressure in the cuff is slowly released until the First Korotkoff sound is heard. D. Pressure in the cuff continues to be released until there is a muffling or disappearance of the Korotkoff sounds.

In resting and exercise recovery situations, mean arterial pressure is determined by first calculating pulse pressure. Pulse pressure is equal to the difference between SBP and DBP₂ ($PP = SBP - DBP_2$). MAP is then calculated as

mean arterial pressure (mmHg) = pulse pressure
(mmHg) / 3 + diastolic blood pressure₂ (mmHg)
or

11.15a
$$\text{MAP} = \frac{\text{PP}}{3} + \text{DBP}_2$$

MAP is not simply computed as the average of SBP and DBP because diastole lasts longer than systole and is thus weighted more heavily in the computation of MAP.

For adults during exercise or for children at rest, MAP can be computed according to a modified equation that uses DBP₁ (Robinson et al., 1988):

mean arterial pressure (mmHg) = pulse pressure
(mmHg) / 2 + diastolic blood pressure₁(mmHg)
or

11.15b
$$\text{MAP} = \frac{\text{PP}}{2} + \text{DBP}_1$$

This formula provides a more accurate measurement of MAP during exercise because it gives less weight to DBP. Although both systole and diastole time shorten because of the increased heart rate, diastole shortens proportionally more.

Summary

1. The primary functions of the cardiovascular system are to transport oxygen and nutrients to the cells of the body and transport carbon dioxide and waste products from the cells; to regulate body temperature, pH levels, and fluid balance; and to protect the body from blood loss and infection.
2. The cells of the heart, called myocytes (or cardiac muscle cells) or collectively the myocardium, are functionally linked by intercalated discs with gap junctions. When one cell is depolarized, the stimulation spreads over the entire myocardium.
3. The heart has its own conduction system, consisting of the

sinoatrial (SA) node, intermodal fibers and Bachmann's bundle, the atrioventricular (AV) node, the bundle of His, the left and right bundle branches, and Purkinje fibers, all of which rapidly spread the electrical signal throughout the heart.

4. The cardiac cycle refers to the alternating periods of relaxation and contraction of the heart. The contraction phase is called systole, and the relaxation phase is called diastole. There are known relationships among the electrical, pressure, volume, and contractile events throughout the cardiac cycle.
5. The volume of blood ejected from the heart with each beat is known as stroke volume. The amount of blood ejected from the heart each minute is called cardiac output. Under resting conditions, the heart ejects approximately 60% of the blood that is returned to it; this percentage represents the ejection fraction.
6. The cardiovascular system is primarily controlled by the medulla oblongata (cardioaccelerator, cardioinhibitor, and vasomotor centers). Factors affecting these centers include the following:
 - a. Higher brain centers that exert conscious and unconscious control
 - b. Baroreceptors that are sensitive to mean arterial pressure
 - c. Stretch receptors that sense blood return to the right atrium
 - d. Chemoreceptors that sense the PO_2 , PCO_2 , and H^+
7. Maximal oxygen consumption ($\dot{V}O_2 \text{ max}$) is the highest amount of oxygen that the body can take in, transport, and use. Assessment of $\dot{V}O_2 \text{ max}$ allows for quantifying the functional capacity of the entire cardiovascular system and is related to health risks.

Review Questions

1. Describe the primary functions of the cardiovascular system.
2. Diagram the conduction system of the heart, and describe how activation of the SA node leads to contraction of the heart.
3. Describe the electrical events in the heart in relation to pressure in the left ventricle, the volume of blood in the left ventricle, and the position of the heart valves.
4. Describe the major mechanical, electrical, and volume changes that occur throughout the cardiac cycle.
5. Describe how myocardial oxygen consumption changes with exercise. What determines myocardial oxygen consumption and how can it be estimated during exercise?
6. Identify the different vessels of the peripheral circulation, and describe the velocity and pressure in each. What accounts for the differences?
7. Describe the hormonal mechanisms by which the body attempts to compensate for a decrease in plasma volume.
8. Discuss the neurohormonal regulation of the cardiovascular system and the factors that affect such regulation.
9. Describe how cardiac output can be measured and explain why it is not routinely measured in exercise physiology labs or in field settings.
10. Why is maximal oxygen consumption ($\dot{V}O_2 \text{ max}$) considered to be a cardiovascular variable?
11. Explain the steps involved in attaining an accurate measurement of blood pressure.

For further review and study tools, visit Lippincott Connect.

Literature Search

1. There is an enormous amount of research available on cardiovascular health and the relationship between fitness and health. To better understand the research that has been

done on this important issue, do a literature search using a search engine such as PubMed, Google scholar, or Web of Science.

- a. Search Exercise and Cardiovascular Health. This search will yield many articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example:
 - i. Effect of dynamic resistance exercise on cardiovascular health
 - ii. Effect of high-intensity exercise on vascular function
 - iii. Association between exercise volume and cardiovascular events
 - iv. Continue your search for aspects of this topic that are of particular interest to you

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12

Cardiovascular Responses to Exercise



CHAPTER OUTLINE

Introduction

Cardiovascular Responses to Aerobic Exercise

Short-Term, Light to Moderate Submaximal Aerobic Exercise

Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

Incremental Aerobic Exercise to Maximum

High-Intensity Interval Exercise

Upper-Body versus Lower-Body Aerobic Exercise

Cardiovascular Responses to Static Exercise

Intensity of Muscle Contraction

Blood Flow during Static Contractions

Comparison of Aerobic and Static Exercise

Cardiovascular Responses to Dynamic Resistance Exercise

Varying Load/Constant Repetitions

Varying Load/Repetitions to Failure

Constant Load/Repetitions to Failure

Male-female Cardiovascular Differences during Exercise

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

Incremental Aerobic Exercise to Maximum

Static Exercise

Cardiovascular Responses of Children and Adolescents to Exercise

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

Incremental Aerobic Exercise to Maximum

Static Exercise

Cardiovascular Responses of Older Adults to Exercise

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

Incremental Aerobic Exercise to Maximum

Static Exercise

Summary

Review Questions

OBJECTIVES

After studying the chapter, you should be able to:

- Graph and explain the pattern of response for the major cardiovascular variables during short-term, light to moderate submaximal aerobic exercise.
- Graph and explain the pattern of response for the major cardiovascular variables during long-term, moderate to heavy

submaximal aerobic exercise.

- Graph and explain the pattern of response for the major cardiovascular variables during incremental aerobic exercise to maximum.
- Explain the importance of measuring maximal oxygen consumption ($\dot{V}O_2 \text{ max}$).
- Graph and explain the pattern of response for the major cardiovascular variables during dynamic resistance exercise.
- Graph and explain the pattern of response for the major cardiovascular variables during static exercise.
- Compare the response of the major cardiovascular variables to short-term, light to moderate submaximal aerobic exercise; long-term, moderate to heavy submaximal aerobic exercise; incremental aerobic exercise to maximum; static exercise; and dynamic resistance exercise.
- Discuss the similarities and differences between the sexes in the cardiovascular responses to the various categories of exercise.
- Discuss the similarities and differences between children/adolescents and young adults in the cardiovascular responses to the various categories of exercise.
- Discuss the similarities and differences between young/middle-aged and older adults in the cardiovascular responses to the various categories of exercise.

Introduction

All types of human movement, no matter what the mode, duration, or intensity, require an expenditure of energy above resting values. Much of this energy comes from the use of oxygen. To supply the working muscles with the needed oxygen, the cardiovascular and respiratory systems work together. The responses of the respiratory system during exercise are detailed in [Chapter 10](#). This chapter describes the parallel cardiovascular responses to dynamic aerobic activity, static exercise, and dynamic resistance exercise. Minimal attention is paid here to short-term, high-intensity anaerobic exercise because this type of activity is typically performed to stress the metabolic system and is therefore discussed in detail in the metabolic unit.

Cardiovascular Responses to Aerobic Exercise

Aerobic exercise requires more energy—and therefore more oxygen—than either static or dynamic resistance exercise. How much oxygen is needed depends primarily on the intensity of the activity and secondarily on its duration. As in the discussion of respiration, this chapter categorizes exercises as:

1. Short-term (5–10 minutes), light (30–49% maximal oxygen consumption, $\dot{V}O_2 \text{ max}$) to moderate (50–74% $\dot{V}O_2 \text{ max}$) submaximal exercise
2. Long-term (>30 minutes), moderate to heavy (60–85% $\dot{V}O_2 \text{ max}$) submaximal exercise
3. Incremental exercise to maximum (increasing from ~30 to 100% $\dot{V}O_2 \text{ max}$).

Short-Term, Light to Moderate Submaximal Aerobic Exercise

Figure 12.1 depicts generalized cardiovascular responses to short-term, light to moderate submaximal aerobic exercise. The actual magnitude of each variable's change depends on the work rate or load, environmental conditions, and the individual's genetic makeup and fitness level. At the onset of light- to moderate-intensity exercise, cardiac output (\dot{Q}) initially increases to plateau at a steady state (see [Figure 12.1A](#)). Cardiac output plateaus within the first 2 minutes of exercise, reflecting the fact that cardiac output is sufficient to transport the oxygen needed to support the metabolic demands of the activity. Cardiac output increases because of an initial increase in both stroke volume (SV) (**Figure 12.1B**) and heart rate (HR) (**Figure 12.1C**); both level off within 2 minutes.

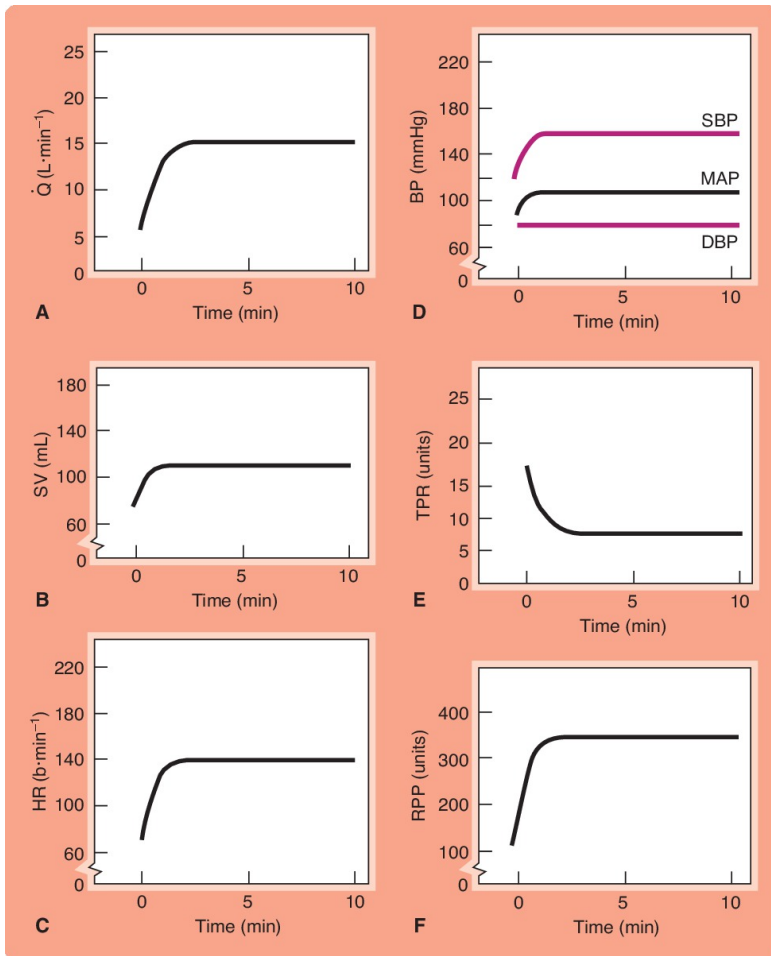


Figure 12.1 Cardiovascular Responses to Short-Term, Light to Moderate Submaximal Aerobic Exercise.

A. Cardiac output (\dot{Q}). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Blood pressure (SBP, MAP, and DBP). **E.** Total peripheral resistance (TPR). **F.** Rate-pressure product (RPP).

During exercise of this intensity, the cardiorespiratory system can meet the body's metabolic demands; thus, this type of exercise is often called **steady-state** or steady-rate exercise. During steady-state exercise, energy provided aerobically is balanced with the energy required to perform the exercise. The

plateau in cardiovascular variables (in **Figure 12.1**) indicates that a steady state has been achieved.

Steady-State A condition in which the energy provided during exercise is balanced with the energy required to perform that exercise, and factors responsible for the provision of this energy reach elevated levels of equilibrium.

Stroke volume (SV) increases rapidly at the onset of exercise due to an increase in venous return, which in turn increases the end-diastolic volume (EDV) (preload). The increased preload stretches the myocardium and causes it to contract more forcibly, as described by the Frank-Starling law of the heart (Chapter 11). Contractility of the myocardium is also enhanced by the sympathetic nervous system, which is activated during physical activity. An increase in the EDV and a decrease in the end-systolic volume (ESV) both contribute to the increase in the SV during light to moderate dynamic exercise ([Poliner et al., 1980](#)). HR increases immediately at the onset of activity as a result of parasympathetic withdrawal. As exercise continues, further increases in the HR result from the sympathetic nervous system activation ([Rowell, 1986](#)).

Systolic blood pressure (SBP) rises in a pattern very similar to that of cardiac output: an initial increase followed by a plateau once steady state is achieved (**Figure 12.1D**). The increase in SBP results from the increased cardiac output. SBP would be even higher if not for the fact that resistance decreases, thereby partially offsetting the increase in cardiac output. When blood pressure (BP) is measured intra-arterially, diastolic blood pressure (DBP) does not change. When it is measured by auscultation, it either does not change or may go down slightly. DBP remains relatively constant because of peripheral vasodilation, which facilitates blood flow to the working muscles. The small rise in SBP and the lack of a significant change in DBP cause the mean arterial pressure (MAP) to rise only slightly, following the pattern of SBP.

Total peripheral resistance (TPR) decreases because of vasodilation in the active muscles (**Figure 12.1E**). This

vasodilation results primarily from the influence of local chemical factors (lactate, K^+ , and so on), which reflect increased metabolism. An increase in blood flow due to an increase in or BP also creates a shear stress that causes the endothelium to release vasodilatory factors, mostly nitric oxide, which also contributes to vasodilation. The TPR can be calculated using Equation 11.8:

$$\text{TPR} = \frac{\text{MAP}}{\dot{Q}}$$

Example

Calculate TPR for an individual doing short-term, light to moderate submaximal aerobic exercise by using the following information from **Figure 12.1A and D**:

$$\text{MAP} = 110 \text{ mmHg}; \dot{Q} = 15 \text{ L} \cdot \text{min}^{-1}$$

The computation is

$$\text{TPR} = \frac{110 \text{ mmHg}}{15 \text{ L} \cdot \text{min}^{-1}} = 7.33 \text{ (TPR units)}$$

Thus, in this example, TPR is 7.33.

The decrease in TPR has two important implications. First, vasodilation of the vessels supplying the active muscle causes decreased resistance that leads to an increased blood flow, thereby increasing the availability of oxygen and nutrients. Second, the decreased resistance keeps MAP from increasing dramatically. The increase in the MAP is determined by the relative changes in cardiac output and the TPR. Since cardiac output increases more than resistance decreases, the MAP increases slightly during dynamic aerobic exercise.

Myocardial oxygen consumption increases during dynamic

aerobic exercise because the heart must do more work to increase cardiac output to supply the working muscles with additional oxygen. The rate-pressure product (RPP) increases due to increases in the HR and the SBP. This increase reflects the greater myocardial oxygen demand of the heart during exercise (**Figure 12.1F**). In the **Check Your Comprehension 1** box, calculate cardiovascular variables based on measured values, which are examples of normal responses to several exercise categories. Refer back to these answers as the categories are discussed in this chapter.

Blood volume decreases during submaximal aerobic exercise. **Figure 12.2** shows the reduction of plasma volume during 30 minutes of moderate cycle ergometer exercise (60–70% $\dot{V}O_2 \text{ max}$) in a warm environment (Fortney et al., 1981). The largest decrease occurs during the first 5 minutes of exercise, and then plasma volume stabilizes. This rapid decrease in plasma volume suggests that it is fluid shifts, rather than fluid loss, that account for the initial decrease (Wade and Freund, 1990). The magnitude of the decrease in plasma volume depends on the intensity of exercise, environmental factors, and the individual's hydration status.

CHECK YOUR COMPREHENSION 1

The following measurements were obtained from a 42-year-old man at rest and during several exercise sessions:

| Condition | HR (b·min ⁻¹) | SBP (mmHg) | DBP (mmHg) | Q̇ (L·min ⁻¹) |
|---|------------------------------|---------------|---------------|------------------------------|
| Rest | 80 | 134 | 86 | 6 |
| Short-term, light to moderate submaximal aerobic exercise | 130 | 150 | 86 | 10 |
| Long-term, moderate to heavy submaximal aerobic exercise | 155 | 170 | 88 | 13 |
| Incremental aerobic exercise to maximum | 180 | 200 | 88 | 15 |
| Static exercise | 135 | 210 | 100 | 8 |
| Dynamic resistance exercise | 126 | 180 | 92 | 10 |

Calculate MAP, TPR, and RPP for each condition.

Check your answer in Appendix C.

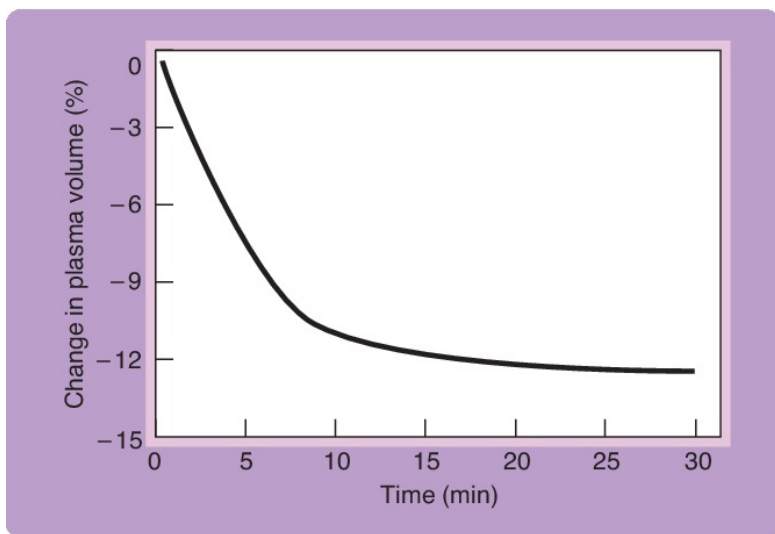


Figure 12.2 Percent Reduction of Plasma Volume during 30-Minute Moderate Cycle Ergometer Exercise.

Source: Fortney et al. (1981).

Cardiac output reflects the total amount of blood pumped per minute by the heart. Cardiac output increases during exercise, but there are also adjustments in how the blood flow is distributed to different organs. **Figure 12.3** shows the distribution of cardiac output at rest and during light aerobic exercise. Notice that cardiac output increases from 5.8 to 9.4 L·min⁻¹ in this example (the increase in \dot{Q} is illustrated by the larger pie chart). The most dramatic change in cardiac output distribution with light exercise is the increased percentage (from 21 to 47%) and the increased blood flow (from 1,200 to 4,500 mL) to the working muscles to support energy production. Skin blood flow also increases to meet the thermoregulatory demands of exercise. The absolute blood flow to the coronary muscle also increases, although its percentage of cardiac output remains relatively constant. The absolute amount of cerebral blood flow remains constant, while the percentage of cardiac output distributed to the brain decreases. Both renal and splanchnic blood flow are modestly decreased during light exercise. In summary, with aerobic exercise, cardiac output increases, and it is redistributed so that

tissues that need increased blood flow, such as the muscles and skin, receive it, and other tissues receive either equal or less blood flow.

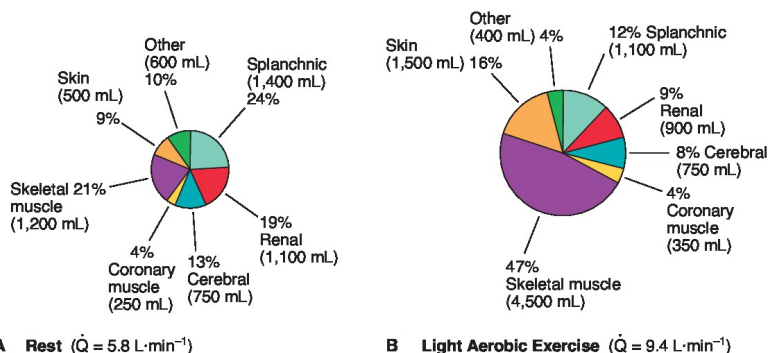


Figure 12.3 Distribution of Cardiac Output at Rest (A)



and during Light Aerobic Exercise (B).

Values are given as a percentage (%) of total cardiac output and parenthetically as an absolute volume of blood (mL) delivered to the tissue each minute. **Source:** Data from [Anderson \(1968\)](#).

Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

The cardiovascular responses to long-term, moderate to heavy submaximal aerobic exercise (60–85% of $\dot{V}O_2 \text{ max}$) are shown in **Figure 12.4**. Similar to light to moderate workloads, cardiac output increases rapidly during the first minutes of exercise and then plateaus and remains relatively constant throughout the exercise (**Figure 12.4A**). Notice, however, that the absolute cardiac output attained is higher during heavy exercise than during light to moderate exercise. The increase in cardiac output results from increased SV and HR.

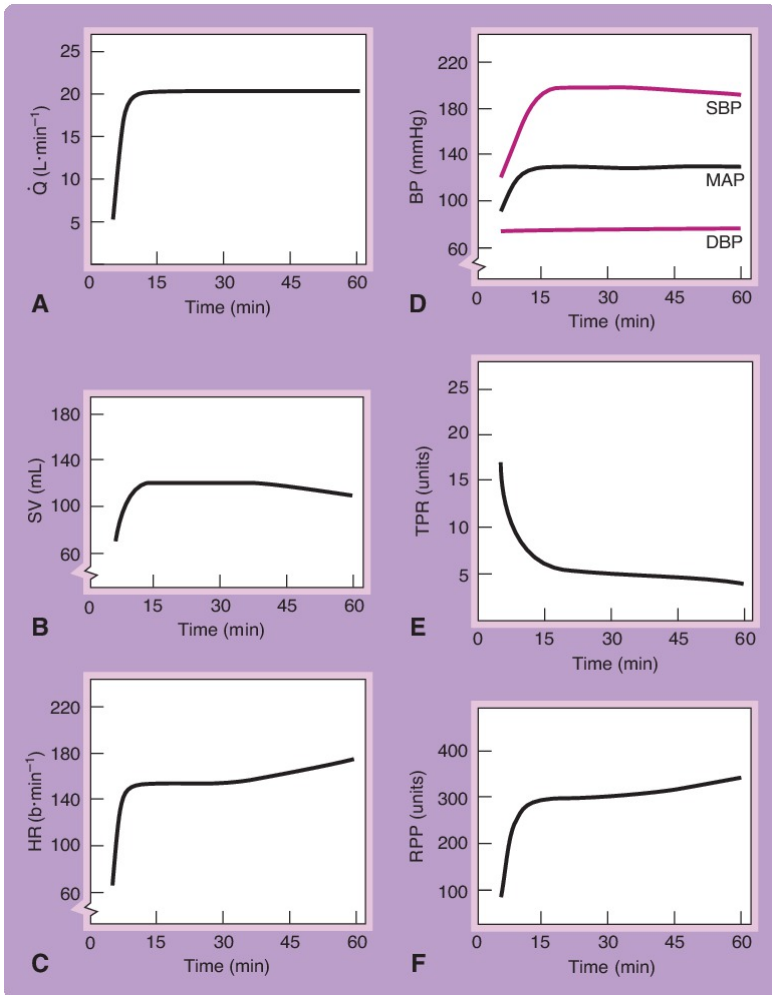


Figure 12.4 Cardiovascular Responses to Long-Term, Moderate to Heavy Submaximal Aerobic Exercise.

A. Cardiac output (\dot{Q}). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Blood pressure (SBP, MAP, and DBP). **E.** Total peripheral resistance (TPR). **F.** Rate-pressure product (RPP).

SV has an initial increase, plateaus, and then has a negative (downward) drift as exercise duration continues past approximately 30 minutes. SV increases rapidly during the first minutes of exercise and then plateaus after a workload of

approximately 40–50% of $\dot{V}O_2 \text{ max}$ is achieved (Åstrand et al., 1964) (Figure 12.4B). Thus, during work that requires more than

50% $\dot{V}O_2 \text{ max}$, the SV response does not depend on intensity. SV remains relatively constant during the first 30 minutes of heavy exercise.

As with short-term, light to moderate submaximal aerobic exercise, the increase in SV is believed to result from an increased venous return (leading to the Frank-Starling mechanism) and increased contractility due to sympathetic nerve stimulation. Thus, changes in SV occur because EDV increases and ESV decreases (Poliner et al., 1980). EDV increases primarily because of the increased venous return of blood to the heart by the active muscle pump and increased venoconstriction, which decreases venous pooling. ESV decreases because of augmented contractility of the heart, which effectively ejects more blood.

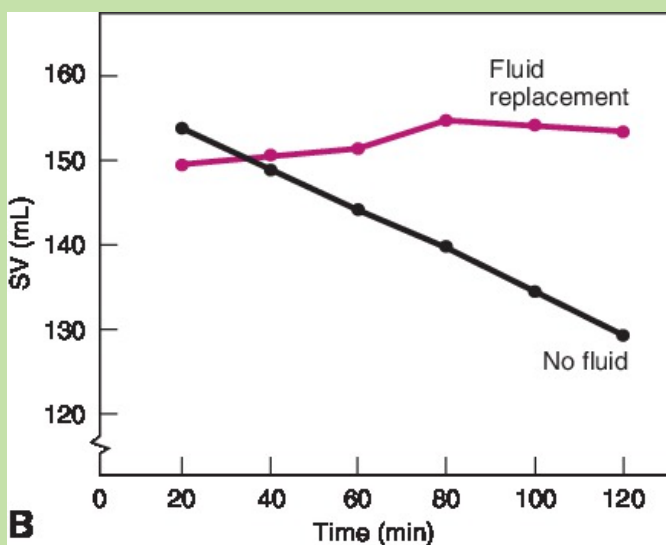
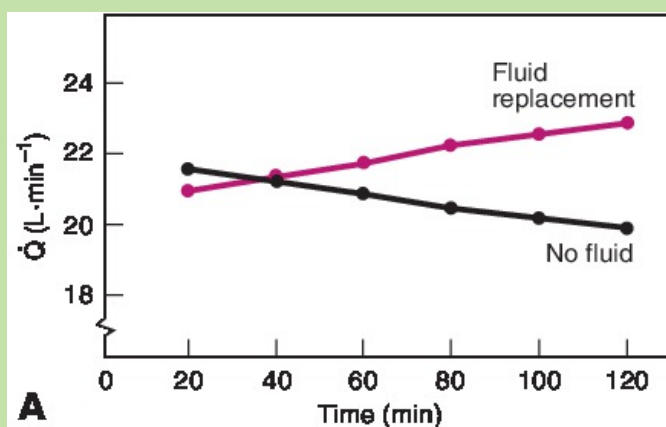
If the exercise continues beyond approximately 30 minutes, SV gradually drifts downward while remaining above the resting value. This downward shift is most often attributed to thermoregulatory stress, which results in vasodilation, plasma loss, and a redirection of blood to the cutaneous vessels to dissipate heat, effectively reducing venous return and thus SV. This theory suggests that HR increases to compensate for a decrease in SV in order to maintain \dot{Q} . An alternate viewpoint suggests that the downward drift in SV is due to an increase in HR (due to augmented sympathetic nerve activity) that leads to a reduced filling time, thus leading to a reduced SV (Rowland, 2005b).

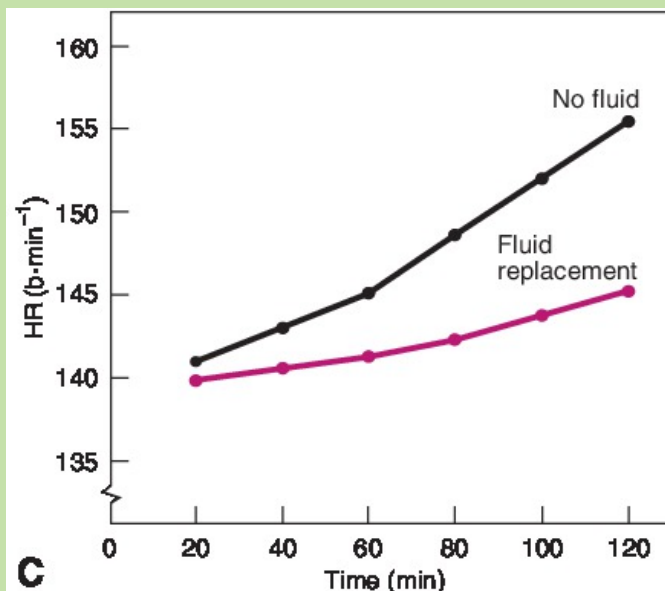
FOCUS ON APPLICATION | *Clinically Relevant*

The Importance of Fluid Ingestion

The magnitude of cardiovascular drift is heavily influenced by fluid ingestion. The accompanying figure presents data from a study in which participants cycled for 2 hours with or without fluid replacement. Values are for minutes 20 through

120; the initial increase in these variables is not shown. When participants consumed enough water to completely replace the water lost through sweat, cardiac output remained nearly constant throughout the first hour of exercise and actually increased during the 2nd hour (panel A). Cardiac output was maintained in the fluid replacement trial because SV did not drift downward (panel B). HR was significantly lower when fluid replacement occurred (panel C). This information is important for coaches and fitness leaders. If your clients exercise for prolonged periods, they must replace the fluids that are lost during exercise, or performance will suffer.





Source: Reprinted with permission from Hamilton, M. T., J. G. Alonso, S. J. Montain, & E. F. Coyle: Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. *Journal of Applied Physiology*. 71(3):871–877 (1991). Copyright © 1991 The American Physiological Society. All rights reserved.

HR initially increases, plateaus at steady state, and then has a positive drift as exercise continues for a prolonged period. HR increases sharply during the first 1–2 minutes of exercise, the magnitude of which depends on the intensity of exercise (**Figure 12.4C**). The increase in HR is brought about by parasympathetic withdrawal and activation of the sympathetic nervous system. After approximately 30 minutes of heavy exercise, HR begins to drift upward. The increase in HR is proportional to the decrease in SV, so cardiac output is maintained during exercise.

These cardiovascular changes, notably in HR and SV, during long-term, moderate to heavy submaximal aerobic exercise without a change in workload, are known as **cardiovascular drift**. Cardiovascular drift is probably associated with rising body

temperature during prolonged exercise. Exercise and heat stress produce competing regulatory demands, as the skin and the muscles compete for increased blood flow. SV decreases as a result of vasodilation, a progressive increase in the fraction of blood being directed to the skin, a decrease in filling time, and a loss of plasma volume (Rowell, 1974; Sjogaard et al., 1988).

Cardiovascular Drift The changes in observed cardiovascular variables that occur during prolonged, heavy submaximal exercise without a change in workload.

SBP response to long-term, moderate to heavy submaximal aerobic exercise is characterized by an initial increase, a plateau at steady state, and a negative drift. SBP increases rapidly during the first 1–2 minutes of exercise, the magnitude of increase depending on the intensity of the exercise (**Figure 12.4D**). SBP then remains relatively stable or drifts slightly downward as a result of continued vasodilation and a resultant decrease in resistance (Ekelund and Holmgren, 1967). DBP does not change, or changes so little that it has no physiological significance, during prolonged exercise in a thermoneutral environment. But it may decrease slightly in a warm environment because of increased vasodilation resulting from heat production. Because of the increased SBP and the relatively stable DBP, MAP increases modestly during prolonged activity. Again, as in light to moderate exercise, MAP increases modestly due to a large increase in \dot{Q} that is offset by a significant decrease in resistance.

TPR decreases rapidly, plateaus, and then has a slight negative drift during long-term heavy exercise (**Figure 12.4E**) because of vasodilation in active muscle and because of vasodilation in cutaneous vessels (Rowell, 1974). Finally, because both HR and SBP increase substantially during heavy work, the rate-pressure product increases markedly with the onset of exercise and then plateaus at steady state (**Figure 12.4F**). An upward drift in rate-pressure product may occur after approximately 30 minutes of exercise because the HR increases more than SBP decreases. The high rate-pressure product reflects the large amount of work the heart must perform to support heavy exercise.

During prolonged exercise, particularly in a warm environment, total body fluid is continually lost due to sweating. This loss typically ranges from 900 to 1,300 mL·hr⁻¹, depending on work intensity and environmental conditions (Wade and Freund, 1990). If fluid is not replaced during long-duration exercise, plasma volume will continually be reduced throughout the exercise.

Figure 12.5 shows the distribution of cardiac output at rest and during heavy aerobic exercise. Notice that cardiac output increases from 5.8 L·min⁻¹ at rest to 17.5 L·min⁻¹ in this example, a threefold increase. The most dramatic change here is the increased blood flow to the working muscle, which now receives 71% of cardiac output. Skin blood flow is also increased to meet the thermoregulatory demands. The absolute blood flow to the coronary muscle increases, while its percentage of cardiac output remains relatively constant. The absolute cerebral blood flow remains constant, while its percentage of cardiac output decreases. Both renal and splanchnic blood flow are further decreased as exercise intensity increases. Although blood flow to the working muscle increases during aerobic exercise, blood flow to the inactive muscle decreases because of vasoconstriction. Vasoconstriction in inactive muscle is necessary to ensure that cardiac output can supply adequate blood flow to the working muscle. In summary, cardiac output increases, and it is redistributed during heavy aerobic exercise so that tissues that need increased blood flow receive it and other tissues receive either equal or less blood flow.

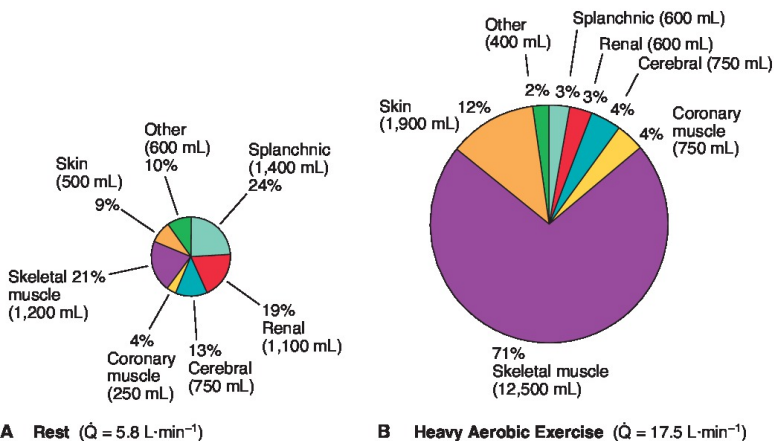


Figure 12.5 Distribution of Cardiac Output at Rest (A) and during Heavy Aerobic Exercise (B).

Source: Data from [Anderson \(1968\)](#).

Figure 12.6 presents data from a study in which participants exercised on a cycle ergometer for 8 minutes, then added an arm-cranking exercise ([Secher et al., 1977](#)). The combination of leg and arm cycling increased cardiac output modestly but actually caused a decrease in leg blood flow because a portion of cardiac output now had to be distributed to the working muscles in the arm.

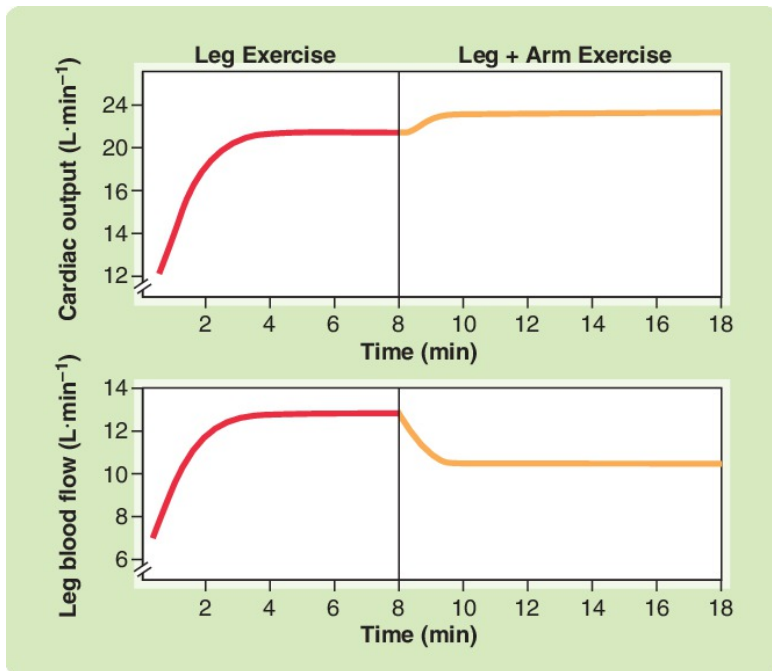


Figure 12.6 Effect of Combined Arm and Leg Exercise on Leg Blood Flow.

Source: Based on [Secher et al. \(1977\)](#).

Incremental Aerobic Exercise to Maximum

Figure 12.7 presents cardiovascular responses to incremental aerobic exercise to maximum. Note that unlike the graphs for light to moderate and heavy exercise (presented in [Figures 12.1](#) and [12.4](#)), the cardiovascular variables are now presented with percentage of maximum work on the x-axis. Incremental exercise to maximum (or a max test) consists of a series of work stages, each becoming progressively harder, that continue until volitional fatigue, which represents the maximal work an individual can do. The duration of each work stage (level of intensity) varies from 1 to 3 minutes to allow a steady state to occur, at least at the lower workloads. Max tests are performed in laboratory settings to quantify physiological responses to the maximal work that an individual can perform. Incremental tests to maximum may or

may not include the direct measurement of oxygen consumption. The techniques used in the direct measurement of oxygen consumption are presented in [Chapter 4](#).

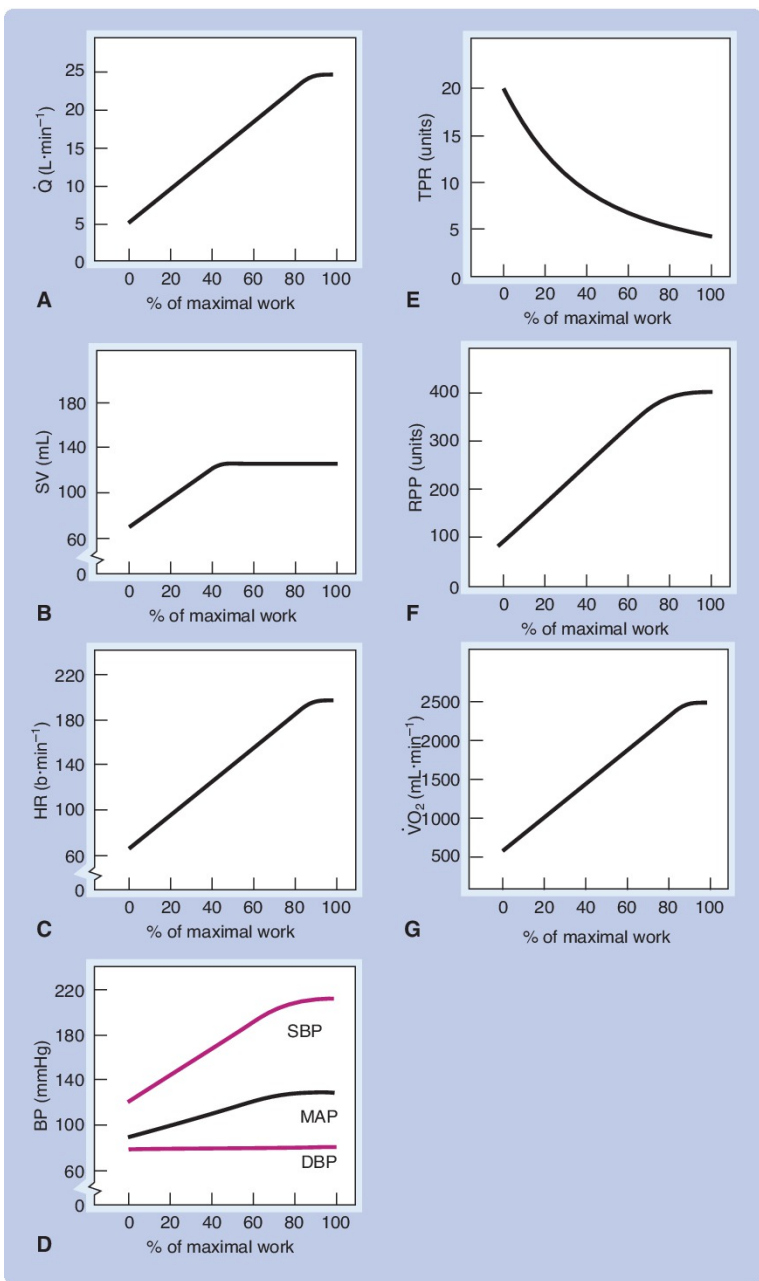


Figure 12.7 Cardiovascular Response to Incremental



Aerobic Exercise to Maximum.

A. Cardiac output (Q.). B. Stroke volume (SV). C. Heart rate (HR). D. Blood pressure (SBP, MAP, and DBP). E. Total peripheral resistance (TPR). F. Rate-pressure product (RPP). G. Oxygen consumption ($\dot{V}O_2$).

During an incremental test, cardiac output has a rectilinear increase and plateaus at maximal exercise (**Figure 12.7A**). The initial increase in cardiac output reflects an increase in the SV and the HR; however, at workloads greater than 40–50%

$\dot{V}O_{2\text{ max}}$, the continued increase in cardiac output in untrained individuals is achieved almost completely by an increase in the HR. As shown in **Figure 12.7B**, in untrained individuals, the SV increases rectilinearly initially and then

plateaus at approximately 40–50% of $\dot{V}O_{2\text{ max}}$ ([Åstrand et al., 1964](#); [Higginbotham et al., 1986](#)). The exact SV response to incremental exercise continues to be debated ([González-Alonso, 2008](#); [Munch et al., 2014](#); [Rowland, 2005a, 2005b](#); [Warburton and Gledhill, 2008](#)). As indicated above, it has traditionally been

believed that the SV plateaus at approximately 50% $\dot{V}O_{2\text{ max}}$ in untrained individuals. However, there appears to be considerable interindividual variability in this response, and many laboratories have reported an increase in the SV at maximal exercise in most endurance athletes and some untrained individuals ([Ferguson et al., 2001](#); [Gledhill et al., 1994](#); [Warburton et al., 1999](#)). In contrast, other researchers have documented a decrease in the SV at the maximal exercise ([Mortensen et al., 2005](#); [Stringer et al., 1997](#)), and some researchers contend that after an initial increase (due to the skeletal muscle pump returning the pooled venous blood to the heart), the SV remains essentially unchanged during the incremental maximal exercise ([Rowland, 2005b](#)). Much of the controversy is undoubtedly associated with difficulties in measuring the SV during maximal exercise (see [Chapter 11](#)), with the use of different exercise protocols, and with individual variability.

Theoretically, stroke volume could increase due to an increase in EDV, a decrease in ESV, or a combination of both. The EDV increases largely because of the return of blood to the heart by the active muscle pump and the increased sympathetic outflow to the veins causing venoconstriction and augmenting venous return. ESV decreases because of augmented contractility of the heart, which ejects more blood and leaves less in the ventricle. **Figure 12.8** presents data for left ventricular (LV)EDV and LVESV in athletic participants (mean age = 39 years) and in patients with heart failure (mean age = 69 years) using cardiac magnetic resonance imaging (cardiac MRI). These data suggest that the changes in ventricular volume during exercise are dependent upon health status and that changes in LVESV primarily account for the increase in SV during maximal exercise in healthy individuals ([La Gerche et al., 2013](#)).

FOCUS ON RESEARCH

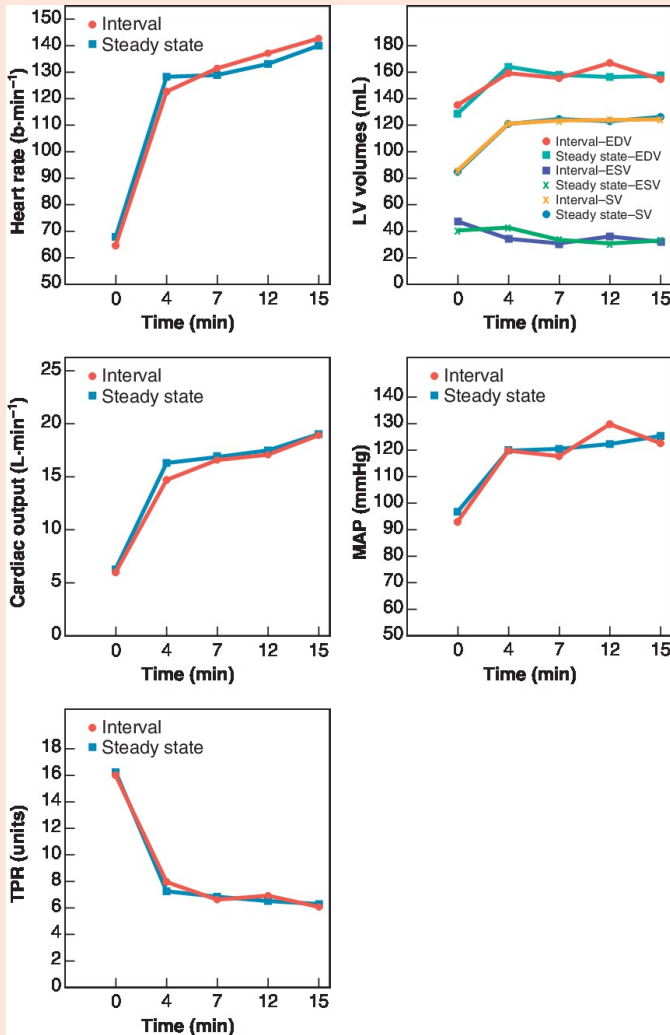
Interval Exercise versus Steady-State Exercise

Throughout this book, we examine the exercise response to various categories of exercise, such as the cardiovascular responses in this chapter. Most studies examining cardiovascular responses to exercise have used continuous activity. Yet, many clinical populations (e.g., people undergoing cardiac rehabilitation) and many athletic populations use interval training. Interval workouts generally use repetitions from several seconds to several minutes in length and intensities from light to very hard.

Foster et al. set out to determine the cardiovascular responses to continuous exercise compared to those of very short-term, high-intensity interval exercise when the total power output remained constant. A group of adults (mean age = 52.9 years) participated in two separate 15-minute cycling trials—one involving steady-state exercise and the other using interval exercise. Participants cycled at 170 W for

the full 15 minutes in one trial and alternated 1-minute “hard” (220 W) and “easy” (120 W) periods in the second trial, resulting in an equal power output (170 W) for both trials. Cardiovascular measurements were obtained before exercise (0 minute) and after minutes 4, 7, 12, and 15 and are presented in the accompanying graphs.

The results showed no significant difference in any of the variables between the steady-state exercise and the interval exercise. The authors concluded that heart function during interval exercise is remarkably similar to continuous steady-state exercise at the same average power output, when moderate duration and evenly timed hard and easy periods are used. These results are good news for individuals with low levels of fitness who may not be able to perform 15 minutes of continuous activity when starting an exercise program. Even if such individuals are unable to perform the high-intensity interval exercise used in this experiment, fitness professionals can assure them that alternating periods of “hard” and “easy” work results in cardiovascular responses similar to those resulting from sustained exercise of the same average power output.



Source: Foster, C., K. Meyer, N. Georgakopoulos, et al. Left ventricular function during interval and steady state exercise. *Medicine & Science in Sports & Exercise*. 31(8):1157-1162 (1999).

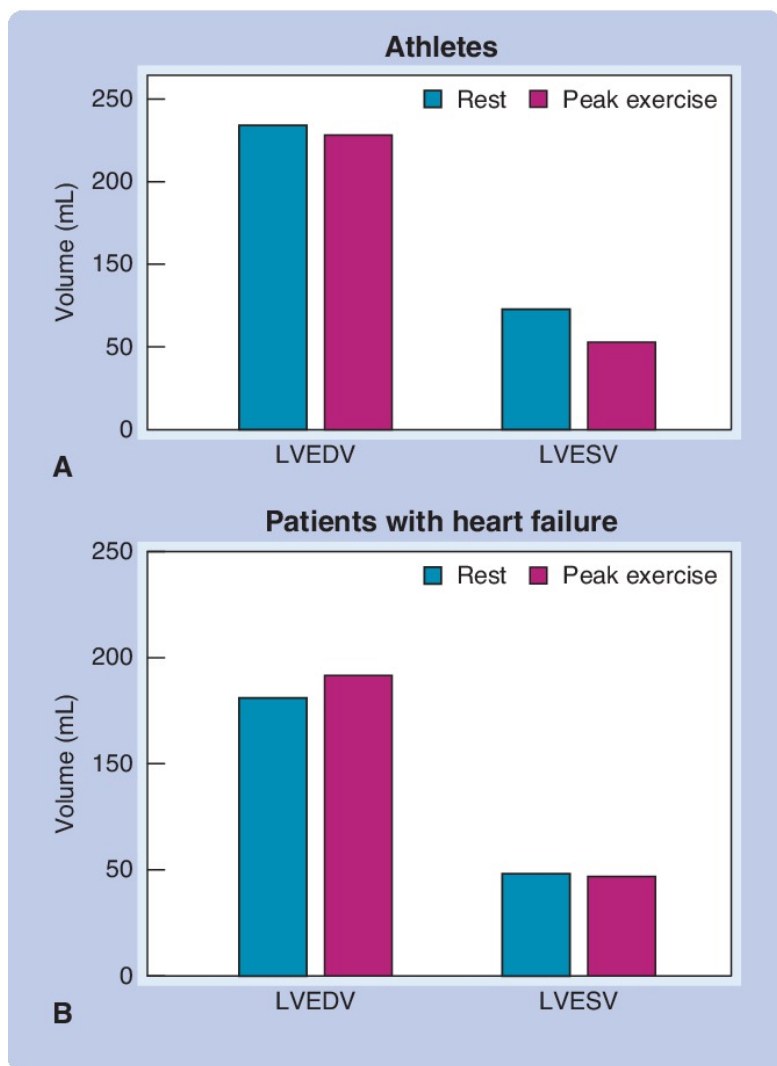


Figure 12.8 Changes in LVEDV and LVESV during Peak Exercise in Athletic Adults (A) and Patients with Heart Failure (B).

Source: Based on data from La Gerche et al. (2013).

HR increases in a rectilinear fashion throughout much of the submaximal ($\sim 120\text{--}170\text{ b}\cdot\text{min}^{-1}$) portion of incremental exercise and plateaus at maximal exercise (**Figure 12.7C**) ([Åstrand and](#)

Rhyming, 1954; Hale, 2008). Myocardial cells can contract at over 300 b·min⁻¹ but rarely exceed 210 b·min⁻¹ because a faster HR would not allow for adequate ventricular filling. Thus, SV and ultimately cardiac output would decrease. Consider the simple analogy of a bucket brigade. Up to a point, it is useful to increase the speed of passing buckets under the water source, but the maximum rate is limited because of the time required for the buckets to be filled with water.

The arterial BP responses to incremental dynamic exercise to maximum are shown in **Figure 12.7D**. SBP increases rectilinearly and plateaus at maximal exercise, often reaching values in excess of 200 mmHg in very fit individuals. This increase is caused by the increased cardiac output, which outweighs the simultaneous decrease in resistance. SBP and HR are routinely monitored during exercise tests to ensure the safety of the participant. If either of these variables fails to rise with an increasing workload, cardiovascular insufficiency and an inability to adequately perfuse tissue may result, and the exercise test should be stopped. A drop in SBP of 10 mmHg or more that occurs despite an increase in workload represents an absolute indication that the exercise test or session should be discontinued. Similarly, a rise in SBP above 250 mmHg or in DBP above 115 mmHg are relative indications (meaning they may be superseded by clinical judgment) that the exercise test or session should be discontinued because these are not normal blood pressure responses ([American College of Sports Medicine \[ACSM\], 2022](#)).

DBP typically remains relatively constant or changes so little it has no physiological significance, although it may decrease at high levels of exercise. Diastolic pressure remains relatively constant because vasodilation in the vasculature of the active muscle is balanced by vasoconstriction in other vascular beds. Diastolic pressure is most likely to decrease when exercise is performed in a hot environment because skin vessels are more dilated and there is decreased resistance to blood flow.

Individuals with an exaggerated BP response to exercise are at greater risk for hypertension, stroke, and cardiovascular disease mortality than those with a normal exercise BP response ([ACSM, 1993](#); [Jae et al., 2006](#); [Kurl et al., 2001](#); [Miyai et al., 2002](#); [Mundal et al., 1994](#)). See Focus on Research Box and work

through the **Check Your Comprehension 2** box to ensure understanding of this information.

CHECK YOUR COMPREHENSION 1—CASE STUDY 1

Janet has been making lifestyle decisions to improve her health. She has been taking yoga classes for about a year and decided to take part in a fitness assessment offered by the YMCA.

Participant Information

Age = 46 years

Ht = 5 ft, 8 in. Wt = 140 lb

Smoker = previous 2 packs per day; quit 5 years ago

Exercise: walks 30 minutes per day; 4–5 days per week for past 2 years; yoga 2 times per week

Medical history: unremarkable

Protocol: Janet performed a graded exercise test on a treadmill using a Bruce protocol. At 8 minutes into the test, the technician ended the test and the following information was recorded.

| | Preexercise | Immediately Postexercise |
|---------------------------------------|-------------|--------------------------|
| HR ($\text{b}\cdot\text{min}^{-1}$) | 90 | 146 |
| SBP (mmHg) | 138 | 260 |
| DBP (mmHg) | 88 | 120 |
| RPE (using Borg scale) | | 16 |

The data indicate that this was not a maximal exercise test. So, why did the technician stop the test? Check your answer in Appendix C.

FOCUS ON RESEARCH

Exaggerated Blood Pressure Responses and Carotid Atherosclerosis

It has been known for some time that an exaggerated blood pressure response to exercise, in otherwise apparently healthy individuals, is predictive of future hypertension, stroke, and cardiovascular disease mortality. In this study, researchers investigated the hypothesis that an exaggerated SBP response to incremental aerobic exercise to maximum was associated with carotid atherosclerosis (deposition of fatty plaque in the carotid artery) in middle-aged (~48 years) men.

The researchers used data from over 9,000 apparently healthy men who had undergone an exercise stress test and a series of cardiovascular tests. No participants had a history of hypertension or cardiovascular disease. Approximately 4% or 375 participants had an exaggerated SBP response to exercise (defined as $\text{SBP} > 210 \text{ mmHg}$). When the researchers compared the group that had an exaggerated SBP response to exercise to those who had a normal response, they found that individuals with an exaggerated SBP response to exercise had a 2.02 times increased risk for carotid atherosclerosis.

These results suggest that an exaggerated SBP response to exercise is independently related to an increased risk of carotid atherosclerosis.

Source: Jae, S. Y., B. Fernhall, K. S. Heffernan, et al. Exaggerated blood pressure response to exercise is associated with carotid atherosclerosis in apparently healthy men. *Journal of Hypertension*. 24:881–887 (2006).

TPR decreases in a negative curvilinear pattern and reaches its lowest level at maximal exercise (**Figure 12.7E**). Decreased resistance reflects maximal vasodilation in the active tissue in response to the need for increased blood flow during maximal exercise. As with other intensities of aerobic exercise, this vasodilation is mediated by local factors produced by muscle contraction and by the release of nitric oxide from endothelial

cells that causes smooth muscle relaxation in the vessel wall. The large drop in resistance is also important for keeping MAP from becoming too high. The rate-pressure product increases in a rectilinear fashion plateauing at maximum in an incremental exercise test (**Figure 12.7F**), paralleling the increases in HR and SBP.

The body responds to exercise in a coordinated and predictable way. Recall from [Chapter 11](#) that $MAP = \dot{Q} \times TPR$. Furthermore, $\dot{Q} = SV \times HR$. Work through the accompanying [Check Your Comprehension 3](#) box to see the magnitude of change in these variables and how they are related.

CHECK YOUR COMPREHENSION 3

The box below contains information on the HR and SV and obtained from a 30-year-old recreational athlete before and after a maximal treadmill test to volitional fatigue.

1. Calculate the and TPR.
2. Calculate the percent change in each variable with maximal exercise.
3. Is this a normal exercise response?

| | MAP (mmHg) | HR (b·min ⁻¹) | SV (mL) | \dot{Q} (L·min ⁻¹) | TPR (units) |
|--------------------------|---------------|------------------------------|------------|-------------------------------------|----------------|
| Preexercise | 90 | 68 | 75 | | |
| Immediately postexercise | 110 | 192 | 130 | | |
| Calculate percent change | | | | | |

Check your answer in Appendix C.

The reduction in plasma volume during submaximal exercise also occurs in incremental exercise to maximum. Because the magnitude of the reduction depends on the intensity of exercise,

the reduction is greatest at maximal exercise. A decrease of 10–20% can be seen during incremental exercise to maximum (Wade and Freund, 1990).

Considerable changes in cardiac output occur during maximal incremental exercise. **Figure 12.9** illustrates the distribution of cardiac output at rest and at maximal aerobic exercise. Maximum cardiac output in this example is 25 L·min⁻¹. Again, the most striking change is the tremendous amount of cardiac output that is directed to the working muscles (88%). At maximal exercise, skin blood flow is reduced to direct the necessary blood to the muscles. Renal and splanchnic blood flows also decrease considerably. Blood flow to the brain and cardiac muscle is maintained.

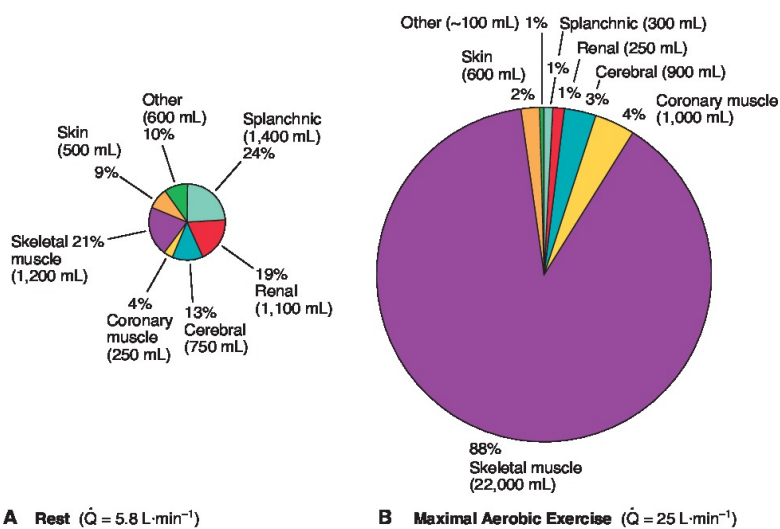


Figure 12.9 Distribution of Cardiac Output at Rest (A)

and during Maximal Aerobic Exercise (B).

Source: Data from Anderson (1968).



Table 12.1 summarizes the cardiovascular responses to exercise.

TABLE 12.1 Cardiovascular Responses to Exercise*

| | Short-Term, Light to Moderate Submaximal Aerobic Exercise | Long-Term, Moderate to Heavy Submaximal Aerobic Exercise [†] | Incremental Aerobic Exercise to Maximum | Static [‡] Exercise | Resistance [‡] Exercise |
|-----|---|---|--|---|--|
| Q | Increases rapidly; plateaus at steady state within 2 min | Increases rapidly; plateaus | Rectilinear increase with plateau at max | Modest gradual increase | Modest gradual increase |
| SV | Increases rapidly; plateaus at steady state within 2 min | Increases rapidly; plateaus; negative drift | Increases initially; plateaus at 40–50% $\dot{V}O_2$ max | Relatively constant at low workloads; decreases at high workloads; rebound rise in recovery | Little change, slight decrease |
| HR | Increases rapidly; plateaus at steady state within 2 min | Increases rapidly; plateaus; positive drift | Rectilinear increase with plateau at max | Modest gradual increase | Increases gradually with numbers of reps |
| SBP | Increases rapidly; plateaus at steady state within 2 min | Increases rapidly; plateaus; slight negative drift | Rectilinear increase with plateau at max | Marked steady increase | Increases gradually with numbers of reps |
| DBP | Shows little or no change | Shows little or no change | Shows little or no change | Marked steady increase | No change or increase |
| MAP | Increases rapidly; plateaus at steady state within 2 min | Increases initially; little if any drift | Small rectilinear increase | Marked steady increase | Increases gradually with numbers of reps |
| TPR | Decreases rapidly; plateaus | Decreases rapidly; plateaus; slight negative drift | Curvilinear decrease | Decreases | Slight increase |
| RPP | Increases rapidly; plateaus at steady state within 2 min | Increases rapidly; plateaus; positive drift | Rectilinear increase with plateau at max | Marked steady increase | Increases gradually with numbers of reps |

*Resting values are taken as baseline.

†The difference between a plateau during the short-term, light to moderate and long-term, moderate to heavy submaximal exercise response is one of magnitude, that is, a plateau occurs at a higher value with higher intensities.

‡The magnitude of a plateau change depends on the %MVC/load.

Maximal Oxygen Consumption

Oxygen consumption rises rectilinearly in direct proportion to the exercise intensity during an incremental exercise to maximum (**Figure 12.7G**). The highest amount of oxygen an individual can take in, transport, and utilize to produce ATP aerobically while breathing air during heavy exercise is called **maximal oxygen**

consumption ($\dot{V}O_2 \text{ max}$). As described above, the maximal oxygen intake is measured during an incremental maximal

exercise test. As described in **Chapter 11**, $\dot{V}O_2 \text{ max}$ can be defined by rearranging Fick's equation (see Equation 11.13) to

the following equation:

Maximal Oxygen Consumption ($\dot{V}O_2 \text{ max}$) The highest amount of oxygen an individual can take in, transport, and utilize to produce ATP aerobically while breathing air during heavy exercise.

$$\dot{V}O_2 \text{ max} = \dot{Q} \text{ max} \times (a-vO_2 \text{ diffmax})$$

The rectilinear increase in cardiac output during a maximal incremental exercise test is described above. The changes in the $a-vO_2 \text{ diff}$ (discussed in [Chapter 10](#)) are an increase with a plateau at approximately 60% $\dot{V}O_2 \text{ max}$. The result of these changes is the rectilinear rise in oxygen up to maximum, which has been discussed.

Maximal exercise tests that include the measurement of oxygen consumption for the determination of $\dot{V}O_2 \text{ max}$ are routinely administered by coaches and trainers to determine an athlete's fitness or to track changes in fitness, by researchers to better understand the mechanisms that limit exercise or to probe questions related to physiological function under stressful conditions, and by medical personnel to assess cardiorespiratory function. The criteria for determining if an individual reaches maximal oxygen consumption during an exercise test are discussed in [Chapter 4](#).

$\dot{V}O_2 \text{ max}$ is commonly used as the criterion measure of cardiorespiratory (also called aerobic) fitness. In reality, $\dot{V}O_2 \text{ max}$ is an integrated measure of fitness that encompasses the ability of the body to take in (respiratory system), transport (cardiovascular system), and utilize (the metabolic system) oxygen. Thus, $\dot{V}O_2 \text{ max}$ may be considered a cardiovascular, respiratory, and metabolic variable, and, indeed, for that reason, it is also discussed in other chapters. However, $\dot{V}O_2 \text{ max}$ has important implications for cardiovascular health and is normally thought to be limited by cardiovascular function; therefore, we

will consider $\dot{V}O_2 \text{ max}$ primarily as a cardiovascular variable, and it is discussed in depth in this chapter.

FACTORS LIMITING At some point, an individual cannot continue to increase the intensity of the exercise load or to work at maximum effort because the body cannot provide and utilize more oxygen to support an additional workload. But what specifically limits ? **Figure 12.10** summarizes possible limitations to oxygen consumption within the major systems involved in oxygen delivery and use during exercise.

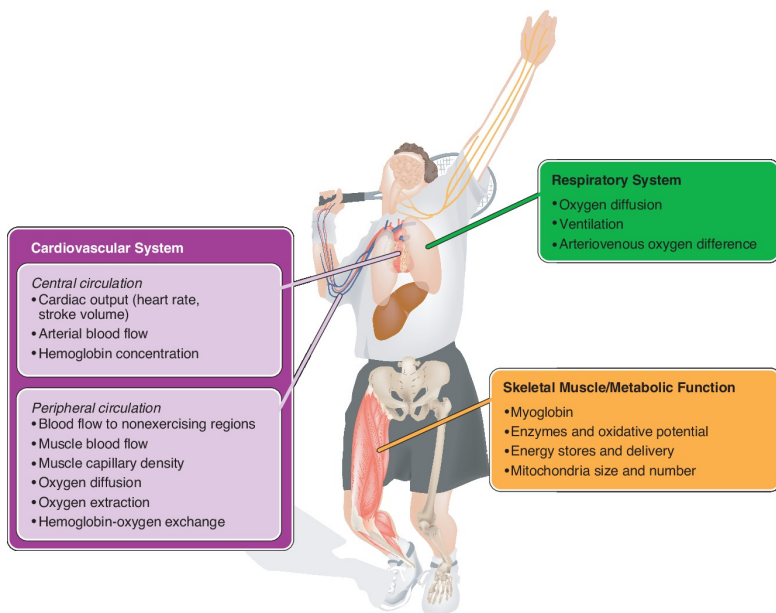


Figure 12.10 Possible Limitations to Maximal Oxygen

Consumption. 

Source: Modified from Rowell (1993).

Theoretically, maximal oxygen uptake could be limited by any system (or step) along the pathway of bringing oxygen into the body and delivering it to the mitochondria for the production of ATP. Thus, any of the following systems may limit $\dot{V}O_2 \text{ max}$.

1. The respiratory system, because of inadequate ventilation, oxygen diffusion limitations, or an inability to maintain the gradient for the diffusion of O₂ (a-vO₂diff)
2. The cardiovascular system, because of inadequate blood flow (\dot{Q}) or oxygen-carrying capacity (Hb)
3. The metabolic functions within skeletal muscle, such as an inability to produce additional ATP because of limited number of mitochondria, limited enzyme levels or activity, or limited substrates

Evidence suggests that each of these systems may limit $\dot{V}O_2 \text{ max}$ in certain conditions (Bergh et al., 2000). For example, a reduction in the partial pressure of oxygen (PO₂) at altitude or with asthma causes a reduction in $\dot{V}O_2 \text{ max}$. Medications (such as beta-blockers) that limit cardiac output also cause a decrease in $\dot{V}O_2 \text{ max}$, as does a reduction in hemoglobin associated with anemia. Certain diseases in which muscle enzymes involved in metabolism are deficient can also result in reduced $\dot{V}O_2 \text{ max}$.

Although factors in each of these systems may limit $\dot{V}O_2 \text{ max}$, the question remains: What limits $\dot{V}O_2 \text{ max}$ in healthy humans performing maximal exercise? This question has energized exercise physiologists for decades, beginning with the work of A. V. Hill in the 1920s, and it continues to engender lively debate among physiologists. Current research suggests that maximal oxygen uptake is limited by the ability of the cardiorespiratory system to deliver oxygen to the muscle, rather than the ability of the muscle mitochondria to utilize oxygen (Bassett and Howley, 2000; Bergh et al., 2000; Elliott et al., 2015; Ferretti, 2014; Grassi, 2000; Hale, 2008). Specifically, cardiac output appears to be the limiting factor in $\dot{V}O_2 \text{ max}$ (Bergh et al., 2000; di Prampero, 2003; Saltin, 1985).

Research evidence suggests that oxygen uptake is not limited by pulmonary ventilation in normal, healthy athletes without exercise-induced arterial hypoxemia (Chapter 10). Generally, the functional capacity of the respiratory system is believed to exceed the demands of maximal exercise (Rowell, 1993). The only

respiratory or cardiovascular variable likely to impose a limitation on oxygen transport is $a-vO_2\text{diff}$.

Many researchers report that skeletal muscles have the ability to use more oxygen than can be supplied by the respiratory and cardiovascular systems (Richardson, 2000; Rowell, 1993; Saltin, 1985). Not all researchers agree with this view, though, and some have proposed that failure of muscle performance may explain exhaustion during maximal exercise (Noakes, 1988).

Possibly, the factors limiting $\dot{V}O_2 \text{ max}$ vary with the fitness level of the individual. According to this hypothesis, in an untrained individual, the respiratory capacity for gas exchange exceeds the cardiovascular system's capacity to deliver oxygen. A training program results in little change in the respiratory capacity but large changes in the cardiovascular capacity. Thus, in some highly trained individuals who have exercise-induced arterial hypoxemia (Chapter 10), the increased cardiovascular capacity may exceed the respiratory capacity (Dempsey, 1986; Legrand et al., 2005; Powers et al., 1989). In this case, the respiratory system becomes the factor limiting $\dot{V}O_2 \text{ max}$. One final point to remember, although it is interesting to probe the question, "what limits $\dot{V}O_2 \text{ max}$?" we must resist the temptation to allow the search for an answer to obscure the fact that a close interaction exists among the various systems ensuring a continuous supply of oxygen to the working tissue during exercise (Mitchell and Saltin, 2003).

High-Intensity Interval Exercise

High-intensity interval exercise is a type of maximal exercise in which an individual performs short bouts of intense exercise interspersed with active recovery. For example, an individual may sprint for 20 seconds at a pace that equals their pace at $\dot{V}O_2 \text{ max}$ and then walk for one to two minutes and perform another 20-second sprint for 6–8 cycles. Because the exercise bouts are so short, there is very limited data on acute exercise responses to high-intensity interval exercise, but it could be inferred that values will mimic those achieved near the end of an incremental exercise to maximum because of the intensity of the

exercise. While exercise responses to high-intensity interval training are seldom reported, there is a great deal of information emerging on the training adaptations to this type of exercise.

Upper-Body versus Lower-Body Aerobic Exercise

Upper-body exercise is routinely performed in a variety of industrial, agricultural, military, recreational, and sporting activities. The cardiovascular responses to exercise using muscles of the upper body are different in some important ways from exercise performed using muscles of the lower body. **Figure 12.11** presents data about cardiovascular responses to incremental exercise to maximum in able-bodied individuals using the upper body (arm cranking on an arm ergometer) versus lower body (cycling on a cycle ergometer). Notice that a higher peak $\dot{V}O_2$ was achieved during lower-body exercise. Comparisons at any given level of oxygen consumption also show differences in cardiovascular responses to submaximal upper- and lower-body exercise. When the oxygen consumption required to perform a submaximal workload is the same, cardiac output is similar for upper- and lower-body exercise (**Figure 12.11A**). However, the mechanism to achieve the required increase in cardiac output is not the same. As shown in **Figure 12.11B and C**, upper-body exercise results in a lower SV and a higher HR at any given submaximal workload (Clausen, 1976; Miles et al., 1989; Pendergast, 1989). SBP, DBP, MAP (**Figure 12.11D**), total peripheral resistance (**Figure 12.11E**), and rate-pressure product (**Figure 12.11F**) are significantly higher in upper-body exercise than in lower-body exercise performed at the same oxygen consumption.

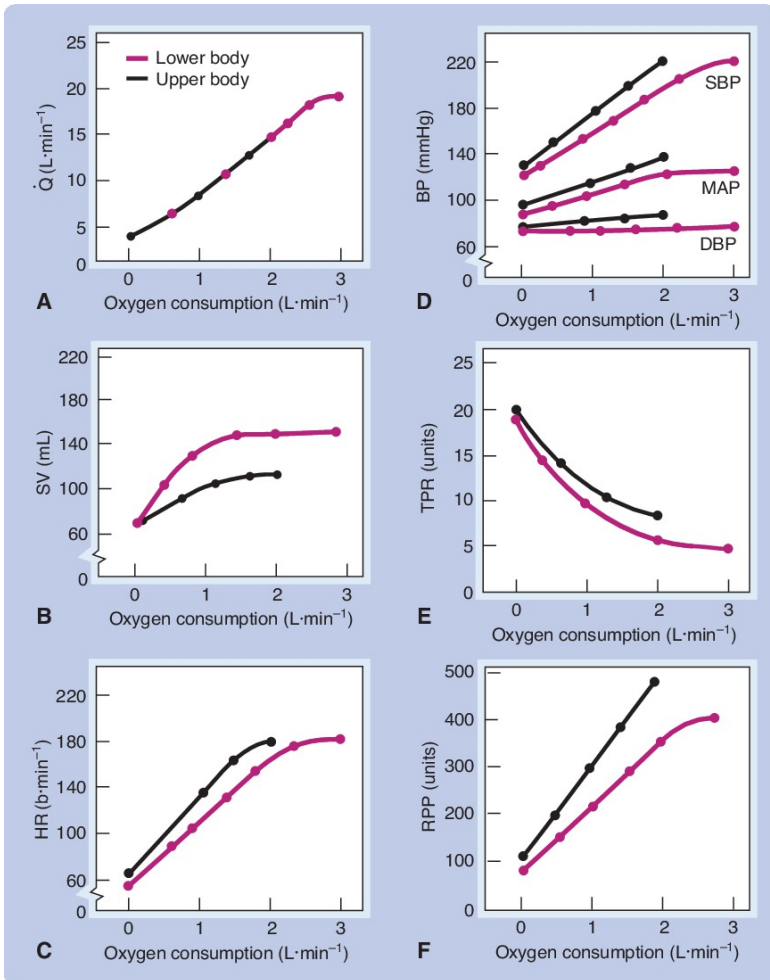


Figure 12.11 Cardiovascular Response to Incremental Aerobic Maximal Upper-Body and Lower-Body Exercise.



A. Cardiac output (\dot{Q}). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Blood pressure (SBP, MAP, and DBP). **E.** Total peripheral resistance (TPR). **F.** Rate-pressure product (RPP).

There are several likely reasons for the differences. The higher HR observed during upper-body exercise is thought to reflect a

greater sympathetic stimulation (Åstrand et al., 2003; Davies et al., 1974; Miles et al., 1989). SV is lower during upper-body exercise because of the absence of the skeletal muscle pump augmenting venous return from the legs. The greater sympathetic stimulation that occurs during upper-body exercise may also be partially responsible for the increased BP and total peripheral resistance. Upper-body exercise often involves a static component that causes an exaggerated BP response. For instance, using an arm-cranking ergometer has a static component because the individual must grasp the hand crank.

When maximal exercise is performed using upper-body muscles, $\dot{V}O_2 \text{ max}$ values are approximately 30% lower than when maximal exercise is performed using lower-body muscles (Miles et al., 1989; Pendergast, 1989). Maximal HR values for upper-body exercise are 90–95% of those for lower-body exercise, and SV is 30–40% less during maximal upper-body exercise. Maximal SBP and the rate-pressure product are usually similar, but DBP is typically 10–15% higher during upper-body exercise (Miles et al., 1989).

The different cardiovascular responses to an absolute workload performed with the upper body versus the lower body dictate that exercise prescriptions for arm work cannot be based on data obtained from testing with leg exercises. Furthermore, the greater cardiovascular strain associated with upper-body work must be kept in mind when one prescribes exercise for individuals with cardiovascular disease. Complete the [Check Your Comprehension 4](#) to confirm your understanding of physiological responses to arm versus leg exercise.

FOCUS ON APPLICATION | *Clinically Relevant*

Cardiovascular Demands of Shoveling Wet, Heavy Snow

As the text has described, upper-body exercise is associated with greater cardiovascular strain (exemplified by higher HRs

and higher BPs at any given submaximal level of oxygen consumption) than lower-body exercise. Similarly, both static and dynamic resistance exercise are characterized by modest increases in HR but exaggerated increases in BP. Snow shoveling involves a unique combination of a predominantly upper-body activity with both static and dynamic components. In addition, snow shoveling is always done in cold and sometimes frigid conditions and often when the individual is under the added stress of digging out to get somewhere on time. It is not unusual to hear about individuals collapsing and dying of heart attacks while clearing snow.

In a classic study, Franklin et al. performed an experiment to determine the specific demands of snow shoveling on the heart. Ten sedentary, healthy, young adult males cleared two 15-m paths of wet, heavy snow 5–13 cm deep outside in the cold (2°C) for 10 minutes. In one trial, they used a 1.4-kg plastic shovel. They were told to repeatedly lift and throw the snow to the side at a self-selected rate. The group mean was 12 ± 2 loads per minute at approximately 7.3 kg per load for a total of 872.7 kg (1,920 lb) over the 10-minute time span. In the second trial, they used a motorized snow blower. Ten to fifteen minutes of rest was permitted between the randomly assigned trials. On another day, each participant underwent a treadmill maximal oxygen consumption test in the laboratory. The results are presented in the accompanying table.

After only 2 minutes of snow shoveling, the participants' average HR was 85% of the treadmill HR_{max}. The HR continued to increase until it reached 98% HR_{max}. The SBP during the snow shoveling exceeded the treadmill maximum by 9.3%. The total body oxygen consumption was only 61.3%

VO₂ max, but the myocardial oxygen consumption, as indicated by the rate-pressure product, was 107% of that required during maximal treadmill work. The disproportionate increase in myocardial oxygen demand relative to total body oxygen demand during shoveling was attributed to several factors: a reduced myocardial efficiency of arm exercise, a large static exercise component, the

Valsalva maneuver, and the inhalation of cold air that could cause a spasm or constriction in the coronary arteries. These results clearly indicate that shoveling wet, heavy snow even for a short time period (10 minutes) places a tremendous physiological demand on the heart.

In contrast, using the snow blower resulted in elevations to only 69% HRmax, 89% maximal SBP, 25% $\dot{V}O_2$ max and 61% maximal myocardial oxygen consumption, as reflected by the rate-pressure product. Whereas the manual shoveling was perceived as “very heavy” work, using the snow blower resulted in a “fairly light” rating.

The healthy but untrained participants in this study completed the shoveling without any adverse cardiac or musculoskeletal complications. Such work, especially if continued for 20–60 minutes, would provide a heavy but acceptable workout. However, these results suggest that individuals with a history of heart disease, symptoms suggestive of cardiac disorder (dizziness, chest pain, and abnormal electrocardiograms), or one or more major coronary risk factors (see [Chapter 15](#)) should avoid this work or take precautions when faced with the task of clearing wet, heavy snow. These precautions include the following:

- Take frequent breaks or use a work-rest approach.
- Use both arms and legs in the lift-throw action.
- Regulate body temperature with a hat, scarf over the mouth, and layers that can easily be added or removed.
- Avoid large meals, alcohol consumption, and smoking immediately before and after shoveling.
- Consider using a motorized snow blower.



Sources: Franklin (1997); Franklin et al. (1995).

| Variable | Snow Shoveling | Snow Blower | Treadmill (Max) |
|--|----------------|-------------|-----------------|
| HR (b·min ⁻¹) | 175 ± 15 | 124 ± 18 | 179 ± 17 |
| SBP (mmHg) | 198 ± 17 | 161 ± 14 | 181 ± 25 |
| Rate-pressure product | 347 | 199.6 | 324 |
| $\dot{V}O_2$ (mL·kg ⁻¹ ·min ⁻¹) | 19.9 ± 2.8 | 8.4 ± 2.5 | 32.5 ± 6.3 |
| Rating of perceived exertion | 16.7 ± 1.7 | 9.9 ± 1.0 | 17.9 ± 1.5 |

CHECK YOUR COMPREHENSION 4

Kara has been working out at the YMCA for over a year. Her typical work includes 15 minutes of stair climbing and 15 minutes of treadmill walking at an estimated 60% of HR_{max} (or 100 b·min⁻¹). Kara is interested in adding additional modalities to her workout routine and is considering using the newly purchased arm crank ergometer. Is it appropriate for Kara to work out on the arm ergometer at an intensity that elicits a HR of 100 b·min⁻¹? Why or why not?

Check your answer in Appendix C.

Cardiovascular Responses to Static Exercise

Static work occurs repeatedly during daily activities, such as lifting and carrying heavy objects. It is also a common form of activity encountered in many occupational settings, particularly manufacturing jobs where lifting is common. Additionally, many sports and recreational activities have a static component associated with their performance. For example, weight lifting, rowing, and racquet sports all involve static exercise. The magnitude of the cardiovascular response to static exercise is affected by several factors, but most importantly by the intensity of muscle contraction.

Intensity of Muscle Contraction

The cardiovascular response to static exercise depends on the intensity of contraction, provided the contraction is held for a specified time period. The intensity of a static contraction is expressed as a percentage of maximal voluntary contraction (%MVC). **Figure 12.12** illustrates the cardiovascular response to static contractions of the forearm (handgrip) muscles at 10, 20, and 50% MVC. Notice that at 10 and 20% MVC, the contraction could be held for 5 minutes, but at 50% MVC, the contraction could be held for only 2 minutes. Thus, as in aerobic exercise, intensity and duration are inversely related. Also note that the data presented in this figure are from handgrip exercises. Although the pattern of response appears to be similar for different muscle groups, the actual values may vary considerably depending on the amount of active muscle involved.

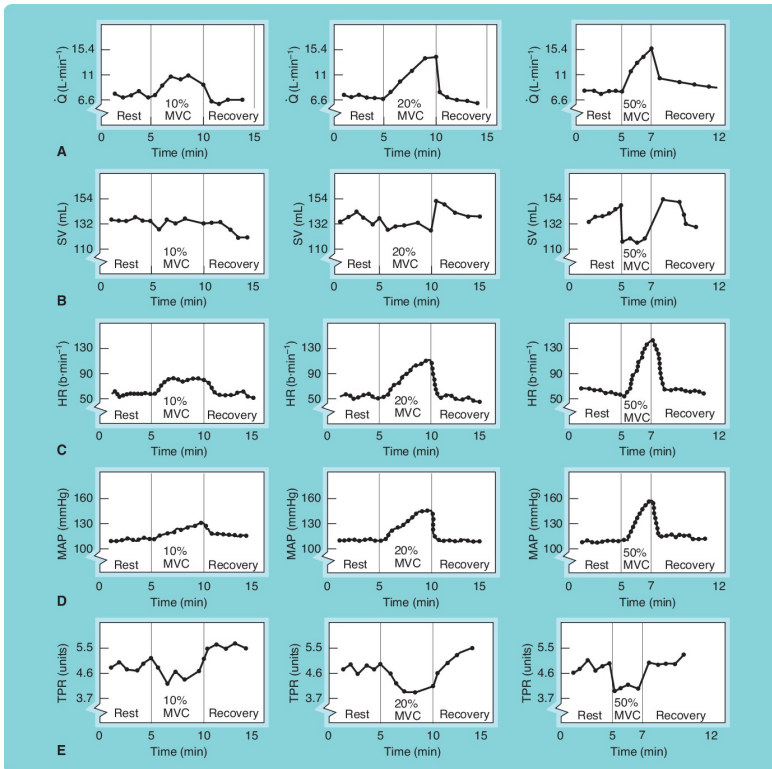


Figure 12.12 Cardiovascular Responses to Varying



Intensities of Static Handgrip Exercise.

A. Cardiac output (\dot{Q}). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Mean arterial pressure (MAP). **E.** Total peripheral resistance (TPR).

Source: Modified with permission of Portland Press, Ltd. from Lind, A. R., S. H. Taylor, P. W. Humphreys, B. M. Kennelly, & K. W. Donald: The circulatory effects of sustained voluntary muscle contraction. *Clinical Science*. 27:229–244 (1964); permission conveyed through Copyright Clearance Center, Inc.

Cardiac output increases during static contractions due to an increase in HR, with the magnitude of the increase dependent on

the intensity of exercise. SV (**Figure 12.12B**) remains relatively constant or decreases slightly during low-intensity contractions and decreases during high-intensity contractions. There is a marked increase in SV immediately following the cessation of high-intensity contractions ([Lind et al., 1964](#); [Smith et al., 1993](#)). This is the same rebound rise in recovery as seen in a-vO₂diff,

minute ventilation (\dot{V}_E), and $\dot{V}O_2$ (see **Figure 4.5**). The reduction in SV during high-intensity contractions probably results from both a decreased preload and an increased afterload. Preload is decreased because of high intrathoracic pressure, which compresses the vena cava and thus decreases the return of venous blood to the heart. Because arterial BP is markedly elevated during static contractions (increased afterload), less blood is ejected at a given force of contraction. HR (**Figure 12.12C**) increases during static exercise. The magnitude and the rate of the increase in HR depend on the intensity of contraction. The greater the intensity, the greater the HR response.

Static exercise is characterized by a rapid increase in both systolic pressure and diastolic pressure, termed the **pressor response**, which appears to be inappropriate for the amount of work produced by the contracting muscle ([Lind et al., 1964](#)). Since both systolic and diastolic pressures increase, there is a marked increase in MAP (**Figure 12.12D**) ([Donald et al., 1967](#); [Lind et al., 1964](#); [Seals et al., 1985](#); [Tuttle and Horvath, 1957](#)). As in any muscular work, static exercise increases metabolic demands of the active muscle. However, in static work, high intramuscular tension results in mechanical constriction of the blood vessels, which impedes blood flow to the muscle. The reduction in muscle blood flow during static exercise results in a buildup of local by-products of metabolism. These chemical by-products (H⁺, adenosine diphosphate, and others) stimulate sensory nerve endings, which leads to a pressor reflex, causing a rise in MAP (pressor response). This rise is substantially larger than the increase during aerobic exercise requiring similar energy expenditure ([Asmussen, 1981](#); [Hanson and Nagle, 1985](#)). Notice in **Figure 12.12D** that holding a handgrip dynamometer at 20% MVC for 5 minutes resulted in an increase of 20–30 mmHg in MAP and holding 50% MVC for 2 minutes caused a 50 mmHg increase in MAP!

Pressor Response The rapid increase in both systolic pressure and diastolic pressure during static exercise.

Total peripheral resistance, indicated by TPR in **Figure 12.12E**, decreases during static exercise, although not to the extent seen in dynamic aerobic exercise. The smaller decrease in resistance helps to explain the higher BP response to static contractions. The high BP generated during static contractions helps overcome resistance to blood flow from mechanical occlusion. Because the SBP and the HR both increase during static exercise, there is a large increase in myocardial oxygen consumption and thus rate-pressure product. This is often a concern in clinical populations such as cardiac rehabilitation patients.

Work through the accompanying **Check Your Comprehension Box 5** to see the magnitude of change in key cardiovascular variables following static exercise and how they are related.

Table 12.1 summarizes cardiovascular responses to static exercise.

CHECK YOUR COMPREHENSION 5

The box below contains information on the HR and SV and obtained from a 70-year-old research participant before and after a 2-minute maximal handgrip exercise.

1. Calculate the Δ and TPR.
2. Calculate the percent change in each variable with maximal exercise.
3. Is this a normal response to static exercise?

| | MAP (mmHg) | HR (b·min ⁻¹) | SV (mL) | Q̇ (L·min ⁻¹) | TPR (units) |
|--------------------------------|---------------|------------------------------|------------|------------------------------|----------------|
| Preexercise | 110 | 68 | 75 | | |
| Immediately postexercise | 150 | 90 | 80 | | |
| Calculate percent change | | | | | |

4. Why does the magnitude of change differ from incremental aerobic exercise to maximum?

Check your answer in Appendix C.

Blood Flow during Static Contractions

Blood flow to the working muscle is impeded during static contractions because of the mechanical constriction of the blood vessel supplying the contracting muscle (Freund et al., 1979; Sjogaard et al., 1988). **Figure 12.13** depicts blood flow in the quadriceps muscle when a 5 and 25% MVC contraction were held to fatigue. The 5% MVC load could be held for 30 minutes; the 25% load could be held for only 4 minutes. Quadriceps blood flow is greater during the 5% MVC, suggesting that at 25% MVC, there is considerable impedance to blood flow. In fact, blood flow during the 25% MVC load was very close to resting levels despite the metabolic work done by the muscle. The response occurring during recovery suggests that when contraction ceases, a mechanical occlusion to the muscle is released. The marked increase in blood flow during recovery compensates for the reduced flow during sustained contraction. The relative force at which blood flow is impeded varies greatly among different muscle groups (Lind and McNichol, 1967; Rowell, 1993).

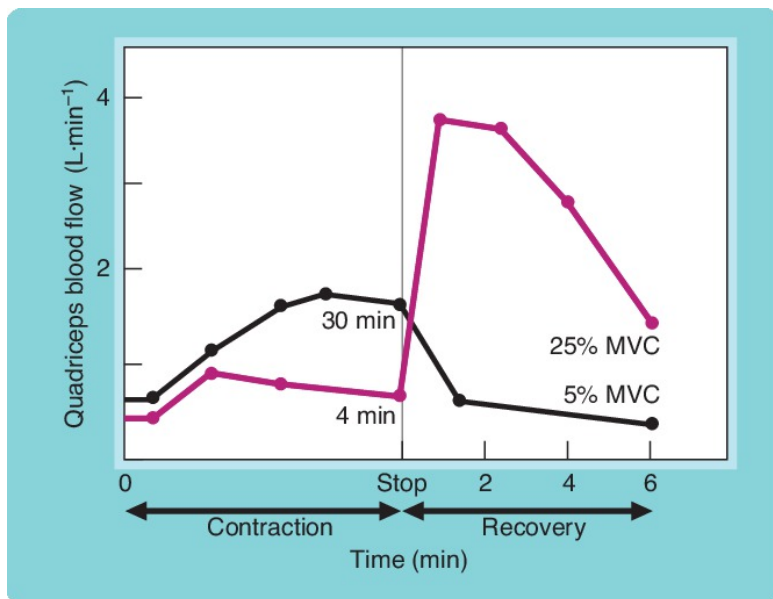


Figure 12.13 Blood Flow in the Quadriceps Muscle

during Different Intensities of Static Contraction.



Source: Adapted by permission from Springer Sjogaard, G., G. Savard, & C. Juel: Muscle blood flow during isometric activity and its relation to muscle fatigue. *European Journal of Applied Physiology and Occupational Physiology*. 57(3):327–335 (1988). Copyright © 1988 Springer Nature.

Mechanical constriction also occurs during dynamic aerobic exercise. However, the alternating periods of muscular contraction and relaxation during rhythmical activity allow—and, indeed, encourage—blood flow, especially through the venous system.

Comparison of Aerobic and Static Exercise

Figure 12.14 compares the HR and the BP responses to fatiguing handgrip (static) exercise (30% MVC held to fatigue) and a maximal treadmill (incremental aerobic) test to fatigue. The

incremental aerobic exercise is characterized by a large increase in the HR, which contributes to an increased cardiac output. The treadmill exercise response also shows a modest increase in the SBP and a relatively stable or decreasing DBP. Aerobic exercise is said to impose a “volume load” on the heart. Increased venous return leads to increased SV, which contributes to an increased cardiac output. In contrast, fatiguing static exercise is characterized by a modest increase in the HR, but a dramatic increase in the BP (*pressor response*). Mean BP increases as a result of increased SBP and DBP. Static exercise is said to impose a “pressure load” on the heart. Increased MAP means that the heart must pump harder to overcome the pressure in the aorta.

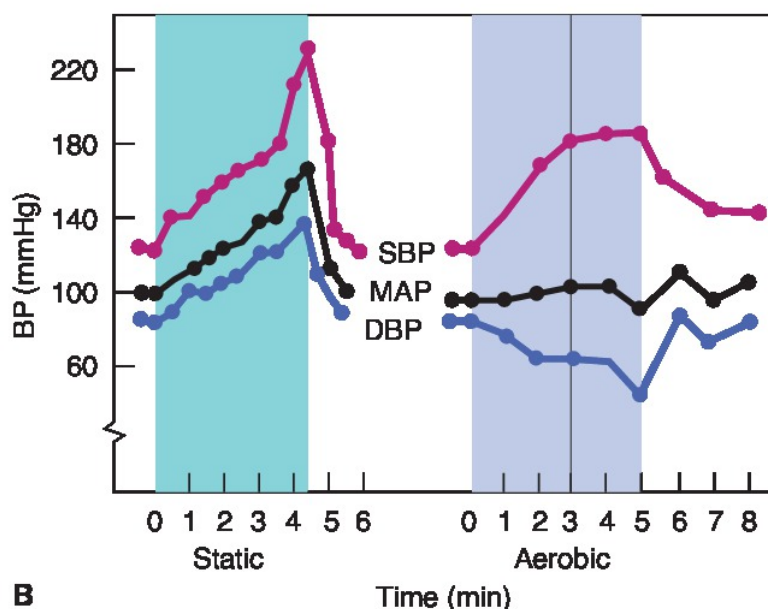
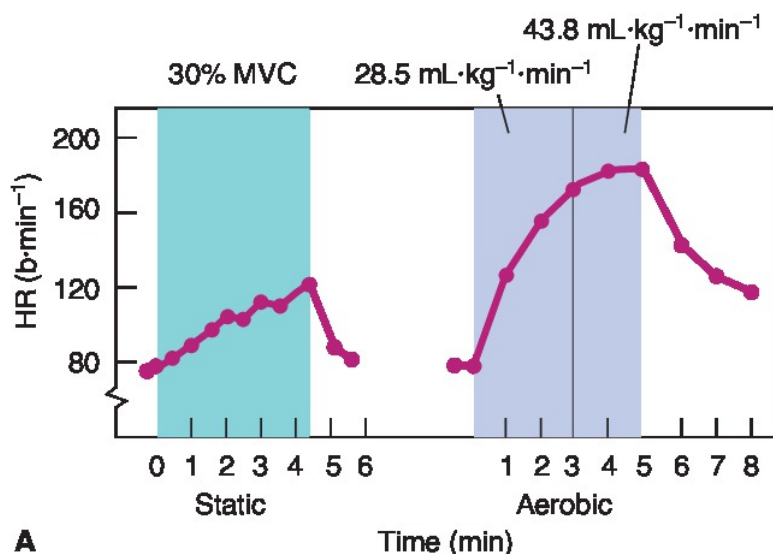


Figure 12.14 Comparison of HR (A) and BP (B) Response to Static and Incremental Aerobic Exercise to Maximum.



Source: Data from Lind and McNichol (1967).

Cardiovascular Responses to Dynamic Resistance Exercise

Weight-lifting or resistance exercise includes a combination of dynamic and static contractions (Hill and Butler, 1991; MacDougall et al., 1985). At the beginning of the lift, a static contraction exists until muscle force exceeds the load to be lifted and movement occurs, leading to a dynamic concentric (shortening) contraction as the lift continues. This is then followed by a dynamic eccentric (lengthening) contraction during the lowering phase (McCartney, 1999). A static component is always associated with gripping the barbell. During dynamic resistance exercise, cardiorespiratory system responses are dissociated from the energy demand. In contrast, during dynamic endurance activity, responses in the cardiorespiratory system are directly related to the use of oxygen for energy production. In part, the reason for this dissociation between oxygen use and cardiovascular response to resistance exercise is that much of the energy required for resistance exercise comes from anaerobic (without oxygen) sources. Another important difference between resistance exercise and aerobic exercise is the mechanical constriction of blood flow during resistance exercise because of the static nature of part of the contraction.

The magnitude of the cardiovascular response to resistance exercise depends on the intensity of the load (the weight lifted) and the number of repetitions performed. Cardiovascular responses also depend on how the load and repetitions are combined.

Varying Load/Constant Repetitions

As expected, cardiovascular responses are greater when heavier loads are lifted, assuming the number of repetitions is constant (Fleck, 1988; Fleck and Dean, 1987). For example, as shown in **Figure 12.15**, when participants performed 10 repetitions of arm

curling exercises with dumbbells of three different weights (identified as light, moderate, and heavy), the SBP was highest at the completion of the heaviest set (Wescott and Howes, 1983). The SBP increased 16, 22, and 34% during the light, moderate, and heavy sets, respectively. The DBP, measured by auscultation, did not change significantly with any of the sets. There is disagreement about the DBP response to resistance exercise; some authors report an increase, while others report no change (Fleck, 1988; Fleck and Dean, 1987; Wescott and Howes, 1983). In a study investigating blood pressure responses to 12 consecutive rounds of 2-hand kettlebell exercises, diastolic blood pressure did not change with exercise (Wong et al., 2021). These discrepancies may reflect differences in measurement techniques (auscultation vs. intra-arterial assessment) and timing of the measurement.

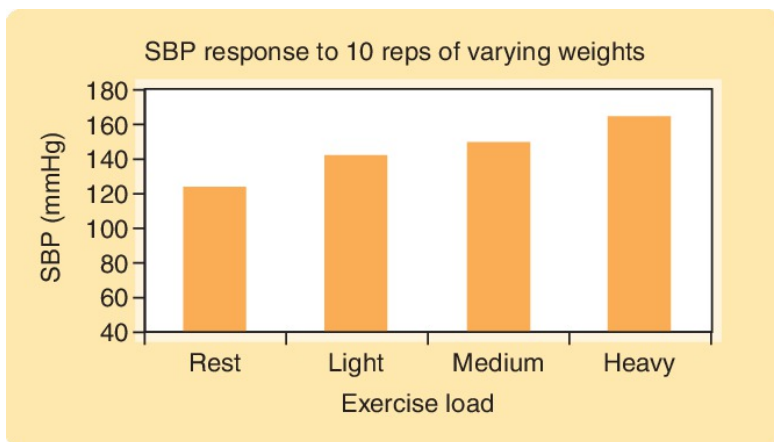


Figure 12.15 Systolic Blood Pressure (SBP) Response at the Completion of 10 reps of Arm Curls Using Different



Weights.

Source: Based on data from Wescott and Howes (1983).

Varying Load/Repetitions to Failure

A different pattern of response is seen when a given load is performed to fatigue, which lifters typically call failure. In this

case, the individual performs maximal work regardless of the load. **Figure 12.16** shows the cardiovascular response at the completion of leg extension exercise performed to failure. Participants performed 50, 80, and 100% of their one-repetition maximum (1-RM) as many times as they could, and cardiovascular variables were recorded at the end of each set (Falkel et al., 1992). Participants could perform the 100% load only one time, of course, but they could perform the 80 and 50% loads an average of 8 and 15 times, respectively. Thus, the greatest amount (volume) of work was performed when the lightest load was lifted the greatest number of times. Cardiac output at the completion of the set was highest when the lightest load was lifted for the most repetitions—that is, when the total work was greatest (**Figure 12.16A**).

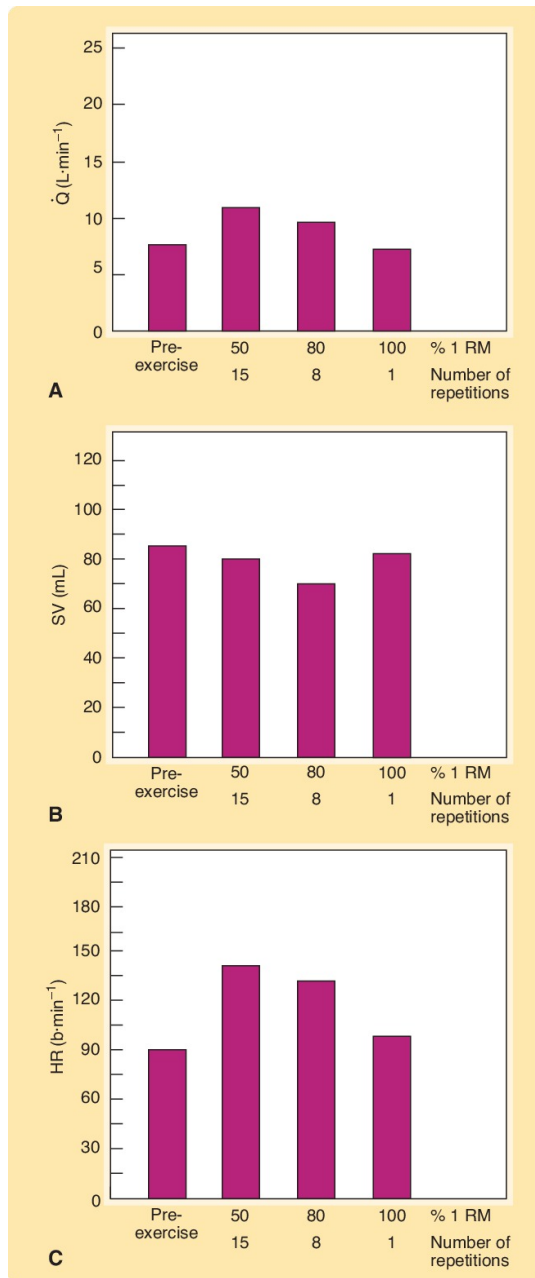


Figure 12.16 Cardiovascular Response at the Completion of Dynamic Resistance Exercise (Concentric Knee Extension Exercise) to Failure with Varying Loads.



A. Cardiac output (Q.). B. Stroke volume (SV). C. Heart rate (HR). **Source:** Based on data from [Falkel et al. \(1992\)](#).

The SV at the end of a set was similar for each condition (**Figure 12.16B**) and was slightly below resting levels. This is in contrast to significant increases in the SV that occur during aerobic exercise. Thus, dynamic resistance exercise does not produce the SV overload of dynamic aerobic exercise ([Hill and Butler, 1991](#); [McCartney, 1999](#)). The HR was highest after completion of the set using the lightest load and lifting it the most times (**Figure 12.16C**). The HR was lowest when a single repetition using the heaviest weight was performed and hence when the least amount of work was done. HRs between 130 and 160 $\text{b}\cdot\text{min}^{-1}$ have been reported during resistance exercise ([Hill and Butler, 1991](#)). There is some evidence that the HR and the BP attained at fatigue are the same when loads between 60 and 100% of 1-RM are used, regardless of the number of times the load can be performed ([Nau et al., 1990](#)).

Constant Load/Repetitions to Failure

When the load is heavy, MAP and HR increase with succeeding repetitions in a set to failure ([Fleck and Dean, 1987](#); [MacDougall et al., 1985](#)). **Figure 12.17A** shows the MAP, measured intra-arterially, during a set of leg press exercises that represented 95% of 1-RM; **Figure 12.17B** shows the HR during these exercises. In this study, peak SBP averaged 320 mmHg, and peak DBP averaged 250 mmHg! The dramatic increase in BP during dynamic resistance exercise results from the mechanical compression of blood vessels and performance of the Valsalva maneuver (as explained in [Chapter 10](#)). The TPR is higher during dynamic resistance exercise than during dynamic aerobic exercise because of the vasoconstriction caused by the pressor reflex. In fact, some studies have reported a slight increase in the TPR during resistance exercise, rather than the decrease observed with aerobic exercise ([Lentini et al., 1993](#); [McCartney, 1999](#); [Miles et](#)

al., 1987). Myocardial oxygen consumption and thus the rate-pressure product can reach extremely high levels because of the tachycardia and the exaggerated SBP response. Dynamic resistance exercise also causes large (about 15%) but transient decreases in plasma volume (Hill and Butler, 1991).

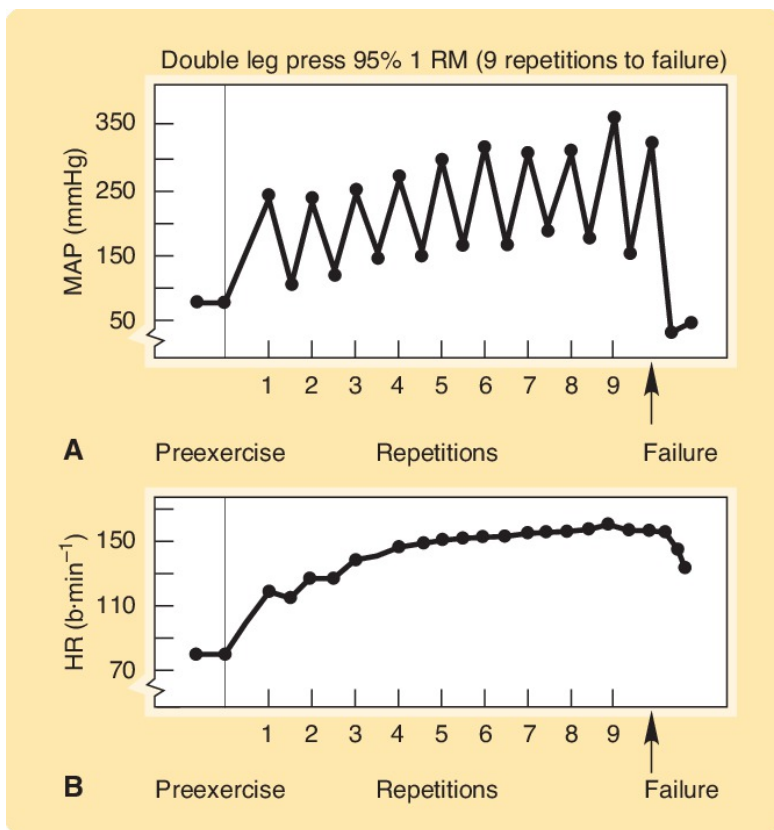


Figure 12.17 Mean Arterial Pressure (MAP; Panel A) and Heart Rate (HR; Panel B) Responses during a Set of

Dynamic Resistance Exercise to Failure.



Source: Reprinted with permission from MacDougall, J. D., D. Tuxen, D. G. Sale, J. R. Moroz, & J. R. Sutton: Arterial blood pressure response to heavy resistance exercise. *Journal of Applied Physiology*. 58(3):785–790 (1985).

Cardiovascular responses to resistance exercise are summarized in **Table 12.1**.

Resistance exercises are generally undertaken to enhance muscle size or to improve muscular health (strength or endurance). The goal is not to stress the cardiovascular system. Hence, there is insufficient evidence to adequately compare cardiovascular responses to resistance exercise among different populations (male vs. female, children vs. adult, and young vs. older adults). Therefore, this exercise category is not included in the following sections.

Male-female Cardiovascular Differences during Exercise

The pattern of cardiovascular responses to aerobic exercise is similar for both sexes, although the magnitude of the response may vary for some variables. Many of the differences in cardiovascular responses between the sexes are related to differences in body size and structure.

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

Females have a higher cardiac output and HR, but a lower SV, than males during submaximal exercise when work is performed at the same absolute workload (Åstrand et al., 1964; Becklake et al., 1965; Freedson et al., 1979). The higher HR more than compensates for the lower SV in females, resulting in the higher cardiac output seen at the same absolute workload. Thus, if a male and a female perform the same exercise, the female will typically be stressing the cardiovascular system to a greater extent (**Figure 12.18**). This relative disadvantage to women

results from several factors. First, females typically are smaller than males; they have a smaller heart and less muscle mass. Second, they have a lower oxygen-carrying capacity than males. Finally, they typically have lower aerobic capacity ($\dot{V}O_2 \max$).



Figure 12.18 Male and Female Exercising Together.

When males and females perform the same relative workload (both working at the same percentage of their $\dot{V}O_2 \text{ max}$), a different pattern emerges. The importance of distinguishing between relative and absolute workloads is shown in **Figure 12.19**. This figure shows results from a study that compared the cardiovascular response of men and women to the same absolute work rate ($600 \text{ kg}\cdot\text{min}^{-1}$) and the same relative work rate (50% $\dot{V}O_2 \text{ max}$). Although cardiac output was higher in women during the same absolute work rate, it is lower for women when the same relative work rate was performed (**Figure 12.19A**). The SV (**Figure 12.19B**) was lower in women than in men whether the work was expressed on an absolute or relative basis. Notice that the values are very similar for both conditions, suggesting that the SV has plateaued as would be expected at 50% $\dot{V}O_2 \text{ max}$ in both conditions. The difference in HR between the sexes (**Figure 12.19C**) was smaller when exercise was performed at the same relative work rate.

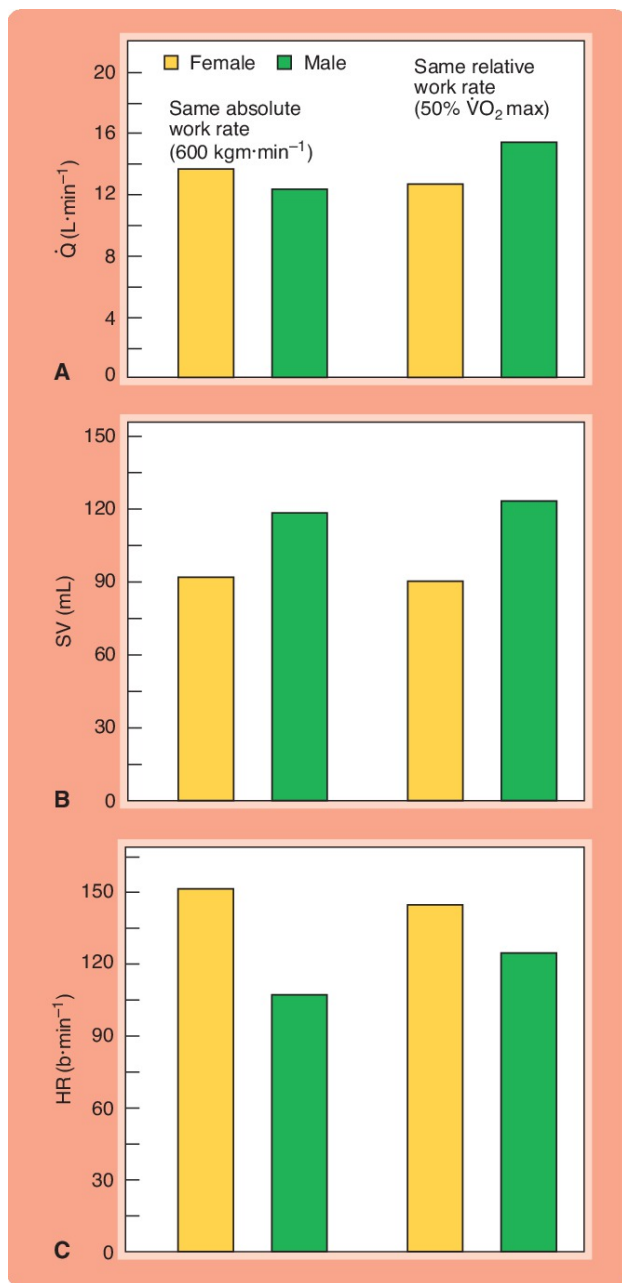


Figure 12.19 Comparison of Cardiovascular Responses of Men and Women to Submaximal Aerobic Exercise.



A. Cardiac output (Q.). B. Stroke volume (SV). C. Heart rate (HR). **Source:** Data from Åstrand (1952).

Males and females display the same pattern of response for BP; however, males tend to have a higher SBP at the same relative workloads (Deschenes et al., 2006; Malina and Bouchard, 2004; Ogawa et al., 1992). Much of the difference in the magnitude of the BP response is attributable to differences in resting SBP. The DBP response to submaximal exercise is very similar for both sexes. Thus, MAP is slightly greater in males during submaximal work at the same relative workload. The pattern of response for resistance is similar for males and females, although males typically have a lower resistance because of their greater cardiac output. Males and females both exhibit cardiovascular drift during heavy, prolonged submaximal exercise. Changes in plasma volume appear to be greater in men than in women at the same absolute submaximal exercise intensity (Deschenes et al., 2006).

Incremental Aerobic Exercise to Maximum

The cardiovascular response to incremental exercise is similar for both sexes, although again there are differences in the maximal values attained. Maximal oxygen consumption ($\dot{V}O_2 \text{ max}$) is higher for males than for females. When $\dot{V}O_2 \text{ max}$ is expressed in absolute values ($\text{L} \cdot \text{min}^{-1}$), males typically have values that are 40–60% higher than in females (Åstrand, 1952; Sparling, 1980). When differences in body size are considered and values are expressed relative to body weight (in $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), the differences between the sexes decrease to 20–30%. If differences in body composition are considered and $\dot{V}O_2 \text{ max}$ is expressed relative to fat-free mass (in $\text{mL} \cdot \text{kg}^{-1}$ of fat-free mass per minute), the difference between the sexes is reduced to 0–15% (Sparling, 1980). Reporting $\dot{V}O_2 \text{ max}$ relative to fat-free

mass is important in terms of understanding the influence of adiposity and fat-free mass on $\dot{V}O_2 \text{ max}$. However, it is not a very practical way to express $\dot{V}O_2 \text{ max}$ because, in reality, consuming oxygen only in relation to fat-free mass is not an option. Individuals cannot leave their fat mass behind when exercising.

Figure 12.20 represents the distribution of $\dot{V}O_2 \text{ max}$ values for males and females expressed per kilogram of weight and per kilogram of fat-free mass. This figure demonstrates the important point that there is considerable variability in $\dot{V}O_2 \text{ max}$ for both sexes. Thus, although males generally have a higher $\dot{V}O_2 \text{ max}$, some females will have a higher $\dot{V}O_2 \text{ max}$ than the average male.

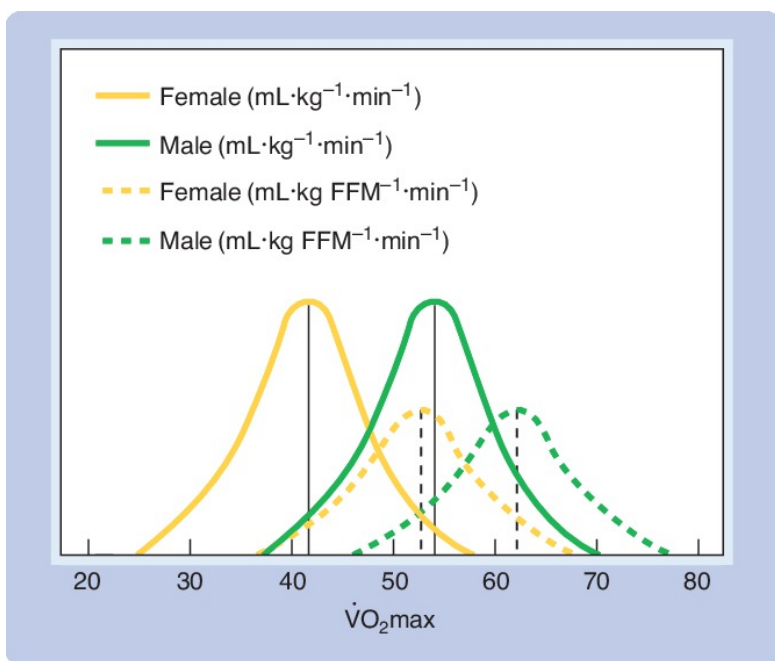


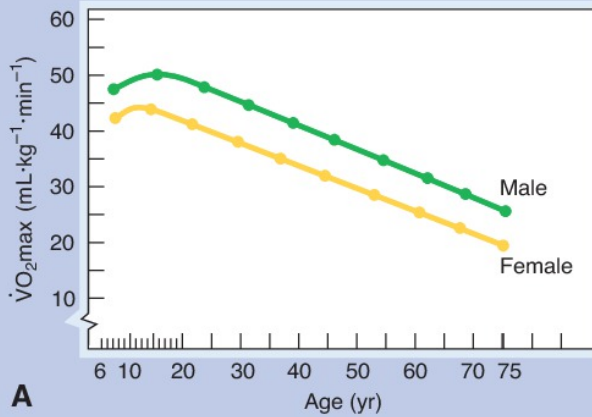
Figure 12.20 Distribution of $\dot{V}O_2 \text{ max}$ for Males and

Females.

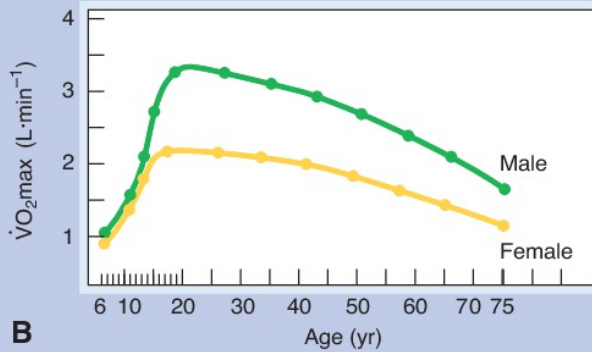


Source: From Wells, C. L., & S. A. Plowman: Sexual differences in athletic performance: Biological or behavioral? *Physician and Sports Medicine*. 11(8):52–63 (1983). Reprinted by permission of Taylor & Francis Ltd., <https://www.tandfonline.com>.

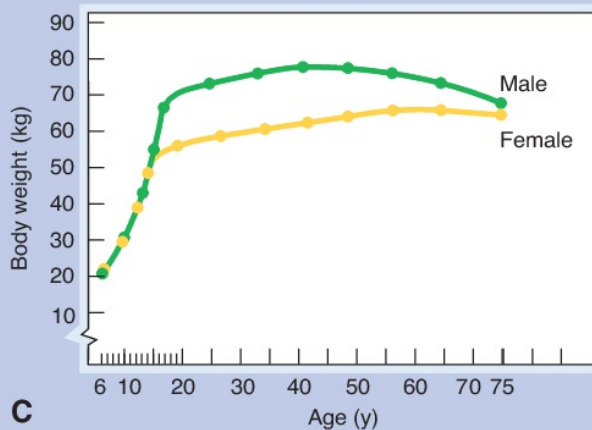
Figure 12.21 shows the differences in $\dot{V}O_2 \text{ max}$, expressed in relative terms (**Figure 12.21A**) and absolute terms (**Figure 12.21B**), and average body weight (**Figure 12.21C**) between the sexes across the age span. Differences in $\dot{V}O_2 \text{ max}$ are largely explained by the differences in the size of the heart (and thus maximal cardiac output) and the differences in the oxygen-carrying capacity of the blood. Males have approximately 6% more red blood cells and 10–15% more hemoglobin than females; thus, males have a greater oxygen-carrying capacity (Åstrand et al., 2003).



A



B



C

Figure 12.21 Maximal Oxygen Consumption ($\dot{V}O_2 \max$) Expressed in Relative (A) and Absolute Terms (B) and Body Weight (C) for Males and Females from 6 to



75 Years.

Source: Reprinted with permission from Shvartz, E., & R. C. Reibold: Aerobic fitness norms for males and females aged 6 to 75 years: A review. *Aviation, Space, and Environmental Medicine*. 61:3–11 (1990).

Males typically have a maximal cardiac output that is 30% higher than that of females (Wells and Plowman, 1983). Maximal SV is higher for men, but the increase in SV during maximal exercise is achieved by the same mechanisms in both sexes (Sullivan et al., 1991). Furthermore, if maximal SV is expressed relative to body weight, there is no difference between the sexes. The maximal HR is similar for both sexes.

Males and females display the same pattern of BP response; however, males attain a higher SBP than females at maximal exercise (Malina and Bouchard, 2004; Ogawa et al., 1992; Wanne and Haapoja, 1988). The DBP response to maximal exercise is similar for both sexes. Thus, MAP is slightly greater in males at the completion of maximal work. The pattern of response for total peripheral resistance and rate-pressure product is the same for both sexes. Total peripheral resistance is greatly reduced during maximal exercise in both sexes. Because the HR response is similar and the SBP is greater in males, males tend to have a higher rate-pressure product at maximal exercise levels than do females.

Table 12.2 summarizes the differences between the sexes in cardiovascular variables at various exercise levels.

TABLE 12.2 Cardiovascular Variables for Females Compared to Males

| Variable | Rest | Exercise Condition | | |
|------------------|--------|-------------------------|-------------------------|-------------------------|
| | | Absolute, Submaximal | Relative, Submaximal | Incremental, Maximal |
| $\dot{V}O_2$ max | — | — | — | Lower |
| \dot{Q} | Lower | Higher | ? | Lower |
| SV | Lower | Lower | Lower | Lower |
| HR | Higher | Higher | Higher | Similar |

Source: Wells (1991).

Static Exercise

The HR response to static exercise (**Figure 12.22A**) is similar in males and females (Misner et al., 1990). However, as shown in **Figure 12.22B**, when a group of young adult, healthy participants held maximal contractions of the handgrip muscles for 2 minutes, the BPs reported for women were significantly lower than those reported for men (Misner et al., 1990). A more recent study in which men and women performed 2 minutes of handgrip exercise at 30% of maximal voluntary contraction while blood pressure was continuously monitored also found that men had a higher blood pressure response to the static exercise. However, when the authors performed statistical analyses to account for differences in maximal voluntary contraction, sex was no longer significantly associated with BP response, suggesting that differences in muscle strength account for much of the difference in blood pressure response to static exercise (Notay et al., 2018). The SV and cardiac output responses in women during maximal static contraction of the finger flexors were similar to the responses previously reported in men, but no direct comparisons between men and women were made in this study (Smith et al., 1993).

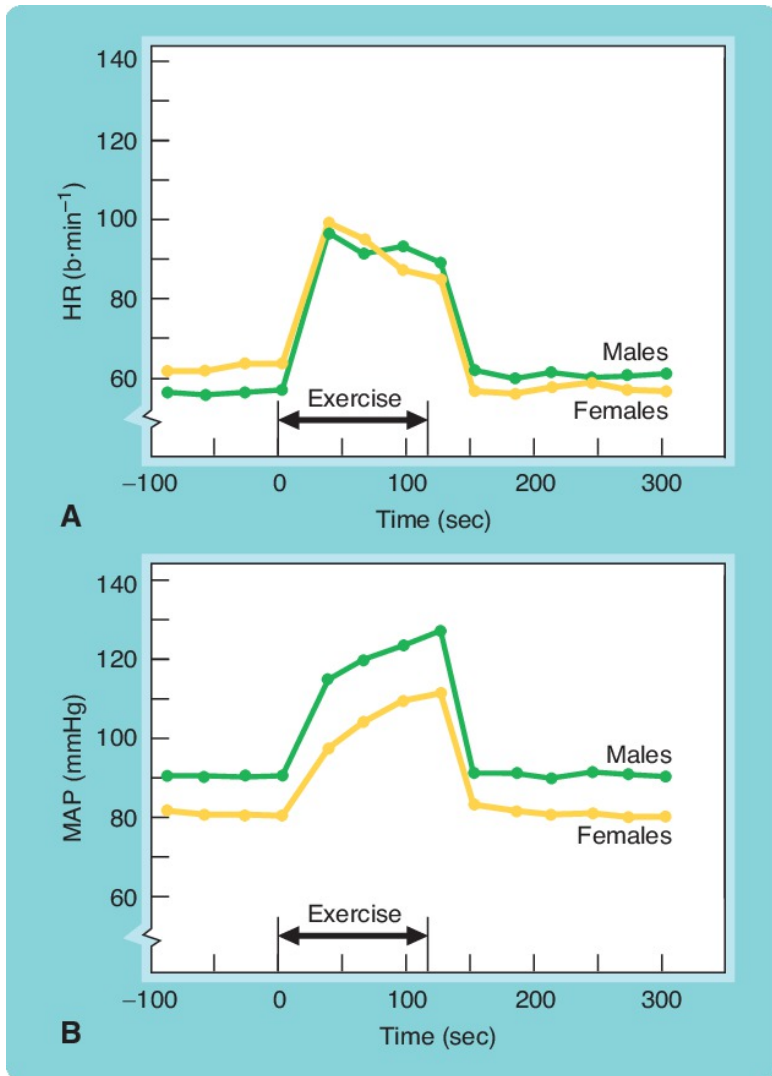


Figure 12.22 Heart Rate (HR; Panel A) and Mean Arterial Pressure (MAP; Panel B) Responses of Males

and Females to Static Exercise.



Source: Reprinted with permission from Misner, J. E., S. B. Going, B. H. Massey, T. E. Ball, M. G. Bemben, & L. K. Essandoh: Cardiovascular response in males and females to

sustained maximal voluntary static muscle contraction. *Medicine and Science in Sports and Exercise*. 22(2):194–199 (1990). Copyright ©1990 The American College of Sports Medicine.

Cardiovascular Responses of Children and Adolescents to Exercise

The cardiovascular response of children to resistance exercise is similar to that of adults, with the HR and the BP increasing progressively throughout a set ([Nau et al., 1990](#)). However, as stated previously, beyond this, there are little data, so resistance exercise responses will not be discussed further in this section.

The pattern of responses in the cardiovascular variables in children and adolescents to aerobic exercise is similar to the pattern in adults. This is not meant to imply that children are simply “little adults.” Often, the actual values are higher or lower than those for adults, but overall, the direction and the relative degree of change are very similar across the age range. Differences can frequently be attributed to differences in body size, structure, and maturity ([Rowland, 2005a, 2005b](#)).

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

The pattern of cardiac output response to submaximal aerobic exercise is similar in children and adolescents to adults, with cardiac output increasing rapidly at the onset of exercise and plateauing at a *steady state*. However, children have a lower cardiac output than adults at all levels of exercise, primarily because children have a lower SV at any level of exercise ([Bar-Or, 1983](#); [Rowland, 1990](#)). As children grow and mature, cardiac output and SV increase at rest and during exercise. The lower SV in children is compensated for, to some extent, by a higher HR. The HR response to any given exercise intensity is highest in

young children (Bar-Or, 1983; Cunningham et al., 1984) and declines as children grow into adolescents (Rowland, 2005a, 2005b). Children, adolescents, and adults all exhibit the cardiovascular drift phenomenon of a slight (~15%) progressive rise in HR, simultaneous decreases in SV and MAP, and no change in cardiac output with prolonged exercise (Asano and Hirakoba, 1984; Rowland, 2005a). SV in girls is less than that in boys at all levels of exercise (Bar-Or, 1983).

As always, the magnitude of the cardiovascular response depends on the intensity of the exercise. **Table 12.3** reports the cardiac output, SV, and HR values of children 8–12 years old during treadmill exercise at 40, 53, and 68% $\dot{V}O_2 \text{ max}$ (Lussier and Buskirk, 1977). Both cardiac output and HR increase in response to increasing intensities of exercise. SV peaks at 40% $\dot{V}O_2 \text{ max}$ and changes little with increasing exercise intensity. This is consistent with the finding that SV plateaus at 40–50% $\dot{V}O_2 \text{ max}$ in untrained adults (Åstrand et al., 1964).

TABLE 12.3 Cardiovascular Responses in Children to Submaximal Exercise of Various Intensities

| Variable | Intensity of Exercise (% $\dot{V}O_2 \text{ max}$) | | |
|----------------------------------|---|-----|-----|
| | 40% | 53% | 68% |
| \dot{Q} (L·min ⁻¹) | 6.7 | 7.6 | 8.5 |
| SV (mL·b ⁻¹) | 53 | 51 | 49 |
| HR (b·min ⁻¹) | 126 | 149 | 173 |

Source: Lussier and Buskirk (1977).

The SBP in children increases during exercise, as it does in adults, and depends on the intensity of the exercise. Boys tend to have a higher SBP than girls (Malina and Bouchard, 2004). The magnitude of the increase in systolic pressure at submaximal exercise is less in children than in adults (James et al., 1980; Wanne and Haapoja, 1988). The failure of SBP to reach adult levels is probably the result of lower cardiac output in children.

As children mature, the increases in the SBP during exercise become greater. Diastolic pressure changes little during exercise but is lower in children than in adults (James et al., 1980; Wanne and Haapoja, 1988).

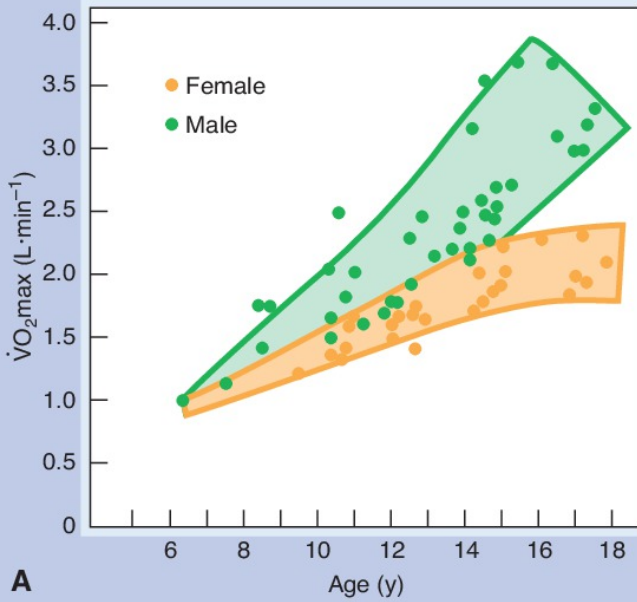
Similar decreases in resistance occur in children as in adults, a result of vasodilation in working muscles. Rate-pressure product increases in children and adolescents during exercise. However, the work of the heart reflects the higher HR and lower SBP for these age groups than for adults. Blood flow through the exercising muscle appears to be greater in children than in adults, resulting in a higher a-vO₂diff and thereby compensating partially for the lower cardiac output (Rowland, 1990; Rowland and Green, 1988).

Incremental Aerobic Exercise to Maximum

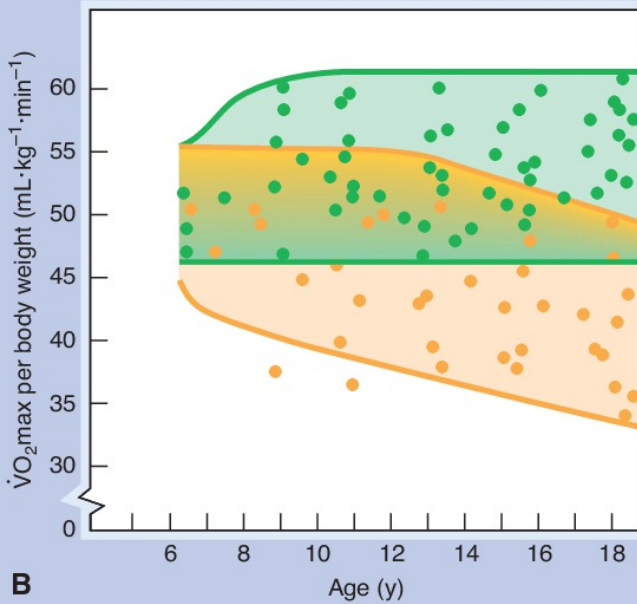
The cardiovascular responses to incremental exercise to maximum are similar for children, adolescents, and adults; however, children and adolescents achieve a lower maximal cardiac output and a lower maximal SV. HR rises in a rectilinear fashion with incremental exercise in children as in adults.

However, at approximately 60% $\dot{V}O_2 \text{ max}$, it begins to taper. Maximal HR is higher in children than in adults (Cunningham et al., 1984; Rowland, 1996, 2005a).

The maximal oxygen consumption typically attained by youths between the ages of 6 and 18 is shown in **Figure 12.23**. As children grow, their ability to take in, transport, and utilize oxygen improves. This improvement represents dimensional and maturational changes—specifically, heart volume, maximal SV, maximal cardiac output, blood volume and hemoglobin concentration, and a-vO₂diff increase.



A



B

Figure 12.23 Maximal Oxygen Consumption ($\dot{V}O_2\text{max}$)



of Children and Adolescents.

A. Changes in $\dot{V}O_2 \text{ max}$ in children and adolescents during the ages of 6–18 years are expressed in absolute terms. The *dots* represent means from various studies. The *outer lines* indicate normal variability in values. **B.** Changes in $\dot{V}O_2 \text{ max}$ in children and adolescents during the ages of 6–18 years are expressed relative to body weight. The *dots* represent means from various studies. The *outer lines* indicate normal variability in reported values. **Source:**

Reprinted by permission from Springer Bar-Or, O.: Physiologic Responses to Exercise of the Healthy Child. In: Bar-Or, O. (ed.): *Pediatric Sports Medicine for the Practitioner: From Physiologic Principles to Clinical Applications*. New York, NY: Springer-Verlag, 1–65 (1983). Copyright © 1983 Springer-Verlag New York Inc.

The rate of improvement in absolute $\dot{V}O_2 \text{ max}$ (expressed in $\text{L}\cdot\text{min}^{-1}$) is similar for boys and girls until approximately 12 years of age (**Figure 12.23A**). Maximal oxygen uptake continues to increase in boys until the age of 18; it remains relatively constant in girls between the ages of 14 and 18 years.

When $\dot{V}O_2 \text{ max}$ is expressed relative to body weight (expressed as $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), it remains relatively constant throughout the years between 8 and 16 for boys (**Figure 12.23B**).

However, $\dot{V}O_2 \text{ max}$ (expressed as $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) tends to decrease in girls as they enter puberty and their adiposity increases (**Figure 12.23B**). As children mature, they also grow, and the developmental changes indicated previously are largely offset if $\dot{V}O_2 \text{ max}$ is described per kilogram of body weight.

The large area of overlap for reported values of $\dot{V}O_2 \text{ max}$ for boys and girls in **Figure 12.23** reflects the large variability in $\dot{V}O_2 \text{ max}$ among children and adolescents.

There appears to be a major difference between children/

adolescents and adults in terms of the meaning of $\dot{V}O_2 \text{ max}$. In adults, $\dot{V}O_2 \text{ max}$ reflects both physiological function (cardiorespiratory power) and cardiovascular endurance (the ability to perform strenuous, large-muscle exercise for a prolonged period of time) (Taylor et al., 1955). In children and adolescents, $\dot{V}O_2 \text{ max}$ is not as directly related to cardiorespiratory endurance as in adults or at least more complex than in adult individuals (Bar-Or, 1983; Krahenbuhl et al., 1985; Rowland, 1990, 2013). **Figure 12.24B** shows performance as determined by the number of stages or minutes completed in the PACER test (Léger et al., 1988). This progressive aerobic cardiovascular endurance run (PACER—or 20-m shuttle test) was described in Chapter 11. Recall that a higher number of laps completed is positively associated with a higher $\dot{V}O_2 \text{ max}$. **Figure 12.24A** shows that for boys, the mean estimated value of $\dot{V}O_2 \text{ max}$, expressed in $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, changes very little from age 6 to 18 years. However, mean performance on the PACER test (**Figure 12.24B**) shows a definite linear improvement with age. The girls show the same trend as the boys before puberty, but thereafter, $\dot{V}O_2 \text{ max}$ declines steadily and PACER performance plateaus. Similar results have been reported for treadmill endurance times and other distance runs (Cumming et al., 1978), and for measured and estimated $\dot{V}O_2 \text{ max}$ (Rowland, 2005a). Thus, in general, endurance performance improves progressively throughout childhood, at least until puberty, but $\dot{V}O_2 \text{ max}$, expressed relative to body size, does not.

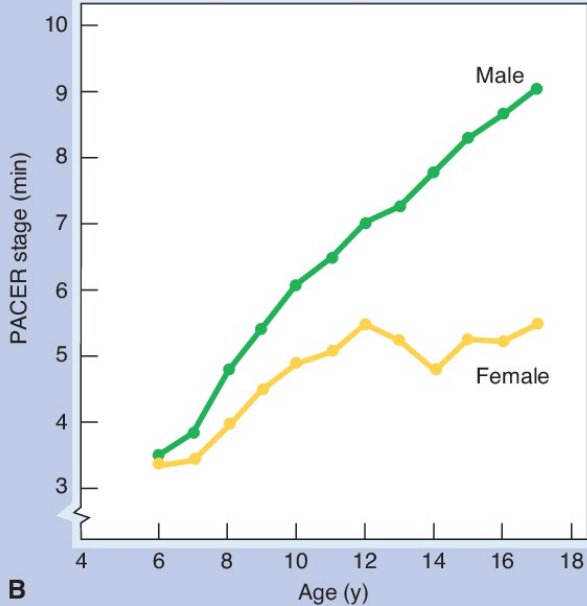
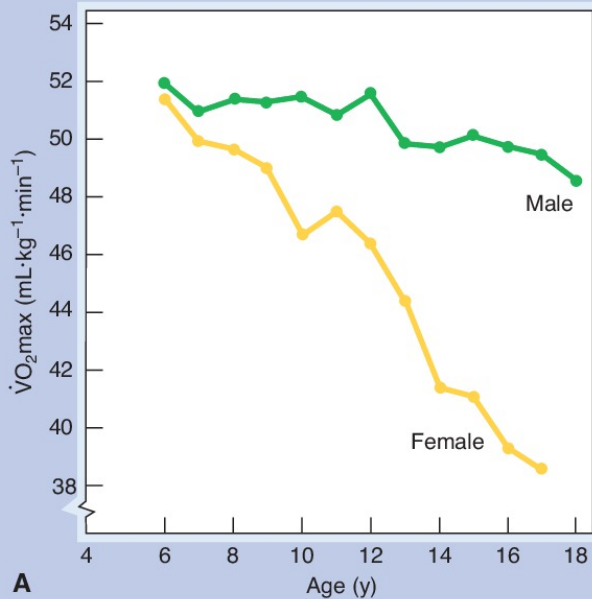


Figure 12.24 Maximal Oxygen Consumption ($\dot{V}O_2\text{max}$; Panel A) and Endurance Performance (Panel B) in



Children and Adolescents.

Source: Modified from Léger, L. A., D. Mercer, C. Gadoury, & J. Lambert: The multistage 20 metre shuttle run test for aerobic fitness. *Journal of Sports Sciences*. 6(2):93–101 (1988). Reprinted by permission of Taylor & Francis Ltd., <https://www.tandfonline.com>.

The reason for the weak association between $\dot{V}O_2 \text{ max}$ and endurance performance in young people is unknown. The most frequent suggestion is that children use more aerobic energy (require greater oxygen) than adults at any submaximal pace. This phenomenon is called running economy and is fully discussed in the unit on metabolism (Chapter 4). More important than the actual oxygen consumption at a set pace, however, may

be the percentage of $\dot{V}O_2 \text{ max}$ that value represents, and more so in children than in adolescents (McCormack et al., 1991). Other factors that may affect endurance running performance in children and adolescents include body composition, particularly the percentage of body fat; sprint speed, possibly as a reflection of a high percentage of muscle fibers differentiated for speed and power; and various aspects of body size (Cureton et al., 1977, 1991; Mayhew and Gifford, 1975; McVeigh et al., 1995). There is also the possibility that many children and adolescents are not motivated to perform exercise tests and therefore do not perform well despite high $\dot{V}O_2 \text{ max}$ capabilities.

The BP response is similar for children and adults; however, there are again age- or size-related quantitative differences. For a given level of exercise, a small child responds with a lower SBP and DBP than an adolescent, and an adolescent responds with lower BP than an adult. The lower BP response in young children is consistent with their lower SV response. Typically, boys have a higher peak SBP than girls (Riopel et al., 1979; Wade and Freund, 1990). This difference too is most likely attributable to differences in SV. Myocardial oxygen consumption at maximal exercise increases as children grow—predominantly through the

influence of higher maximal SBP since maximal HR is stable until late adolescence.

Table 12.4 reports typical cardiovascular responses to maximal exercise in prepubescent and postpubescent children.

TABLE 12.4 Cardiovascular Responses to Maximal Exercise in Prepubescent and Postpubescent Children

| Variable | Boys | | Girls | |
|-------------------------------------|------|------|-------|-------|
| | 10 y | 15 y | 10 y | 15 y |
| \dot{Q} (L·min ⁻¹) | 12 | 18 | 11 | 14 |
| SV (mL·b ⁻¹) | 60 | 90 | 55 | 70 |
| HR (b·min ⁻¹) | 200 | 200 | 200 | 200 |
| $\dot{V}O_2$ (L·min ⁻¹) | 1.7 | 3.5 | 1.5 | 2.0 |
| SBP (mmHg) | 144 | 174 | 140 | 170 |
| DBP (mmHg) | 64 | 64 | 64 | 64 |
| MAP (mmHg) | 105 | 110 | 103 | 117.5 |
| TPR (units) | 7.0 | 6.1 | 9.4 | 8.4 |
| RPP (units) | 290 | 350 | 280 | 340 |

Sources: Åstrand (1952); Rowland (1990).

Static Exercise

Children's and adolescents' cardiovascular responses to static exercise appear to be similar to adults' (Rowland, 2005a, 2005b). **Table 12.5** presents data from two studies that investigated cardiovascular responses to 3 minutes of leg extension exercise at 30% of MVC. One study tested young men between the ages of 25 and 34 years (Bezucha et al., 1982) and the other young boys aged 7–12 years (Rowland et al., 2006). In both studies, static exercise resulted in typical responses: an increased MAP, an elevated HR, a decreased SV, and a small rise in cardiac output. Similarly, a study that compared premenarcheal girls and young

women found no differences in cardiovascular responses to 3 minutes of 30% MVC of the handgrip muscles (Smith et al., 2000).

TABLE 12.5 Cardiovascular Responses of Boys and Men to Static Exercise

| | Men | | Boys | |
|---------------------------|------|----------|------|----------|
| | Rest | Exercise | Rest | Exercise |
| HR (b·min ⁻¹) | 70 | 110 | 77 | 106 |
| SV (mL·b ⁻¹) | 85 | 62 | 59 | 52 |
| Q̇ (L·min ⁻¹) | 5.7 | 6.8 | 4.8 | 5.6 |
| MAP (mmHg) | | | 86 | 109 |

Sources: Based on the data from Bezucha et al. (1982); Rowland et al. (2006).

Cardiovascular Responses of Older Adults to Exercise

Aging is associated with diminishing function in many systems of the body. Thus, aging is characterized by a decreased ability to respond to physiological stress (Skinner, 1993). There is considerable debate, though, about how much loss of function is inevitably related to age, how much is related to disease, and how much can be attributed to a sedentary lifestyle often accompanying aging. Each of these factors causes decrements in function, but for an individual, it is often difficult to know which one or which combination may cause an observed change.

Many older adults remain active into their later years and perform amazing athletic feats. For example, Mavis Lindgren began an exercise program of walking in her early 60s. She slowly increased her training volume and began jogging. At age 70, she completed her first marathon. In the next 12 years, she

raced in over 50 marathons (Nieman, 1990). Many studies of physical activity suggest that by remaining active in the older years, individuals can markedly reduce loss of cardiovascular function, even if they do not run a marathon.

Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

At the same absolute submaximal workload, cardiac output and SV are lower in older adults, but HR is higher than in younger adults. The pattern of systolic and diastolic pressure is the same for younger and older individuals. The difference in resting BP is maintained throughout the exercise, so that older individuals have a higher SBP, DBP, and MAP at any given level of exercise (Ogawa et al., 1992). The higher BP response is related to a higher TPR in older individuals, resulting from a loss of elasticity in the blood vessels. Because HR and SBP are higher for any given level of exercise in older adults, myocardial oxygen consumption and thus rate-pressure product are also higher in older individuals than in younger adults.

Incremental Aerobic Exercise to Maximum

Cardiorespiratory responses to incremental exercise change with advancing age (Edvardsen et al., 2013). Maximal cardiac output is lower in older individuals than in younger adults. This results from a lower maximal HR and a lower maximal SV. Maximal SV decreases with advancing age, and the decline is of similar magnitude for both men and women, although women have a much smaller maximal SV initially. Maximal HR decreases with age but does not vary significantly between the sexes. A decrease of approximately 10% per decade, starting at approximately age

30, has been reported for $\dot{V}O_2 \text{ max}$ in sedentary and active adults (Åstrand, 1960; Heath et al., 1981; Wilson and Tanka, 2000). There is some indication that the rate of decline in $\dot{V}O_2 \text{ max}$ is greater in men than in women (Stathokostas et al., 2004; Weiss et al., 2006). **Figure 12.21A and B** depicts the

change in $\dot{V}O_2 \text{ max}$ from childhood to 75 years of age.

Like resting BP, SBP and DBP responses to maximal aerobic exercise are typically higher in older individuals than in younger individuals of similar fitness (Ogawa et al., 1992). Maximal SBP may be 20–50 mmHg higher in older individuals and maximal DBP 15–20 mmHg higher. As a result of an elevated SBP and DBP, MAP is considerably higher at maximal exercise in older than in younger adults.

TPR decreases during aerobic exercise in older adults but not to the same extent as in younger individuals. This difference is a consequence of the loss of elasticity of the connective tissue in the vasculature that accompanies aging. Since the decrease in maximal HR for older individuals is greater than the increase in maximal SBP when compared to younger adults, older individuals have a lower rate-pressure product at maximal exercise. **Table 12.6** presents typical cardiovascular values at maximal exercise in young and older adults of both sexes.

TABLE 12.6 Cardiovascular Responses to Maximal Exercise in Young and Older Adults

| Variable | Men | | Women | |
|-------------------------------------|------|------|-------|------|
| | 25 y | 65 y | 25 y | 65 y |
| \dot{Q} (L·min ⁻¹) | 25 | 16 | 18 | 12 |
| SV (mL·b ⁻¹) | 128 | 100 | 92 | 75 |
| HR (b·min ⁻¹) | 195 | 155 | 195 | 155 |
| $\dot{V}O_2$ (L·min ⁻¹) | 3.5 | 2.5 | 2.5 | 1.5 |
| SBP (mmHg) | 190 | 200 | 190 | 200 |
| DBP (mmHg) | 70 | 84 | 64 | 84 |
| MAP (mmHg) | 130 | 143 | 128 | 143 |
| TPR (units) | 5.2 | 8.9 | 7.1 | 11.9 |
| RPP (units) | 371 | 310 | 371 | 310 |

Source: Ogawa et al. (1992).

Static Exercise

Many studies have described the cardiovascular responses to static exercise in older adults (Goldstraw and Warren, 1985; Petrofsky and Lind, 1975; Sagiv et al., 1988; Van-Loan et al., 1989). As an example, **Figure 12.25** depicts the cardiovascular responses of young and older men to sustained handgrip exercise over a range of submaximal static workloads (VanLoan et al., 1989). Note that cardiac output (**Figure 12.25A**) and SV (**Figure 12.25B**) values are lower than normally reported, because of the measurement technique. However, the relative differences between the responses of the young and the older participants show that cardiac output, SV, and HR (**Figure 12.25C**) were lower for the older men than for the younger men at each intensity. In contrast, BP responses (**Figure 12.25D and E**) were higher for the older men at each intensity. As with dynamic aerobic exercise, the differences in the cardiovascular responses between the two age groups are probably due to an age-related increase in resistance due to a loss of elasticity in the vasculature and a decreased ability of the myocardium to stretch and contract forcibly (VanLoan et al., 1989). The rate-pressure product (**Figure 12.25F**) was higher for the younger participants than for the older participants at 30, 45, and 60% MVC. The small difference in rate-pressure product reflected a higher HR in younger participants at each intensity of contraction, which was not completely offset by a lower SBP in the younger participants.

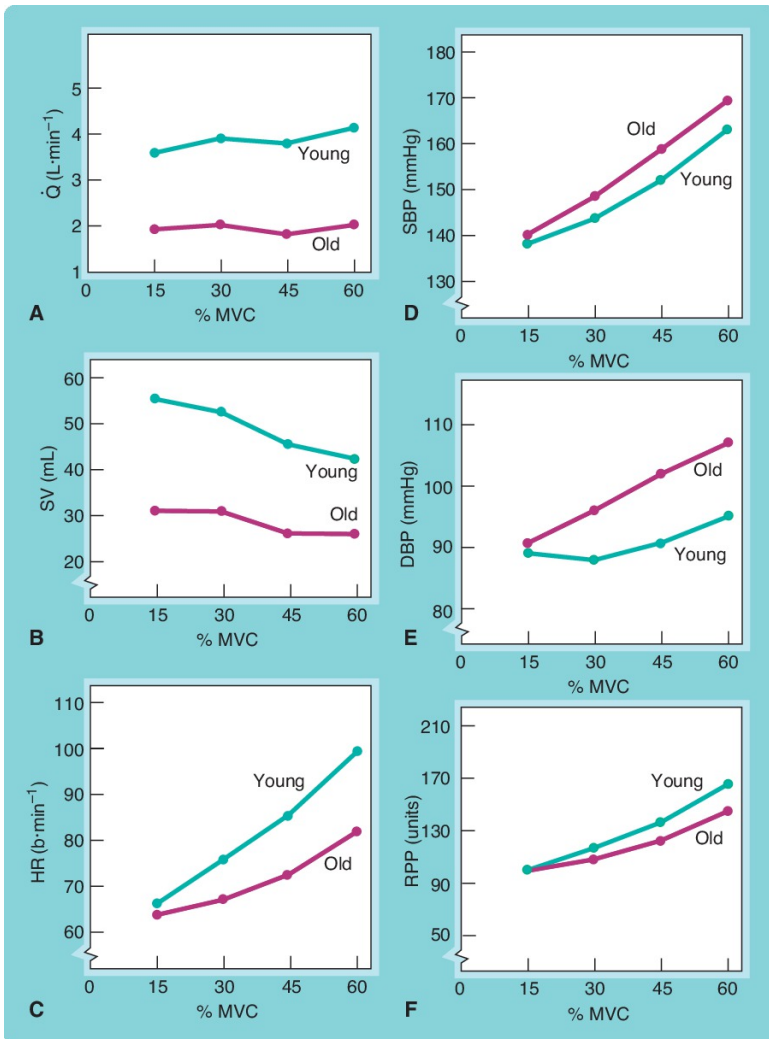


Figure 12.25 Cardiovascular Responses of Males by Age

to Static Exercise.



A. Cardiac output (\dot{Q}). B. Stroke volume (SV). C. Heart rate (HR). D. Systolic blood pressure (SBP). E. Diastolic blood pressure (DBP). F. Rate-pressure product (RPP). *Source:* [VanLoan et al. \(1989\)](#).

Summary

1. During short-term, light to moderate aerobic exercise, cardiac output (\dot{Q}), stroke volume (SV), heart rate (HR), systolic blood pressure (SBP), and rate-pressure product (RPP) increase rapidly at the onset of exercise and reach steady state within approximately 2 minutes. Diastolic blood pressure (DBP) remains relatively unchanged, and resistance decreases rapidly and then plateaus.
2. During long-term, moderate to heavy aerobic exercise, SV, HR, SBP, and RPP increase rapidly. Once steady state is achieved, cardiac output remains relatively constant owing to the downward drift of SV and the upward drift of HR. SBP and resistance may also drift downward during prolonged, heavy work. This cardiovascular drift is associated with rising body temperature.
3. During incremental exercise to maximum, HR, SBP, and RPP increase in a rectilinear fashion with increasing workload. SV increases initially and then plateaus at a workload corresponding to approximately 40–50% $\dot{V}O_2 \text{ max}$ in normally active adults and children. DBP remains relatively constant throughout an incremental exercise test. Resistance decreases rapidly with the onset of exercise and reaches its lowest value at maximal exercise.
4. The decrease in resistance during aerobic exercise has two important implications. It allows greater blood flow to the working muscles and keeps blood pressure from rising excessively. The increase in cardiac output would produce a much greater rise in blood pressure if it were not for the fact that there is a simultaneous decrease in resistance.
5. The highest oxygen consumption during an incremental test may or may not represent an actual maximal value. The term $\dot{V}O_{2\text{peak}}$ is used to represent the highest value obtained during the test if the tester is not certain that a true maximal value was achieved. Specific criteria should be used to determine whether the test is truly a maximal test.
6. Maximal oxygen uptake may be limited by any system (or

step) along the pathway of bringing oxygen into the body and delivering it to the mitochondria for the production of ATP. Although factors in each of these systems may limit the $\dot{V}O_2 \max$, research suggests that cardiac output is the limiting factor in $\dot{V}O_2 \max$ in most individuals.

7. Blood volume decreases during aerobic exercise. Most of the decrease occurs within the first 10 minutes of activity and depends on exercise intensity. A decrease of 10% of blood volume is not uncommon.
8. SV initially increases during dynamic aerobic exercise and then plateaus at a level that corresponds to 40–50% $\dot{V}O_2 \max$ or continues to rise slightly. The increase in SV can result from increased end-diastolic volume (EDV) or decreased end-systolic volume (ESV) but decreased ESV due to increased contractility seems to account for most of the increase in SV in healthy individuals.
9. The pattern of cardiovascular response is the same for both sexes. However, males have a higher cardiac output, SV, and SBP at maximal exercise. Additionally, males have a higher $\dot{V}O_2 \max$. Most of these differences are attributable to differences in body size and heart size between the sexes and to the greater hemoglobin concentration of males.
10. The pattern of cardiovascular response in children and adolescents is similar to the adult response. However, children have a lower \dot{Q} , SV, and SBP at an absolute workload and at maximal exercise. Most of these differences are attributable to differences in body size and heart size.
11. As adults age, their cardiovascular responses change. Maximal \dot{Q} , SV, HR, and $\dot{V}O_2 \max$ decrease. Maximal SBP, DBP, and mean arterial pressure (MAP) increase.
12. Static exercise is characterized by modest increases in HR and \dot{Q} and exaggerated increases in SBP, DBP, and MAP, known as the pressor response.
13. Dynamic resistance exercise results in a modest increase in \dot{Q} , an increase in HR, little change or a decrease in SV, and a large increase in blood pressure.

Review Questions

1. Graph and explain the pattern of response for each of the major cardiovascular variables during short-term, light to moderate aerobic exercise. Explain the mechanisms responsible for each response.
 2. Graph and explain the pattern of response for each of the major cardiovascular variables during long-term, moderate to heavy aerobic exercise. Explain the mechanisms responsible for each response.
 3. Graph and explain the pattern of response for each of the major cardiovascular variables during incremental aerobic exercise to maximum. Explain the mechanisms responsible for each response.
 4. Discuss the factors that may limit $\dot{V}O_2 \text{ max}$ and indicate which factor is mostly likely to limit $\dot{V}O_2 \text{ max}$ in normal healthy individuals. Graph and explain the pattern of response for each of the major cardiovascular variables during static exercise. Explain the mechanisms responsible for each response.
 5. Graph and explain the pattern of response for each of the major cardiovascular variables during dynamic resistance exercise. Explain the mechanisms responsible for each response.
 6. Discuss the change that occurs in TPR during exercise, and explain its importance for blood flow and BP. Why is resistance altered in older adults?
 7. Describe the pressor response to static exercise, and explain the mechanisms by which BP is elevated.
 8. Describe the differences in male and female response to incremental maximal exercise.
 9. Explain why the $\dot{V}O_2 \text{ max}$, expressed in relative terms, of females decrease as children age.
 10. Compare the cardiovascular responses to incremental exercise in young and older adults.
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13

Cardiorespiratory Training Principles and Adaptations



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Summary

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Objectives

After studying the chapter, you should be able to:

- Describe the exercise/physical activity recommendations of the American College of Sports Medicine (ACSM), the U.S. Department of Health and Human Services, the Canadian Society for Exercise Physiology, and SHAPE America. Discuss the commonalities and differences of these reports.
- Discuss the application of each of the training principles in a cardiorespiratory training program.
- Explain how the FIT acronym is related to the overload principle.
- Differentiate among the methods used to classify exercise intensity.
- Calculate training intensity ranges by using different methods including the percentage of maximal heart rate, the percentage of heart rate reserve, and the percentage of oxygen consumption reserve.
- Discuss the merits of specificity of modality and cross-training in bringing about cardiovascular adaptations.
- Identify central and peripheral cardiovascular adaptations that occur at rest, during submaximal exercise, and at maximal exercise following an aerobic endurance or dynamic resistance training program.

Introduction

As described in [Chapter 1](#), there are two primary goals for *physical fitness*—sport related or health related. Physical fitness has long been recognized as a necessary foundation for athletic performance and remains so. However, as cardiovascular diseases escalated in the general population, fitness research evolved and emerged to the point where it became evident that physical fitness/physical activity was beneficial in improving health. Thus public health guidelines were established. Both the physical fitness and public health guidelines are relevant in practice for the exercise leader, exercise scientist, physical educator, physical therapist, and other associated professionals.

Early scientific investigations that led to the development of

training principles for the cardiovascular system almost always focused on the improvement of physical fitness, operationally defined as an improvement of maximal oxygen consumption ($\dot{V}O_{2\max}$). Such studies formed the basis for the guidelines developed by the American College of Sports Medicine (ACSM) and first published in 1978 as “the recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults.” These guidelines were revised in 1998 and again in 2011 to “Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: Guidance for prescribing exercise.” After 1978, these guidelines were increasingly applied not only to healthy adults intent on becoming more *fit* but also to individuals seeking only *health benefits* from exercise training.

Although evidence shows that health benefits accrue when fitness is improved, health and fitness are different goals, and exercise training and physical activity are different processes (Plowman, 2005). The quantity and quality of exercise required to develop or maintain cardiorespiratory fitness may not be (and probably is not) the same as the amount of physical activity required to improve and maintain cardiorespiratory health (ACSM, 2011; Haskell, 1994, 2005; Haskell et al., 2007; Nelson et al., 2007). Furthermore, most exercise science or physical education majors and competitive athletes who want or need high levels of fitness can handle physically rigorous and time-consuming training programs. Such programs, however, carry a risk of injury and are often intimidating to those who are sedentary, elderly, or obese.

Table 13.1 summarizes recommendations for cardiorespiratory fitness and health from leading authorities. Notice that the ACSM guidelines were prepared for developing and maintaining fitness, while the other guidelines were developed primarily to promote physical activity for health benefits. Since this chapter focuses on cardiovascular fitness and cardiorespiratory health, only the cardiovascular portion of the recommendations are included.

TABLE 13.1 Physical Activity and Exercise

Prescription for Health and Physical Fitness

| Source: Population | Frequency | Intensity | Duration | Notes |
|---|---|---|---|--|
| American College of Sports Medicine (ACSM) (2011): Adults | 5 d-wk ⁻¹ or 3 d-wk ⁻¹ or Combination | Moderate Vigorous Moderate + vigorous | ≥30 min-d ⁻¹ ≥20 min-d ⁻¹ | |
| Canadian Society for Exercise Physiology (CSEP) (2021): Adults 18–64 y | | Moderate to vigorous | 150 min-wk ⁻¹ | Activity can be done in 10 min bouts More activity provides more health benefits |
| Canadian Society for Exercise Physiology (CSEP) (2021): Children/Adolescents 5–17 y | Daily ≥3 d-wk ⁻¹ | Moderate to vigorous vigorous | 60 min-d ⁻¹ | More activity provides more health benefits |
| U.S. Department of Health and Human Services (2018): Adults | | Moderate to vigorous or Vigorous | 150–300 min-wk ⁻¹ 75–150 min-wk ⁻¹ | More activity provides more health benefits |
| U.S. Department of Health and Human Services (2018)*: Children/Adolescents 6–17 y | Daily ≥3 d-wk ⁻¹ | Moderate to vigorous and Vigorous | 60 min-d ⁻¹ | Should include vigorous activity at least 3x/week More activity provides more health benefits |

*Endorsed by SHAPE America

Application of the Training Principles to Improve Cardiorespiratory Fitness

Athletes and many fitness participants are explicitly interested in improving their cardiovascular fitness and this section will emphasize the application of the training principles for that goal. Of course, there is tremendous overlap between the benefits of increased physical exercise, an increase in cardiorespiratory fitness, and an increase in cardiovascular health.

The emphasis in the remainder of the chapter will be on changes that accompany a change in cardiorespiratory fitness ($\dot{V}O_{2\max}$) with the implementation of a training program. Additional information about physical fitness and physical activity in relation to cardiovascular disease is presented in [Chapter 15](#), Cardiovascular Disease Risk Factors and Physical Activity.

Obviously, there are other goals for exercise prescription and physical activity guidelines in addition to cardiovascular ones. There is also some overlap in the cardiovascular benefits of physical activity/exercise with other health and fitness areas, especially those pertaining to body weight/composition and metabolic function. Body weight aspects are discussed in the metabolic unit, and the recommendations for and benefits of resistance training, flexibility, and balance are discussed in the

neuromuscular unit.

This section of the chapter, focusing on how the training principles are applied for cardiovascular fitness, relies heavily on the cardiorespiratory portion of the 2011 ACSM Position Statement on the recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults. **Cardiorespiratory fitness** (also known as cardiovascular fitness) is defined as the ability to deliver and use oxygen during intense and prolonged exercise or work. Cardiorespiratory fitness is evaluated by measures of maximal oxygen consumption (

$\dot{V}O_{2\max}$). Sustained exercise training programs using these principles to improve $\dot{V}O_{2\max}$ are rarely included in the daily activities of children and adolescents. However, in the absence of more specific exercise prescription guidelines for younger individuals, these guidelines are often applied to adolescent athletes and youngsters in scientific training studies (Rowland, 2005).

Cardiorespiratory Fitness The ability to deliver and use oxygen during intense and prolonged exercise or work.

Specificity

Any activity that involves large muscle groups and is sustained for prolonged periods of time has the potential to increase cardiorespiratory fitness. This includes such exercise modes as aerobics, bicycling, cross-country skiing, various forms of dancing, jogging, rollerblading, rowing, speed skating, stair-climbing or stepping, swimming, and walking. Sports involving high-energy, nonstop action, such as field hockey, lacrosse, and soccer, can also positively benefit the cardiovascular system (ACSM, 2011). Furthermore, there is mounting evidence that short duration exercise training that uses high-intensity training

can lead to improvements in $\dot{V}O_{2\max}$ (Astorino et al., 2017; Bacon et al., 2013; Sultana et al., 2019).

Exercise is beneficial only if an individual participates in it. Therefore, for fitness participants, the choice of exercise

modalities should be based on interest, availability, and minimal risk of injury. An individual who enjoys the activity is more likely to adhere to the program. Although jogging or running may be the most time-efficient way to achieve cardiorespiratory fitness, these activities are not enjoyable for many individuals. They also have a relatively high incidence of overuse injuries. Therefore, other options should be available in fitness programs.

Although many different modalities can improve cardiovascular function, the greatest improvements in performance occur in the modality used for training, that is, there is modality specificity. For example, individuals who train by swimming improve more in swimming than in running (Magel et al., 1975), and individuals who train by bicycling improve more in cycling than in running (Pechar et al., 1974; Roberts and Alspaugh, 1972). Modality specificity has two important practical applications. First, to determine whether improvement is occurring, the individual should be tested in the modality used for training. Second, the more the individual is concerned with sports competition rather than fitness or rehabilitation, the more important the mode of exercise becomes. A competitive rower, for example, whether competing on open water or an indoor ergometer, should train mostly in that modality. Running, however, seems to be less specific than most other modalities; running forms the basis of many sports other than track or road races (Pechar et al., 1974; Roberts and Alspaugh, 1972; Wilmore et al., 1980).

Although modality specificity is important for competitive athletes, cross-training also has value. As used here, the term **cross-training** means the development or maintenance of cardiovascular fitness by training in two or more modalities either alternatively or concurrently. Two sets of athletes in particular are interested in cross-training: first, injured athletes, especially those with injuries associated with high-mileage running, who wish to prevent detraining and, second, an increasing number of athletes who participate in multisport competitions such as biathlons and triathlons and need to be conditioned in each.

Cross-Training The development or maintenance of

cardiovascular fitness by alternating between or concurrently training in two or more modalities.

Theoretically, both specificity and cross-training have value for a training program. Any form of aerobic endurance exercise affects both central and peripheral cardiovascular functioning. **Central cardiovascular adaptations** occur in the heart and contribute to an increased ability to deliver oxygen. Central cardiovascular adaptations are the same in all modalities when the heart is stressed to the same extent. Thus, many modalities can have the same overall training benefit by leading to central cardiovascular adaptations.

Central Cardiovascular Adaptations Adaptations that occur in the heart that increase the ability to deliver oxygen.

Peripheral cardiovascular adaptations occur in the vasculature or the muscles and contribute to an increased ability to extract oxygen. Peripheral cardiovascular adaptations are specific to the modality and the specific muscles used in that exercise. For example, additional capillaries will form to carry oxygen to habitually active muscles but not to habitually inactive ones. Other factors within exercising muscles such as mitochondrial density and enzyme activity also affect the body's ability to reach a high $\dot{V}O_{2\max}$. Specificity of modality operates because peripheral adaptations occur in the muscles that are used in the training. Thus, the specific activity—or closely related activities that mimic the muscle action of the primary sport—is needed to maximize peripheral adaptations. Examples of mimicking muscle action include side sliding or cycling for speed skating and water running in a flotation vest for jogging or running.

Peripheral Cardiovascular Adaptations Adaptations that occur in the vasculature or muscles that increase the ability to extract oxygen.

Table 13.2 lists several situations, in addition to the maintenance of fitness when injured, in which cross-training may be beneficial (Kibler and Chandler, 1994; O'Toole, 1992). Note that multisport athletes may or may not be limited to the sports in which they are competing. For example, although a duathlete needs to train for both running and cycling, this training will have the benefits of both specificity and cross-training. In addition, this athlete may also cross-train by doing other activities such as rollerblading or speed skating. Note also that cross-training can be recommended at any time for a fitness participant to help avoid physiological monotony and mental boredom. For a healthy competitive athlete, the value of cross-training is modest during the season. Cross-training is most valuable for single-sport competitive athletes during the transition (active rest) phase but may also be beneficial during the general preparation phase of periodization.

TABLE 13.2 Situations in Which Cross-Training Is Beneficial

| Reason | Fitness Participant | Competitive Athlete |
|---|-----------------------|--|
| Multisport participation | | General preparation phase, specific preparation phase, competitive phase |
| Injury or rehabilitation; fitness maintenance | As needed | As needed |
| Inclement weather | As needed | As needed |
| Baseline or general conditioning | Always | General preparation phase |
| Recovery | After intense workout | After intense workout or competition |
| Prevention of boredom and burnout | Always | Transition phase |

Source: Kibler and Chandler (1994).

Overload

There is a dose-response association between the volume of exercise and health/fitness outcomes; the greater the amount of physical activity, the greater the health/fitness benefits (ACSM, 2022). Exercise volume is the product of frequency, intensity, and time (duration) of exercise. An increase in volume provides an overload to the cardiovascular system that results in improved health/fitness. Overload of the cardiovascular system is achieved by manipulating the intensity, duration, and frequency of the training bouts. These variables are easily remembered by the acronym FIT (F, frequency; I, intensity; and T, time or duration). **Figure 13.1** presents the results of a study in which the components of overload were investigated relative to their effect on changes in $\dot{V}O_2 \text{ max}$. As the most critical component, intensity will be discussed first.

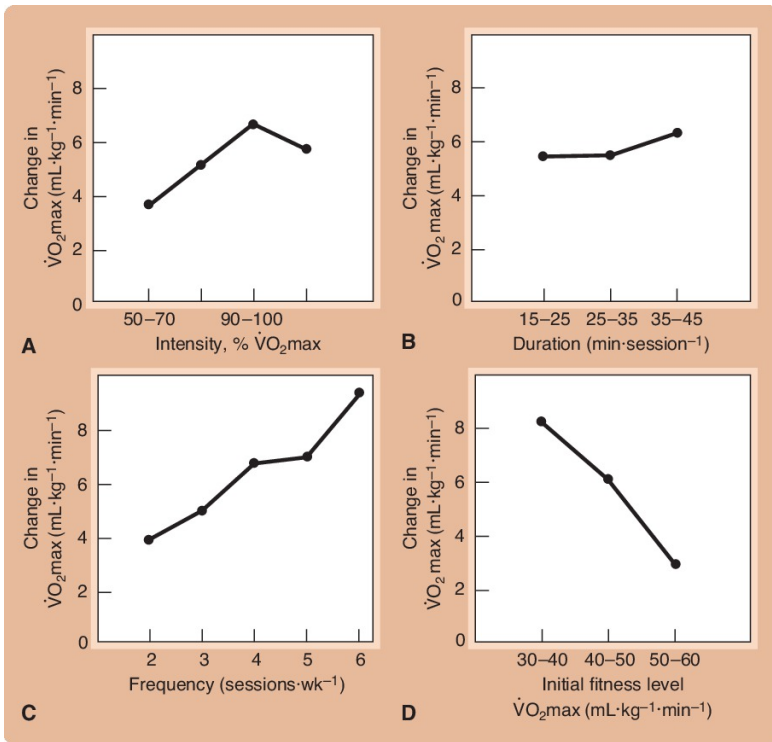


Figure 13.1 Changes in $\dot{V}O_2\text{max}$ Based on Intensity (A), Duration (B), Frequency (C), and Initial Fitness Level (D).



(D).

Source: Reprinted by permission from Springer Wenger, H. A., & G. J. Bell: The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Medicine*. 3(5):346–356 (1986). Copyright © 2012 Springer Nature.

Intensity

Figure 13.1A shows the relationship between change (Δ) in $\dot{V}O_2\text{max}$ and exercise intensity. In general, as exercise intensity increases, so do improvements in $\dot{V}O_2\text{max}$. The

greatest amount of improvement in $\dot{V}O_2\text{max}$ is seen following training programs that utilize exercise intensities of 90–100% $\dot{V}O_2\text{max}$.

In order to achieve such high-intensity training, individuals may alternate work and rest intervals (interval training). At exercise levels greater than 100% (supramaximal exercise), in which the total amount of training that can be performed decreases, improvement in $\dot{V}O_2\text{max}$ is somewhat less than is seen at 90–100% $\dot{V}O_2\text{max}$.

High-intensity interval training (HIIT) is a specific type of training that emphasizes short bouts of high-intensity exercise (often between 10 seconds and 2 minutes) interspersed with an active recovery period. Studies show that HIIT is as effective as moderate-intensity, continuous training (MCT), and in many studies superior to MCT for improving $\dot{V}O_2\text{max}$ (Sultana et al., 2019) and is effective in children, older adults, and for individual who are obese or have cardiovascular disease (Cvetkovic et al., 2018; Keech et al., 2020; Wu et al., 2021; Xie et al., 2017). HIIT is often preferable for fitness participants because it requires less time commitment and many athletes use this training to mimic the high-intensity burst required for competition.

Intensity, both alone and in conjunction with duration, is very important for improving $\dot{V}O_2\text{max}$. Intensity may be described in relation to heart rate, oxygen consumption, or rating of perceived exertion (RPE). Laboratory studies typically use $\dot{V}O_2$ for determining intensity, but heart rate and RPE are more practical for individuals outside the laboratory. **Table 13.3** includes techniques used to classify intensity and suggested percentages for very light to near maximal activity (ACSM, 2022; Mann et al., 2013). Note that these percentages and classifications are intended to be used when the exercise duration is 20–60 minutes and the frequency is 3–5 d·wk⁻¹.

TABLE 13.3 Classification of Intensity of Exercise Based on 20–60 Minutes of Endurance Training

| Classification of Intensity | %HRmax | %HRR/% $\dot{V}O_2R$ | Borg's Rating of Perceived Exertion |
|-----------------------------|-----------|----------------------|-------------------------------------|
| Very light | <57 | <30 | <9 |
| Light | 57–63 | 30–39 | 9–11 |
| Moderate | 64–76 | 40–59 | 12–13 |
| Vigorous | 77–95 | 60–89 | 14–17 |
| Near-maximal to maximal | ≥ 96 | ≥ 90 | ≥ 18 |

HEART RATE METHODS. Exercise intensity can be expressed as a percentage of either maximal heart rate (%HRmax) or heart rate reserve (%HRR). The %HRR method may be preferable because it reflects the rate of energy expenditure during exercise well, and because exercise intensity can be underestimated or overestimated when using %HRmax (ACSM, 2011). Both techniques, explained below, require maximal heart rate to be known or estimated. The methods are most accurate if the maximal heart rate is actually measured during an incremental exercise test to maximum. If such a test cannot be performed, maximal heart rate can be estimated. Despite its acceptance and widespread use, ACSM no longer recommends the traditional formula utilizing $220 - \text{age}$. Calculated values using this formula may either overestimate (in individuals over 40 years) or underestimate (in individuals under 40 years) the true HRmax by as much as $12\text{--}15 \text{ b}\cdot\text{min}^{-1}$ (ACSM, 2022; Miller et al., 1993; Wallace, 2006). Instead ACSM recommends the following equation by Tanaka et al. (2001) for both male and female adults:

$$13.1a \quad \text{maximal heart rate (b}\cdot\text{min}^{-1}) = 208 - [0.7 \times \text{age (yr)}]$$

For obese individuals, the following equation is recommended for added accuracy (Miller et al., 1993):

$$13.1b \quad \text{maximal heart rate (b}\cdot\text{min}^{-1}) = 200 - [0.5 \times \text{age (yr)}]$$

As indicated in Chapter 12, maximal heart rate is independent of age between the growing years of 6 and 16. This means that

the $220 - \text{age}$ formula cannot be used for youngsters at this age (Rowland, 2005). During this age span for both boys and girls, the average HRmax resulting from treadmill running is $200\text{--}205 \text{ b}\cdot\text{min}^{-1}$. Values obtained during walking and cycling are typically $5\text{--}10 \text{ b}\cdot\text{min}^{-1}$ lower at maximum. As with adults, measured values are always preferable but may not be practical. Therefore, the value estimated for HRmax for children and young adolescents should depend on modality rather than age.

Example

Calculate the predicted or estimated HRmax for a 28-year-old female with a normal body composition.

$$\text{HRmax} = 220 - \text{age} = 220 - (28 \text{ yr}) = 192 \text{ b}\cdot\text{min}^{-1}$$

If the female is obese, her estimated maximal heart rate is

$$\begin{aligned}\text{HRmax} &= 200 - (0.5 \times \text{age}) = 200 - (0.5 \times 28 \text{ yr}) \\ &= 186 \text{ b}\cdot\text{min}^{-1}\end{aligned}$$

Once the maximal heart rate is known or estimated, the %HRmax is calculated as follows:

Target exercise heart rate (TExHR) = maximal heart rate ($\text{b}\cdot\text{min}^{-1}$) \times percentage of maximal heart rate (expressed as a decimal)

or

$$13.2 \quad \text{TExHR} = \text{HRmax} \times \% \text{HRmax}$$

1. Determine the desired intensity of the workout.
2. Use **Table 13.3** to find the %HRmax associated with the desired exercise intensity.
3. Multiply the percentages (as decimals) times the HRmax.

Example

Determine the appropriate HR training range for a moderate workout for a nonobese 28-year-old using the HRmax.

1. Determine the HRmax:
 $220 - 28 = 192 \text{ b}\cdot\text{min}^{-1}$
2. Determine the desired intensity of the workout. **Table 13.3** shows 64–76% of HRmax corresponds to a moderate workout.
3. Multiply the percentages (as decimals) times the HRmax for the upper and lower exercise limits. Thus:

| | | |
|-----------------------------|---------------|---------------|
| HRmax | 192 | 192 |
| desired intensity (decimal) | $\times 0.64$ | $\times 0.76$ |
| Target HR Range (rounded) | 123 | 146 |

Therefore, an HR of $120 \text{ b}\cdot\text{min}^{-1}$ represents 64% of HRmax and an HR of $143 \text{ b}\cdot\text{min}^{-1}$ represents 76% of HRmax. To exercise between 64% and 76% of HRmax, a moderate workload, this individual should keep the heart rate between 120 and $143 \text{ b}\cdot\text{min}^{-1}$.

It is always best to provide the potential exerciser with a target heart rate range rather than a threshold heart rate. In fact, the term threshold may be a misnomer since no particular percentage has been shown to be a minimally necessary threshold for all individuals in all situations ([Haskell, 1994](#)). Evidence of a minimum threshold is supported in some, but not all, studies and may be related to the initial fitness level of the individual, the precise program followed, and individual variability of response ([ACSM, 2011](#)). Additionally, a range allows for the heart rate drift that occurs in moderate to heavy exercise after about 30 minutes and for variations in weather, terrain, fluid replacement, and other influences. The upper limit serves as a boundary against overexertion.

Alternatively, a target heart rate range can be calculated as a percentage of heart rate reserve (%HRR), a technique also called the *Karvonen* method. It involves additional information and calculations but has the advantage of considering resting heart rate. The steps are as follows:

1. Determine the heart rate reserve (HRR) by subtracting the resting heart rate from the maximal heart rate:

$$\text{Heart rate reserve (b} \cdot \text{min}^{-1}) = \text{maximal heart rate (b} \cdot \text{min}^{-1}) - \text{resting heart rate (b} \cdot \text{min}^{-1})$$

or

$$13.3 \quad \text{HRR} = \text{HR}_{\text{max}} - \text{RHR}$$

The resting heart rate is best determined when the individual is truly resting, such as immediately on awakening in the morning. However, for purposes of exercise prescription, this can be a seated or standing resting heart rate, depending on the exercise posture. Heart rates taken before an exercise test are anticipatory, not resting, and are higher than actual resting heart rate.

2. Choose the desired intensity of the workout.
3. Use **Table 13.3** to find the %HRR associated with the desired exercise intensity.
4. Multiply the percentages (as decimals) for the upper and lower exercise limits by the HRR and add RHR using Equation 13.4.

$$\text{Target exercise heart rate (b} \cdot \text{min}^{-1}) = [\text{heart rate reserve (b} \cdot \text{min}^{-1}) \times \text{percentage of heart rate reserve (expressed as a decimal)}] + \text{resting heart rate (b} \cdot \text{min}^{-1})$$

or

$$13.4 \quad \text{TE}_{\text{HR}} = (\text{HRR} \times \% \text{HRR}) + \text{RHR}$$

Example

Determine the appropriate HR range for a moderate workout for a normal-weight 28-year-old using the HRR method, assuming an RHR of $80 \text{ b} \cdot \text{min}^{-1}$.

1. Determine the HRR:

$$188 \text{ b} \cdot \text{min}^{-1} - 80 \text{ b} \cdot \text{min}^{-1} = 108 \text{ b} \cdot \text{min}^{-1}$$

2. Determine the desired intensity of the workout. Again, using **Table 13.3**, 40–59% of HRR corresponds to a moderate workout. This reinforces the point that the %HR_{max} does not equal %HRR.
3. Multiply the percentages (as decimals) for the upper and lower exercise limits by the HRR. Thus:

| | | |
|-----------------------------|-------------------------|--------------------------|
| HRR | 108 | 108 |
| desired intensity (decimal) | $\times \frac{0.4}{43}$ | $\times \frac{0.59}{64}$ |

4. Add RHR as follows:

| | | |
|---|--------------------|--------------------|
| resting HR | 43 | 64 |
| target HR training range ($\text{b} \cdot \text{min}^{-1}$) | $\frac{+ 80}{123}$ | $\frac{+ 80}{144}$ |

Therefore, an HR of $123 \text{ b} \cdot \text{min}^{-1}$ represents 40% of HRR and an HR of $144 \text{ b} \cdot \text{min}^{-1}$ represents 59% of HRR. So, in order to be exercising between 40% and 59% of HRR, a moderate workload, this individual should keep the heart rate between 123 and $144 \text{ b} \cdot \text{min}^{-1}$.

This heart rate range ($123\text{--}144 \text{ b} \cdot \text{min}^{-1}$) is only slightly different from the one calculated by using %HR_{max} ($120\text{--}143 \text{ b} \cdot \text{min}^{-1}$), but the %HRR technique has the advantage of taking into account any training adaptations that occur in resting heart rate for future adjustments and is more closely tied with energy expenditure.

Work through the problem presented in the [Check Your Comprehension 1 box](#), paying careful attention to the influence of

resting heart rate when determining the training heart rate range using the heart rate reserve (Karvonen) method. Check your answer in Appendix C.

CHECK YOUR COMPREHENSION 1

Calculate the target HR range for a light workout for two normal-weight individuals, using the %HRmax and %HRR methods and the following information.

| | Age | RHR |
|--------|-----|-----|
| Mei | 50 | 62 |
| Serena | 60 | 82 |

Maximal heart rate declines with advancing age in adults. Thus, the heart rate needed to achieve a given intensity level, calculated by either the maximal heart rate or heart rate reserve method, decreases with age.

OXYGEN CONSUMPTION/% R METHODS. In a laboratory setting where an individual has been tested for $\dot{V}O_{2\max}$ and equipment is available for monitoring $\dot{V}O_2$ during training, % $\dot{V}O_{2R}$ may be used to prescribe exercise intensity. Oxygen reserve is parallel to heart rate reserve in that it is the difference between a resting and maximal value. It is calculated according to the formula:

$$\text{Oxygen consumption reserve (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \text{maximal oxygen consumption (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) - \text{resting oxygen consumption (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$$

or

$$13.5 \quad \dot{V}O_{2R} = \dot{V}O_{2\max} - \dot{V}O_{2\text{rest}}$$

Target exercise oxygen consumption is then determined by the equation:

Target exercise oxygen consumption
 $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = [\text{oxygen consumption reserve}$
 $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) \times \text{percentage of oxygen consumption reserve (expressed as a decimal)}] + \text{resting}$
 oxygen consumption $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$
 or

$$13.6 \quad \text{TE} \dot{V}\text{O}_2 = (\dot{V}\text{O}_2\text{R} \times \% \dot{V}\text{O}_2\text{R}) + \dot{V}\text{O}_2\text{rest}$$

Use these steps to calculate training intensity with this method:

1. Choose the desired intensity of the workout.
2. Use **Table 13.3** to find the % $\dot{V}\text{O}_2\text{R}$ for the desired exercise intensity.
3. Multiply the percentage (as a decimal) of the desired intensity times the $\dot{V}\text{O}_{2\text{max}}$.
4. Add the resting oxygen consumption to the obtained values. Note that this may be an individually measured value or the estimated $3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ that represents 1 MET.
5. Because oxygen drifts, as does heart rate, it is best to use a target range.

Basing the intensity of a workout on % $\dot{V}\text{O}_2\text{R}$ is not very practical because most people do not have access to the needed equipment. However, the technique can be modified for individuals who wish to use it. First, one can use the formula in Appendix B (The Calculation of Oxygen Consumed Using Mechanical Work or Speed of Movement) to solve for the workload (velocity of level or inclined walking or running; resistance for arm cranking or leg cycling; height or cadence for bench stepping). Then the prescription can be based on minutes per mile, cadence of stepping at a particular height, or load setting at a specific revolutions per minute pace. Because the oxygen cost of submaximal exercise is higher for children and changes as they age and grow, this technique is rarely used for children ([Strong et al., 2005](#)).

A second practical use of the $\dot{V}\text{O}_2\text{R}$ approach is based on

the direct relationship between heart rate and oxygen consumption. Look closely again at **Table 13.3**. Note that the column for % $\dot{V}O_2R$ is also the column for %HRR; that is, any given %HRR has an equivalent % $\dot{V}O_2R$ in adults. For example, an adult who is working at 50% HRR is also working at 50% $\dot{V}O_2R$. Therefore, heart rate can be used to estimate oxygen consumption when an individual is training or competing.

The equivalency between % $\dot{V}O_2R$ and %HRR has been demonstrated experimentally in multiple studies including young and elderly adult males and females and for different modalities (Swain, 2000); however, not all studies have confirmed this relationship and the topic continues to be an active area of research (Mann et al., 2013; Marini et al., 2021).

Although there is also a rectilinear relationship between %HRR and % $\dot{V}O_2R$ in children and adolescents, this relationship is not the same as for adults. In children and adolescents, the two percentages are not equal. In one study, 50–85% $\dot{V}O_2R$ was found to equate with 60–89% HRR in boy and girls 10–17 years of age (Hui and Chan, 2006). Therefore, it is probably best to simply use either %HRmax or %HRR when prescribing exercise intensity for children and adolescents, and not make any equivalency assumption with % $\dot{V}O_2R$.

Table 13.4 shows how long one can run at a specific percentage of maximal oxygen consumption. The **Check Your Comprehension 2: Case Study 1** provides an example of how this information can be used in training and competition. Take the time now to work through the situation described in the box.

TABLE 13.4 Time Period of a Selected % O_{2max} Can Be Sustained during Running

| % $\dot{V}O_2$ max | Time (min) |
|--------------------|------------|
| 100 | 8–10 |
| 97.5 | 15 |
| 90 | 30 |
| 87.5 | 45 |
| 85 | 60 |
| 82.5 | 90 |
| 80 | 120–210 |

Source: Daniels and Gilbert (1979).

CHECK YOUR COMPREHENSION 2–Case Study

Four friends meet at the track for a noontime workout. Their physiological characteristics are as follows. (The estimated $\dot{V}O_2$ max values have been calculated from a 1-mi running test.)

| Individual | Age (y) | Estimated $\dot{V}O_2$ max (mL·kg ⁻¹ ·min ⁻¹) | Resting HR (b·min ⁻¹) |
|------------|---------|---|--------------------------------------|
| Janet | 23 | 52 | 60 |
| Juan | 35 | 64 | 48 |
| Mark | 22 | 49 | 64 |
| Gail | 28 | 56 | 58 |

The following oxygen requirements have been calculated for a given speed based on the equations that are presented in Appendix B.

| Speed (mph) | Oxygen Requirement (mL·kg ⁻¹ ·min ⁻¹) |
|-------------|---|
| 4 | 27.6 |
| 5 | 30.3 |
| 6 | 35.7 |
| 7 | 41.0 |
| 8 | 46.4 |
| 9 | 51.7 |

The friends wish to run together in a moderate workout. Assume temperate weather conditions.

1. At what speed should they be running?
2. What heart rate should be achieved by each runner at that pace?

Check your answers with the ones provided in Appendix C.

RATING OF PERCEIVED EXERTION METHODS. The third way exercise intensity can be prescribed is by a subjective impression of overall effort, strain, and fatigue during the activity. Individuals are simply asked to determine the number that represents how they feel at any given time during exercise based on a numerical scale. This impression is known as a **rating of perceived exertion** (RPE). Although RPE can be used alone as a prescriptive method, it is best used to fine-tune an exercise prescription (ACSM, 2011, 2022). Perceived exertion is typically measured using either Borg's 6–20 RPE scale or the revised 0–10+ Category Ratio Scale (Borg, 1982, 1998). **Table 13.5** presents and compares both scales (Borg, 1982). The RPE scale is designed so that these perceptual ratings rise in a rectilinear fashion with heart rate, oxygen consumption, and mechanical workload during incremental exercise; thus, it is the primary scale used for cardiovascular exercise prescription (**Table 13.3**). The CR10 scale increases in a positively accelerating curvilinear fashion and closely parallels the physiological responses of pulmonary ventilation and blood lactate. **Chapter 5** describes the use of these scales for metabolic exercise prescription.

Rating of Perceived Exertion A subjective impression of overall physical effort, strain, and fatigue during acute exercise.

TABLE 13.5 Scales for Ratings of Perceived Exertion

| RPE Scale | CR10 Scale |
|--------------------|----------------------------|
| 6 | 0.0 |
| 7 Very, very light | 0.0 |
| 8 | 0.5 Just noticeable |
| 9 Very light | 1.0 Very weak |
| 10 | 1.5 |
| 11 Fairly light | 2.0 Light/weak |
| 12 | 3.0 Moderate |
| 13 Somewhat hard | 3.5 |
| | 4.0 Somewhat strong |
| 14 | 4.5 |
| | 5.0 |
| 15 Hard | 5.5 |
| | 6.0 |
| 16 | 6.5 Very strong |
| | 7.0 |
| 17 Very hard | 7.5 |
| | 8.0 |
| 18 | 9.0 |
| 19 Very, very hard | 10.0 Extremely strong |
| 20 | 10+ (~12) Highest possible |

Source: Reprinted with permission from Borg, G. A. V.: Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*. 14(5):377–381 (1982). Copyright ©1982 The American College of Sports Medicine.

Both the Borg RPE and CR10 scales are intended for use with postpubertal adolescents and adults. Because children (~6–12 years) have difficulty consistently assigning numbers to words or phrases to describe their exercise-related feelings, [Robertson et al. \(2002\)](#) developed the Children's OMNI Scale of Perceived Exertion. The OMNI Scale uses numerical, pictorial, and verbal descriptors. The original scale, depicted in **Figure 13.2**, was validated for cycling activity. Since then, variations have been developed for walking/running ([Utter et al., 2002](#)) and stepping ([Robertson et al., 2005](#)). Children have been shown to be able to self-regulate their cycling exercise intensity using the OMNI Scale ([Robertson et al., 2002](#)). In addition, observers can determine children's exercise intensity using the OMNI Scale ([Robertson et al., 2006](#)). This could be very helpful for teachers.

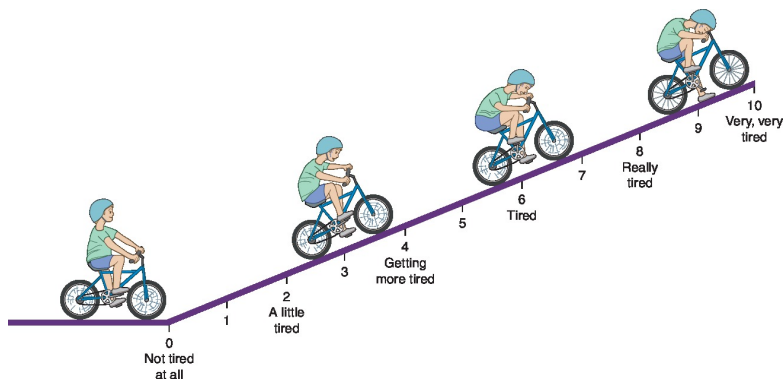


Figure 13.2 Children's OMNI Scale of Perceived



Exertion.

Source: Reprinted with permission from Robertson, R. J., F. L. Goss, N. F. Boer, J. A. Peoples, A. J. Foreman, I. M. Dabayebeh, N. B. Millich, G. Balasekaran, S. E. Riechman, J. D. Gallagher, & T. Thompkins: Children's OMNI scale of perceived exertion: Mixed gender and race validation. *Medicine & Science in Sports & Exercise*. 32(3): 452–458 (2000). Copyright ©2000 The American College of Sports Medicine.

The classification of exercise intensity and the corresponding relationships among %HRmax, % $\dot{V}O_2R$, % $\dot{V}O_{2max}$, %HRR, and RPE presented in **Table 13.3** have been derived from and are intended for use with land-based activities in moderate environments.

Whether a water activity is performed horizontally, as in swimming, or vertically, as in running or water aerobics, postural and pressure changes shift the blood volume centrally and cause changes in blood pressure, cardiac output, resistance, and respiration. Although the magnitude of changes in the cardiovascular system vary considerably among individuals, the most consistent changes are lower submaximal HR (8–12 b·min⁻¹) at any given $\dot{V}O_2$, a lower HRmax (~15 b·min⁻¹), and a lower $\dot{V}O_{2max}$ when exercise is performed in the water. A greater reliance on anaerobic metabolism is evident, and the RPE is higher in water than at the same workload on land (Svedenhag and Seger, 1992). The lower HR is probably a compensation for the increased stroke volume when blood is shifted centrally. As a result, the HR prescription should be about 10% lower for water workouts than for land-based workouts. For example, if an individual normally works out at 75% HRmax on land, the prescription for an equivalent workout in the water should be 65% HRmax.

FOCUS ON APPLICATION | *Clinically Relevant*

Ratings of Perceived Exertion and Environmental Conditions

Rating of perceived exertion (RPE) is a useful, common way to assist in exercise intensity prescription. Note, however, that the estimation of RPE (when exercisers are asked how hard they feel they are exercising) and actual physiological responses to exercise are affected by environmental conditions. Both HR and RPE are higher when exercise is

performed in a hot environment (or while wearing clothing that interferes with heat dissipation) compared to a thermoneutral environment. The relationship between HR and RPE is also affected by environmental conditions. At any given RPE, HR is 10–15 $\text{b}\cdot\text{min}^{-1}$ higher in the heat (Maw et al., 1993). When exercisers are instructed to produce a given exercise intensity based on a specific RPE, they usually automatically adjust the exercise intensity to environmental conditions. For example, running at 8 $\text{min}\cdot\text{mi}^{-1}$ in thermal neutral conditions may elicit an RPE estimation of 13. However, in hot humid conditions, an individual may only run at 9 $\text{min}\cdot\text{mi}^{-1}$ at an RPE of 13.



Regardless of the method chosen to prescribe exercise intensity, always consider three factors:

1. Exercise intensity should generally be prescribed within a range. Many activities require different levels of exertion throughout the activity. This is particularly true of games and athletic activities, but it also applies to activities like jogging and bicycling, in which changes in terrain can greatly affect exertion. In addition, a range allows for cardiovascular and oxygen consumption drifts during prolonged exercise.
2. Exercise intensity must be considered in conjunction with duration and frequency.

- a. Intensity cannot be prescribed without regard to duration. These two variables are inversely related: In general, the more intense an activity is, the shorter it should be.
 - b. The appropriate intensity of exercise also depends on the individual's fitness level and, to some extent, the point within his or her fitness program. Individuals should begin an exercise program at a low exercise intensity and gradually increase intensity in a steploading progression until the desired level is achieved.
3. Using heart rate or perceived exertion to monitor training sessions, rather than merely time over distance, allows the influence of weather, terrain, surfaces, and the way the individual is responding to be taken into account when assessing the person's adaptation to a training program.

Duration/Time (or Quantity)

As shown in **Figure 13.1B**, improvements in $\dot{V}O_2\text{max}$ can be achieved when exercise is sustained for durations of 15–45 minutes ([Wenger and Bell, 1986](#)). Slightly greater improvements are achieved from longer sessions (35–45 minutes) than from shorter sessions (either 15–25 or 25–35 minutes) and high-intensity exercise is more effective than moderate-intensity

exercise for improving $\dot{V}O_2\text{max}$. The current guidelines for public health (**Table 13.1**) reflect these results and recommendations. That is, 30–60 min·d⁻¹ (≥ 150 min·wk⁻¹) of moderate-intensity exercise or 20–60 min·d⁻¹ (≥ 75 min·wk⁻¹) of vigorous intensity exercise or a combination of moderate and vigorous intensity exercise daily are recommended for optimal improvement. In general, 1 min of vigorous exercise is the equivalent of 2 min of moderate exercise. However, these recommendations do not mean that exercise sessions less than 20 minutes are not valuable for health benefits or that at least 20 minutes must be accumulated during one exercise session. Evidence is available that exercise bouts of 10 minutes or less are associated with favorable health-related outcomes. Some exercise is always better than no exercise (ACSM, 2022; [USDHHS, 2018](#)).

Thus, for individuals who claim they do not have time to

exercise, suggesting a short brisk walk in the morning (perhaps to work or walking the kids to school), at noon (to a favorite restaurant and back), and in the evening (perhaps walking to a movie or taking the dog for a walk) might make it easier to achieve a total of 30 minutes of activity than having to do it all at once. The benefit of split sessions is particularly important for those currently sedentary, minimally active, or in rehabilitation programs. An injured person may simply not be able to exercise for a long period, while short bouts may be possible spread throughout the day.

Similarly, although it is most often the case in practice, the duration guidelines do not mean that the exercise must be continuous within any single session. The technique of interval training (alternating predetermined periods of exercise and rest) often used by athletes can be very effective in improving cardiorespiratory fitness (ACSM, 2011, 2022). Several recent reviews and meta-analyses have suggested that compared with relatively long-duration continuous moderate exercise, high-intensity interval training (HIIT) can result in an equal or higher improvement in cardiovascular fitness and health in a much shorter period of time. The pattern for HIIT can be something like 4×4 four min runs at 90–95% HRmax with 3 minutes of recovery between each repeat (ACSM, 2022). As little as 3 high-intensity interval sessions per week have been shown to improve aerobic capacity and markers of disease risk in both healthy individuals and individuals with cardiovascular disorders.

STEP COUNTS Pedometer step counts are another way of assessing the quantity of exercise. The “traditional” recommendation for the attainment of 10,000 or more steps per day actually began as a marketing campaign shortly before the 1964 Tokyo Olympic Games. A Japanese company marketed a pedometer called the “Manpo-kei.” Translated “man” means “10,000”; “po” means “steps”; and “kei” means “meter.” The campaign and pedometer were hugely successful. However, research evidence for this goal is incomplete. Despite sounding overwhelming to many, walking 10,000 steps per day is doable. However, this does not account for age, sex, and/or health status. In addition, humans often want to know “what is the minimum number of steps I can take and still receive health benefit?” So, in

the ensuing years this recommendation has been studied and refined for different populations. **Table 13.6** presents what is currently recommended for adults and children of different ages and health conditions (Tudor-Locke, et al., 2011a, 2011b, 2011c) to meet moderate to vigorous physical activity guidelines. The total steps per day values are without regard to the speed of the steps taken. Less than 5,000 steps per day in adults without disabilities and/or chronic diseases is said to constitute a sedentary, physically inactive lifestyle (Tudor-Locke et al., 2013).

TABLE 13.6 Recommended Daily Step Counts and Cadence

| Population | Total Steps·d ⁻¹ | Number MVPA* | Population | Cadence Steps·min ⁻¹ | |
|--|------------------------------|--------------|------------|---------------------------------|----------|
| | | | | Moderate | Vigorous |
| Adults | | | Adults | | |
| 20–60 y | 7,000–7,500 | ≥3,000 | 20–60 y | 100 | 130 |
| ≥65 y | 7,000 | ≥3,000 | 61–85 y | 100 | |
| Children | | | Children | | |
| 4–6 y | 10,000 | ≥6,000 | 6–8 y | 125 | 155 |
| 6–11 y | 13,000 | ≥6,000 | 9–11 y | 115 | 130 |
| Boys | 11,000 | ≥6,000 | 12–14 y | 110 | 125 |
| Girls | 10,000 | ≥6,000 | 15–17 y | 105 | 125 |
| 12–19 y | | | 18–20 y | 90 | 125 |
| Individuals with disabilities and/or chronic illness | ≥4,000 as medically possible | | | | |

*MVPA = moderate to vigorous physical activity.
 MPA = 3 to ≤6 METs adults and 4 to ≤6 METs children.
 VPA = ≥6 METs.
 Based on 30 min·d⁻¹ for adults and 60 min·d⁻¹ for children.
Sources: Tudor-Locke et al. (2011a, 2011b, 2011c, 2013, 2018, 2019a, 2019b, 2020).

Another way to obtain sufficient moderate to vigorous physical activity by walking is to incorporate step speed or cadence, defined as the number of steps per minute. **Table 13.6** also shows these cadence recommendations (Tudor-Locke et al., 2019a, 2019b, 2020). Moderate-intensity walking (at ~100 steps per minute) for 30 minutes equates to 3,000 steps and can be used as a realistic minimal daily goal for moderate activity for adults. Current recommendations for children are for 60 minutes of moderate physical activity or at least 6,000 steps per day. However, cadence recommendations for children are very dependent on age and vary from 125 for the younger ages to 90

for the oldest. Note that there are two values for the cadence for 20 years olds (90 and 100 steps per minute) in **Table 13.6**. This is the result of different samples in different research studies. These values are close enough that either the 90 or 100 count can be used but 100 steps per minute is probably the easiest to recommend and remember as it will be the same throughout adulthood then.

Research studies are beginning to be available that provide evidence that either or both steps per day and peak 30-min step cadence have beneficial effects on cardiometabolic risk factors for metabolic syndrome ([Adams et al., 2019](#); [Tudor-Locke et al., 2017](#)), cardiovascular events, type 2 diabetes, and all-cause mortality ([Hansen et al., 2020](#); [Kraus et al., 2019](#); [Saint-Maurice et al., 2020](#)). More work needs to be done, but, as seen in **Figure 13.3**, there is strong evidence for a dose-response relationship between steps per day and all-cause mortality ([Saint-Maurice et al., 2020](#)).

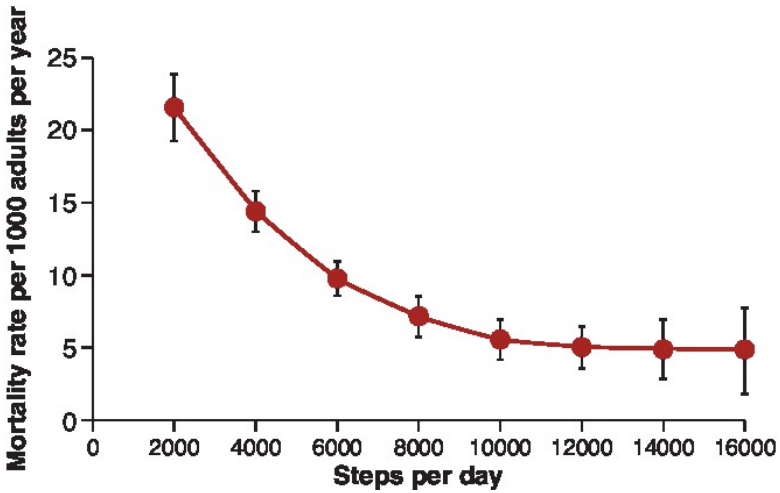


Figure 13.3 Dose-Response Relationship between Step Count per day and Unadjusted All-Cause Mortality. Based on data from [Saint-Maurice et al. \(2020\)](#).

Activity monitors, such as Fitbit, Applewatch, and Garmin, appear to be effective tools for promoting physical activity and

have gained widespread acceptance. Most of these devices provide information that quantifies exercise (in time, steps and/or calories) and also provides heart rate response to exercise.

Frequency

If the total work done or the number of exercise sessions is held constant, there is basically no difference in the improvement of

$\dot{V}O_2\text{max}$ over 2, 3, 4, or 5 days (Pollock, 1973). However, when these conditions are not adhered to, there does seem to be an advantage to more frequent training. As Figure 13.1C shows,

the improvement in $\dot{V}O_2\text{max}$ is proportional to the number of training sessions per week (Wenger and Bell, 1986). In general, training fewer than 2 d·wk⁻¹ did not result in improvements in

$\dot{V}O_2\text{max}$. Likewise, further improvement in $\dot{V}O_2\text{max}$ was not meaningful when exercise participation was increased from 4 to 5 days a week. Although the graph in Figure 13.1C reveals

that there is the potential for further improvement in $\dot{V}O_2\text{max}$ if a 6th day of training is added, it is not generally recommended for those pursuing fitness goals because of a higher incidence of injury and fatigue. The optimal frequency for improving

$\dot{V}O_2\text{max}$ for all intensities appears to be 4 d·wk⁻¹.

The ACSM (2011, 2022) recommendation for healthy individuals is a frequency of 3–5 with moderate intensity being done 5 d·wk⁻¹ or vigorous done 3 d·wk⁻¹ with the assumption that these days are nonconsecutive. However, individuals at very low fitness levels may start a program of only 2 d·wk⁻¹ and only 1–2 d·wk⁻¹ at moderate to vigorous intensity can bring about health benefits. Athletes may train 6 d·wk⁻¹ as a way of increasing their total training volume. In this case, “easy” and “hard” days should be interspersed within most microcycles. Cross-training may also be employed. A special case is the “weekend warrior,” that is, the individual who accumulates a large volume of physical activity in a 2-day weekend. Existing evidence supports the possibility of benefit, although the injury risks are unknown (ACSM, 2011).

Individualization

Fitness programs should be individualized for participants. Not only do individual goals vary, but individuals also respond to and adapt to exercise differently. One of the major determinants of the individual's response is genetics. Another major determinant is initial fitness level. **Figure 13.1D** clearly shows that independent of frequency, intensity, or duration, the greatest

improvements in $\dot{V}O_2\text{max}$ occur in individuals with the lowest initial fitness level. Thus, both absolute and relative increases in $\dot{V}O_2\text{max}$ are inversely related to one's initial fitness level. Although improvements in $\dot{V}O_2\text{max}$ are smallest in highly fit individuals, at this level, small changes may have a significant influence on performance, because many athletic events are won by fractions of a second.

The initial fitness level generalization also applies to health benefits. Health benefits are greatest when a person moves from a low fitness to a moderately fit category. Most sedentary individuals can accomplish this if they participate in a regular, low-to-moderate-endurance exercise program ([Haskell, 1994](#)).

Rest/Recovery/Adaptation

Training programs can be divided into initial, improvement, and maintenance stages. The initial stage usually lasts 1–6 weeks, although this varies considerably among individuals. This stage should include low-level aerobic activities that cause a minimum of muscle soreness or discomfort. It is often prudent to begin an exercise program at an intensity lower than the desired exercise range (40–59% HRR). For individuals at very low levels of fitness, a discontinuous or interval format training program may be warranted, using several repetitions of exercise, each lasting 2–5 minutes. Rest periods between the intervals reduce the overall stress on the individual by allowing intermittent recovery. Frequency may vary from short, light daily activity to longer exercise sessions two or three times per week. Adaptation occurs during the off days. An important part of this stage is helping the individual achieve the “habit” of exercise and orthopedically adapt to workouts. Soreness, discomfort, and injury should be

avoided to encourage the individual to continue.

During the improvement stage, significant changes in physiological function indicate that the body is adapting to the stress of the training program. Again, the individual adapts during rest days when the body is allowed to recover. Adaptation has occurred when the same amount of work is accomplished in less time, when the same amount of work is accomplished with less physiological (homeostatic) disruption, when the same amount of work is accomplished with a lower perception of fatigue or exertion, or when more work is accomplished. Once the body has adapted to the stress of exercise, progression is necessary to induce additional adaptations, or maintenance is required to preserve the adaptations.

Progression

Once adaptation occurs, the workload must be increased for further improvement to occur. The workload can be increased by manipulating the frequency, intensity, and duration of the exercise. Increasing any of these variables effectively increases the volume of exercise and thus provides the overload necessary for further adaptation. The rate of progression depends on the individual's needs or goals, fitness level, health status, exercise tolerance, and age but should always be done in a steploading fashion of 2–3 weeks of increase followed by a decrease for recovery and regeneration before increasing training volume again.

The improvement stage of a training program typically lasts 4–8 months and is characterized by relatively rapid progression. For an individual with a low fitness level, the progression from a discontinuous activity to a continuous activity should occur first. Then the duration of the activity should be increased. This increase in duration should not exceed 5–10 minutes every 1–2 weeks over 4–6 weeks or 20% per week until 20–30 minutes of moderate to vigorous intensity activity can be completed, and 10% per week thereafter. Frequency can then be increased. Intensity should be the last variable to be increased. Adjustments of no more than 5% HRR every six exercise sessions (1.5–2 weeks) are typically well tolerated. Progression in any component

of the exercise prescription should be gradual and the individual monitored for any excessive shortness of breath, fatigue, or muscle soreness that may require downward adjustments (ACSM, 2022).

The principles of adaptation and progression are intertwined. Adaptation and progression may be repeated several times until the desired level of fitness or performance is achieved. Each time an exercise program is modified, there will be a period of adaptation that may be followed by further progression if desired.

Maintenance

Athletes often vary their training levels according to a general preparation phase (off-season), specific preparation phase (preseason), competition phase (in-season), and transition phase (active rest) as described in the periodization section in [Chapter 1](#). In transition and competitive phases, they can shift to a maintenance schedule. For rehabilitation and fitness participants, maintenance typically begins after the first 4–8 months of training. Reaching the maintenance stage indicates that the individual has achieved a personally acceptable level of cardiorespiratory fitness and is no longer interested in increasing the conditioning load.

After attaining a desired level of aerobic fitness, this level can be maintained either by continuing the same volume of exercise or by decreasing the volume of training, as long as intensity is maintained. **Figure 13.4** shows the results of research that

investigated changes in $\dot{V}O_{2\max}$ with 10 weeks of relatively intense interval training and a subsequent 15-week reduction in training frequency (**Figure 13.4A**), duration (**Figure 13.4B**), or intensity (**Figure 13.4C**) ([Hickson and Rosenkoetter, 1981](#); [Hickson et al., 1982, 1985](#)). When training frequency was reduced from 6 d·wk⁻¹ to 4 or 2 d·wk⁻¹ and intensity and duration were held constant, training-induced improvements in

$\dot{V}O_{2\max}$ were maintained. Similarly, when training duration was reduced from 40 to 26 or 13 minutes, improvements in

$\dot{V}O_{2\max}$ were maintained or continued to improve. However, when intensity was reduced by two thirds, improvements in

$\dot{V}O_{2\max}$ were not maintained. These results indicate that intensity plays a primary role in maintaining cardiovascular fitness. Thus, although the total volume of exercise is most important for attaining a given fitness level, intensity is most important for maintaining the achieved fitness level. That is, in general, more exercise is required to improve cardiorespiratory fitness and health than is required to maintain the resultant improvements ([ACSM, 2011](#)).

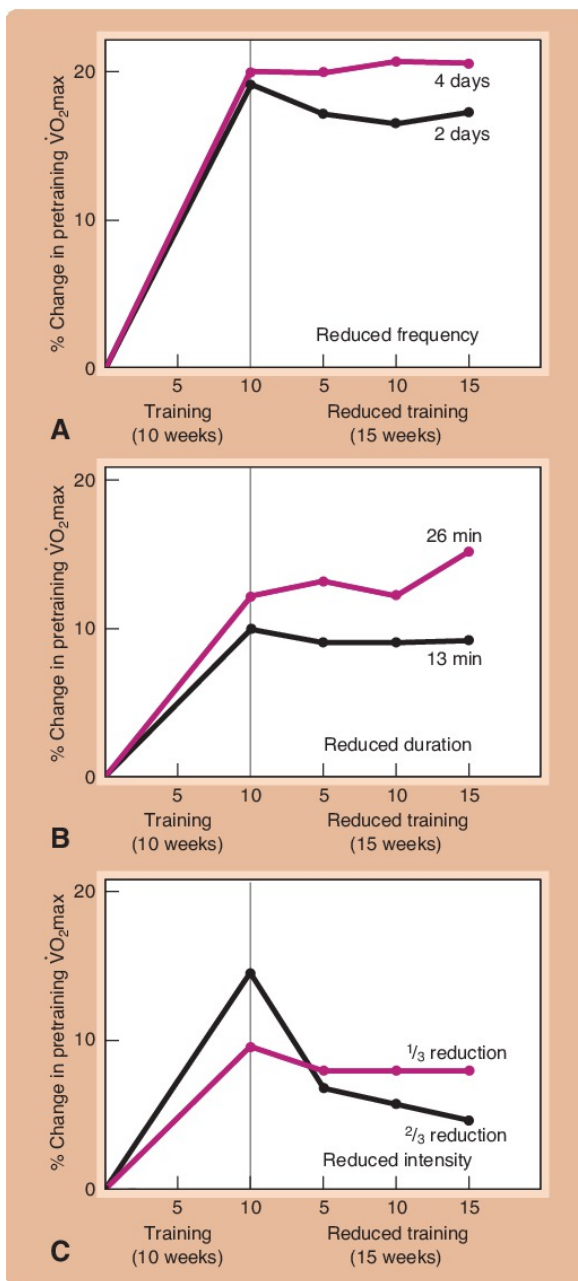


Figure 13.4 Effects of Reducing Exercise Frequency, Intensity, and Duration on Maintenance of O₂max.

A. Improvements in O₂max during 10 weeks of training

(bicycling and running) for 40 minutes a day, 6 days a week were maintained when training intensity and duration were maintained with a reduction in frequency from 6 days a week to 4 or even 2 d-wk⁻¹. **B.** O₂max was maintained when frequency of training and intensity were maintained with a reduction of training duration to 13 minutes. O₂max continued to improve when training duration was reduced to 26 minutes. **C.** O₂max was maintained when frequency and duration were maintained and intensity was reduced by one third. O₂max was not maintained when training was reduced by two thirds. **Sources:** [Hickson and Rosenkoetter \(1981\)](#); [Hickson et al. \(1982, 1985\)](#).

During the maintenance phase of a training program, cross-training is particularly beneficial, especially on days when a high-intensity workout is not called for. For example, one study divided endurance-trained runners into three groups. One third continued to train by running, one third trained on a cycle ergometer, and one third trained by deep water running. The intensity, frequency, and duration of workouts in each modality were equal. After 6 weeks, performance in a 2-mi run had improved slightly (~1%) in all three groups ([Eyestone et al., 1993](#)). Thus, running performance was maintained by each of the modalities. On the other hand, arm ergometer training has not been shown to maintain training benefits derived from leg ergometer activity ([Pate et al., 1978](#)). Apparently, the closer the activities are in terms of muscle action, the greater the potential maintenance benefit of cross-training.

Retrogression/Plateau/Reversibility

Sometimes, an individual in training may exhibit a performance or physiological decrement (retrogression) or fail to improve (plateau), despite progression of the training program. When such a pattern occurs, it is important to check for other signs of overtraining (see [Chapter 22](#)). A shift in training emphasis or the inclusion of more easy days is warranted. Remember that a reduction in the frequency of training does not necessarily lead to

detraining and may actually enhance performance.

If training is discontinued for a significant period of time, detraining will occur. This principle, often referred to as the reversibility concept, holds that when a training program is stopped or reduced, body systems readjust in accordance with the decreased physiological stimuli. Increases in $\dot{V}O_2\text{max}$ with low-to-moderate exercise programs are completely reversed after training is stopped. Values of $\dot{V}O_2\text{max}$ decrease rapidly during a month of detraining, followed by a slower rate of decline during the 2nd and 3rd month (Bloomfield and Coyle, 1993).

Warm-Up and Cooldown

A warm-up period allows the body to adjust to the cardiovascular demands of exercise. At rest, the skeletal muscles receive about 15–20% of the blood pumped from the heart; during moderate exercise, they receive approximately 70%. This increased blood flow is important for warming the body since the blood carries heat from the metabolically active muscle to the rest of the body.

A warm-up period of 5–10 minutes at light to moderate intensity should precede the conditioning portion of an exercise session (ACSM, 2022). The warm-up should gradually increase in intensity until the desired intensity of training is reached. For many activities, the warm-up period simply continues into the aerobic portion of the exercise session. For example, if an individual is going for a noontime run and wants to run at an 8 min-mi–1 pace, he or she may begin with a slow jog for the first few minutes (say a 10 min-mi–1 pace), increase to a faster pace (say a 9 min-mi–1 pace), and then proceed into the desired pace (the 8 min-mi–1 pace).

A warm-up period has the following beneficial effects on cardiovascular function.

- It increases blood flow to the active skeletal muscles.
- It increases blood flow to the myocardium.
- It increases the dissociation of oxyhemoglobin.
- It causes sweating, which plays a role in temperature regulation.
- It may reduce the incidence of abnormal rhythms in the

heart's conduction system (dysrhythmias), which can lead to abnormal heart function ([Barnard et al., 1973](#)).

A cooldown period of 5–10 minutes of light- to moderate-intensity activity should follow the conditioning period of the exercise session (ACSM, 2022). The cooldown period prevents venous pooling by keeping the muscle pump active and thus may reduce the risk of postexercise hypotension (and possible fainting) and dysrhythmias. A cooldown also facilitates heat dissipation and promotes a more rapid removal of lactate and catecholamines from the blood.

Training Principles and Physical Activity Recommendations

Much evidence has been compiled that demonstrates the health-related benefits of moderate physical activity, including reduced incidence of cardiac events, stroke, hypertension, type 2 diabetes, some types of cancer, obesity, the metabolic syndrome, depression, and anxiety. The Surgeon General's Report (SGR) on Physical Activity and Health recommendation ([U.S. Department of Health and Human Services \[USDHH\], 1996](#)) provided an important public health statement that recognized the health benefits associated with moderate levels of physical activity and encouraged increased activity among Americans by widely publicizing those health benefits and recommending levels of activity (an accumulation of 30 minutes of moderate-intensity physical activity on most, if not all, days of the week) that were intended to be nonintimidating for currently sedentary individuals. Since the initial publication, the dose-response relationships between physical activity and health benefit are emphasized. That is, while some activity of moderate intensity is better than no activity, more activity and more vigorous activity is better than less activity, within reasonable limits. The [U.S. Department of Health and Human Services guidelines \(2018\)](#) and Canadian guidelines ([Canadian Society for Exercise Physiology \[CSEP\], 2019](#)) are similar.

Table 13.1 also contains two sets of recommendations for

physical activity for children and adolescents (CSEP, 2019; USDHHS, 2018-endorsed by SHAPE America). Evidence has indicated that 30 min·d⁻¹ is not sufficient exercise for school-age individuals. This is reflected in the recommendations of 60+ min·d⁻¹ of moderate to vigorous physical activity daily for this age group.

FOCUS ON APPLICATION

Manipulation of Training Overload in a Taper

Peaking for performance often involves manipulating the training principles of specificity, overload, and maintenance within a periodization plan. This is exemplified by a study in which 18 male and 6 female distance runners were pretested, matched, and then divided into three groups. The run taper group systematically reduced its weekly training volume to 15% of their previous training volume over a 7-day period, performing 30% of the calculated reduced training distance on day 1, and then 20, 15, 12, 10, 8, and 5% on each succeeding day. Training consisted of 400-m intervals at close

to 5-km pace (~100% $\dot{V}O_{2peak}$) resulting in an HR of 170–190 b·min⁻¹ with recovery to 100–110 b·min⁻¹ before the next interval. The cycle taper group performed approximately the same number of intervals for the same duration as paired athletes in the run taper group, at the same work and recovery heart rates. The control group continued normal training, of which 6–10% of the weekly training distance was interval/fartlek work. All subjects participated in a 10-minute submaximal treadmill run, an incremental treadmill test to volitional fatigue in which the grade remained constant at 0% and the speed increased, and a 5-km time trial on the treadmill.

At the same absolute speed during the submaximal run, the run taper group (and seven of the eight individual

runners) exhibited a 5% reduction ($2.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) in oxygen consumption and a decrease of 7% ($0.9 \text{ kcal}\cdot\text{min}^{-1}$) in calculated energy expenditure. No changes were evident in either the cycle taper or control group. Both maximal treadmill speed (2%) and total exercise time (4%) increased for the run taper group without a concomitant increase in

$\text{VO}_2 \text{ max}$ or HRmax. No changes occurred in any maximal value for the cycle taper or control groups. The run taper group (all eight individuals) significantly improved 5-km performance by a mean of $2.8 \pm 0.4\%$, or an average of almost 30 seconds. No improvement in performance was seen in either the cycle taper or control groups.

These results clearly demonstrate the benefits of a 7-day taper in which intensity is maintained, training volume drastically reduced, and specificity of training utilized. Of the variables measured, the most likely explanation for the improved 5-km performance was the increase in submaximal running economy (decreased submaximal oxygen and energy cost). Note, however, that all three groups maintained their

$\text{VO}_2 \text{ max}$ values. This cross-training benefit exhibited by the cycle taper group is particularly important. Distance runners often have nagging injuries. These results imply that a non-weight-bearing taper may be used in such cases and allow the runner to possibly heal (or at least not aggravate an injury) while maintaining cardiovascular fitness. Performance enhancement, however, appears to require mode specificity during the taper.

Source: [Houmard et al. \(1994\)](#).

As with adults, children/adolescents can accumulate the recommended duration of activity throughout the day rather than in a single more structured training session. Children by nature tend to be sporadic exercisers, and getting them to exercise continuously is both unrealistic and unnecessary ([Corbin et al., 2004](#)). However, the SHAPE America guidelines do recommend that children should participate in several bouts of physical

activity each day each lasting 15 minutes or more. Note that the 60-minute recommendation for those between 5 and 18 years of age is considered a minimum. Also, CSEP and SHAPE America recommend that extended periods of inactivity (2 or more hours at one time) be discouraged for children during waking hours. This includes screen time, motorized transport, extended sitting, and nonactive indoor time.

With few exceptions, children and adolescents ideally should be involved in a wide variety of age-appropriate activities daily. CSEP actually recommends 30 min of “tummy time” for infants less than 1 year and 180 minutes of a variety of physical activities for toddlers (1–2 years) and preschoolers (3–4 years). As with adults, large muscle activities involving rhythmical dynamic muscle contractions are best for the development of cardiovascular fitness, but children and adolescents should try as many different activities as possible to develop their skills and learn which they enjoy most. Enjoyable activities are more likely to be continued throughout life.

All guidelines presented in **Table 13.1** are intended for “apparently healthy” individuals of the appropriate age irrespective of gender, race, ethnicity, or socioeconomic status. Additionally, with appropriate evaluation, medical advisement, and initial modification to gradually progress to achieving the recommendations, they may be used for individuals with certain chronic diseases or disabilities.

Physical activity recommendations are the manifestation of the cardiorespiratory training principles for public health. In addition to documented health benefits, moderate-intensity physical activity was and is promoted as a way to increase the palatability of exercise so that more people would participate. The Centers for Disease Control and Prevention (CDC) periodically polls the U.S. population on a variety of issues. Unfortunately, the latest physical activity poll indicates that there is still a large segment of the adult U.S. population who report no leisure-time physical activity (**Figure 13.5**) (CDC, 2019).

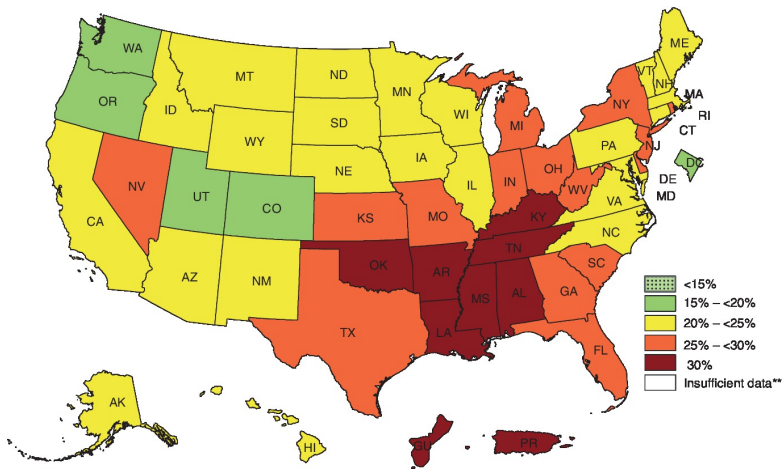


Figure 13.5 Estimates of No Leisure-time Physical Activity.

2019 estimates of the percentage of adults who are physically inactive. **Source:** Centers for Disease Control and Prevention, 2019.

The CDC reports that the proportion of the population reporting no physical activity has decreased from about 32% in 2010 to about 25% in 2018. While this improvement is obviously a good thing, it does not mean that the remaining 75% are meeting the recommended levels. Data from 2018 indicated that 54% of adults participated in at least minimal recommended levels of physical activity. For high school students, only 26% achieved the recommended levels of physical activity (CDC, 2021). There is much room for improvement. Unfortunately these low activity participation levels are reflected in cardiorespiratory fitness results. A systematic review of over 2.5 million adults in eight high- and middle-income countries between 1967 and 2016 (Lamoureux et al., 2019) showed a moderate decline of 7.7% (or 1.6% per decade) in cardiorespiratory fitness (VO_2max). Declines occurred in all countries, more in males than females and in young adults compared with middle-aged adults. Countries with the largest increases in adult obesity showed the largest decline in cardiorespiratory fitness. A parallel study (Tonkinson et al., 2019) of approximately 1 million children and adolescents

from 19 high and upper middle-income countries between 1981 and 2014 presented somewhat more hopeful results. Collectively there was a 7.3% decline in cardiorespiratory fitness. However, the decline diminished with each decade and stabilized near zero around the year 2000. Again the decline was larger for boys than girls but was similar for children and adolescents. In Belgium, Brazil, Canada, and Japan, improvements were actually seen.

Cardiovascular Adaptations to Aerobic Endurance Training

As has been discussed, regular physical activity results in improvements in cardiovascular health and function. Although the primary goal and most obvious adaptation is an increase in $\dot{V}O_{2\max}$, this adaptation is supported and accompanied by changes in numerous other physiological variables. The magnitude of the improvement depends on the training program—specifically on the frequency, intensity, and duration of the exercise and the individual's initial level of fitness. **Figure 13.6** provides a schematic of the cardiovascular system and notes locations/sites where important adaptations occur. The numbering on the schematic coincides with the paragraphs below that detail cardiovascular adaptations. Note that adaptations occur throughout the cardiovascular system, including cardiac changes, vascular changes, and blood/coagulatory changes. **Figure 13.7** presents cardiovascular responses to incremental exercise to maximum following aerobic exercise training. Changes in cardiovascular variables may be evident at rest, during submaximal exercise, and during maximal exercise. Many of these changes have health implications.

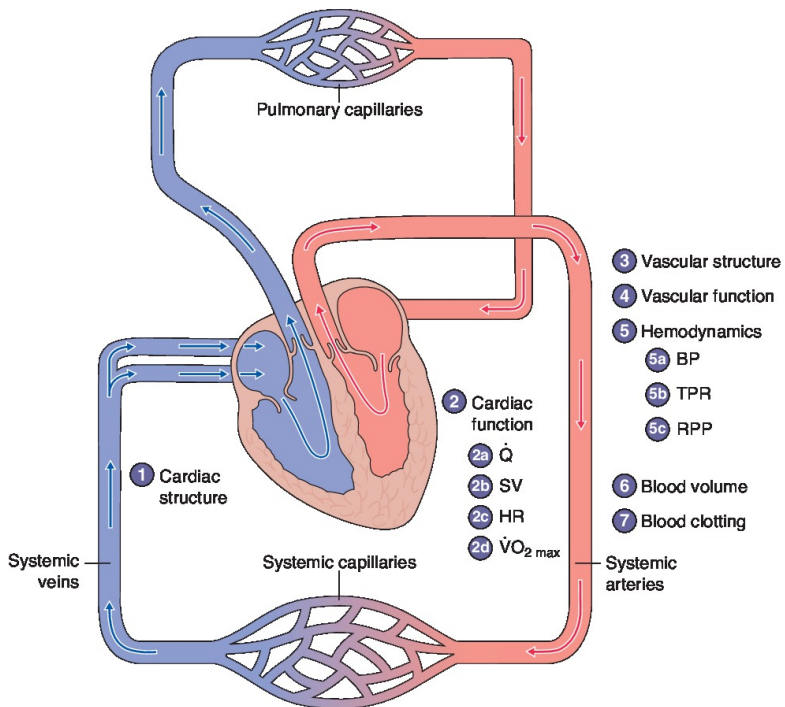


Figure 13.6 Cardiovascular Adaptations.

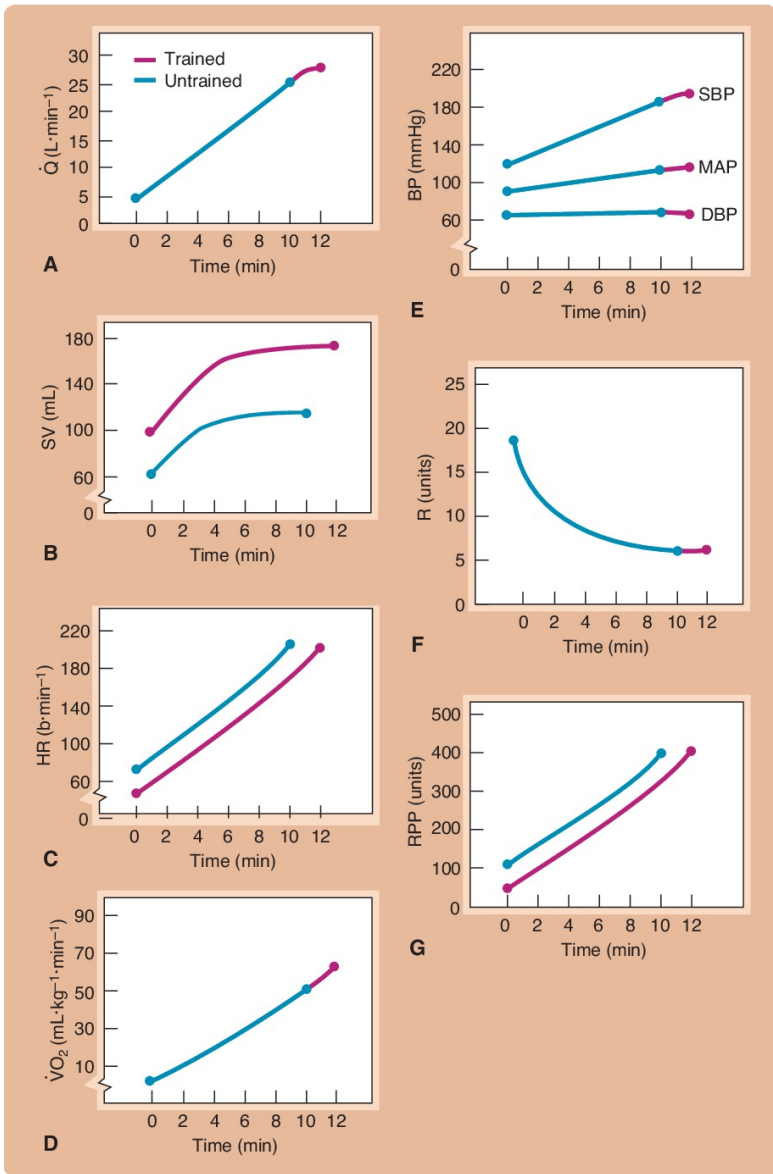


Figure 13.7 Comparison of Cardiovascular Response of Trained and Untrained Individuals to Incremental



Exercise to Maximum

A. Cardiac output (\dot{Q}). B. Stroke volume (SV). C. Heart rate

(HR). D. Oxygen consumption (O_2). E. Blood pressure (BP).
F. Resistance (R). G. Rate-pressure product (RPP).

Cardiac Structure (1)

In 1975, Morganroth conducted a groundbreaking study that used cardiac ultrasound to measure left ventricular changes in endurance athletes and sedentary controls. The findings of that study led to the concept of the “athlete’s heart,” which posits that the heart adapts to the specific demands of a training program. Exercise training programs bring about significant changes in myocardial structure and function known as exercise-induced cardiac remodeling (Weiner and Baggish, 2012). Endurance training leads to increases in left ventricular wall thickness, end-diastolic volume, and mass (Huston et al., 1985; Keul et al., 1981; Longhurst et al., 1981; Morganroth et al., 1975; Spence et al., 2011). These changes are associated with high cardiac output during sustained aerobic exercise. Endurance training exposes the heart to conditions of increased ventricular filling, with subsequent high stroke volume and cardiac output. This chronic exposure to high levels of ventricular filling (large end-diastolic volume) is known as volume overload (Morganroth et al., 1975). Chronic volume overload results in an increased left ventricular end-diastolic diameter (Huston et al., 1985; Keul et al., 1981) and left ventricular mass (Cohen and Segal, 1985; Longhurst et al., 1981). To better characterize the effect of aerobic training on both left and right ventricular mass and volume, Scharhag et al. (2002) used magnetic resonance imaging (MRI) to measure heart size and volume in a group of endurance-trained male athletes and a group of age- and size-matched controls. As shown in **Figure 13.8**, the aerobically trained athletes had greater right and left ventricular mass (**Figure 13.8A**) and greater right and left end-diastolic volume (**Figure 13.8B**). Another study that used MRI to investigate cardiac adaptations following a 6-month endurance training program found a significant increase in left ventricular mass but no significant increase in left ventricular end-diastolic volume (Spence et al., 2011).

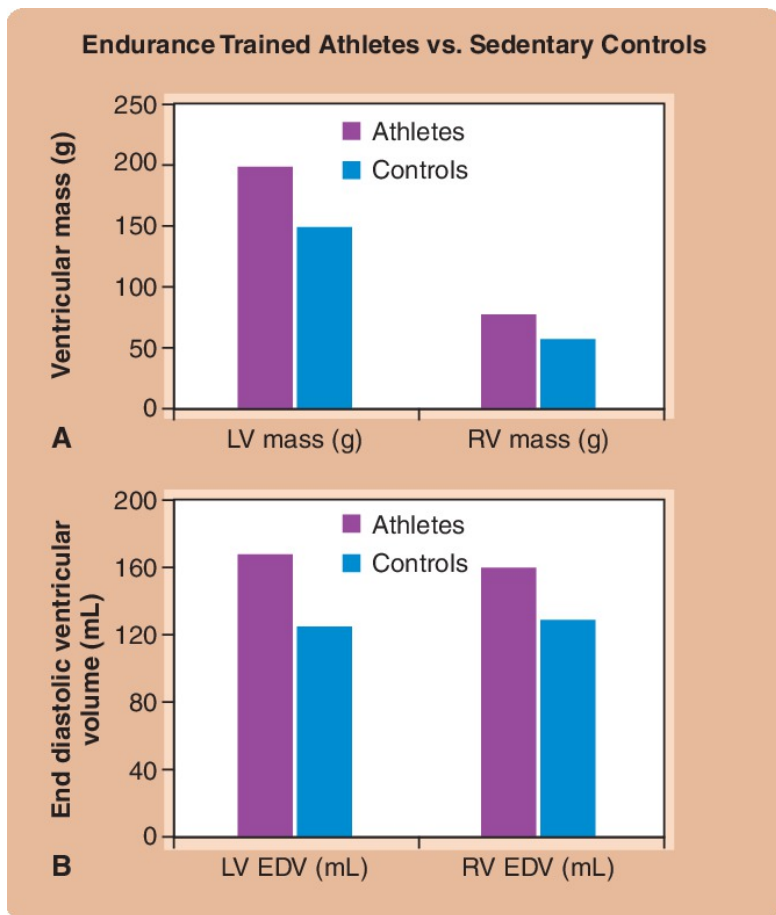


Figure 13.8 Comparison of Ventricular Mass (A) and End-Diastolic Ventricular Volumes (B) in a Group of Endurance-Trained Athletes and Sedentary Controls.

Source: Based on Data in [Scharhag et al. \(2002\)](#).

Cardiac Function (2)

There are several measures of cardiac function that changes as a result of aerobic exercise training.

Cardiac Output (2a)

Cardiac output, the amount of blood pumped by the heart each

minute, is also the amount of blood that flows through the vascular system each minute. As seen in **Figure 13.7A**, cardiac output is unchanged at rest and during submaximal exercise following an aerobic exercise training program. However, following a training program, more work can be done, meaning that the exercise test to maximum can continue longer, and a higher maximal cardiac output can be achieved.

Although resting cardiac output does not change following a training program, it is achieved by a larger stroke volume and a lower heart rate than in the untrained (Saltin, 1969). Cardiac output at an absolute submaximal workload is decreased or unchanged with training, but, as at rest, the relative contribution of stroke volume and heart rate is changed (Åstrand and Rodahl, 1986; Mitchell and Raven, 1994). Maximal cardiac output increases at maximal levels of exercise following an endurance exercise training program (**Figure 13.7A**). This increase results from an increase in stroke volume, since maximal heart rate does not change to a degree that has any physiological meaning with training. The magnitude of the increase in cardiac output depends on the level of training. Elite endurance athletes may have cardiac output values in excess of 35 L·min⁻¹.

Stroke Volume (2b)

Changes in stroke volume represent one of the most fundamental adaptations to endurance training. As shown in **Figure 13.7B**, endurance training results in an increased stroke volume at rest, during submaximal exercise, and during maximal exercise. This increase results from increased plasma volume, increased cardiac dimensions, increased venous return, and increased ventricular distensibility and compliance, which reflects improved diastolic function (Howden et al., 2018; Mitchell and Raven, 1994; Smith and Mitchell, 1993). Since several of these are structural changes, they exert their influence both at rest and during exercise.

It has traditionally been reported that the pattern of stroke volume response during incremental work to maximum is best described as an initial rectilinear rise that plateaus at about 40–50% of $\dot{V}O_2\text{max}$. This is seen in **Figure 13.7B**. However, as shown in **Figure 13.9**, some evidence suggests that stroke volume

does not plateau in highly trained endurance athletes (Gledhill et al., 1994; Wiebe et al., 1999), although most studies suggest that it does in untrained individuals (**Figures 13.7B and 13.9**). The question of whether endurance-trained athletes have a qualitatively different stroke volume response to incremental exercise remains unanswered (Rowland, 2009a).

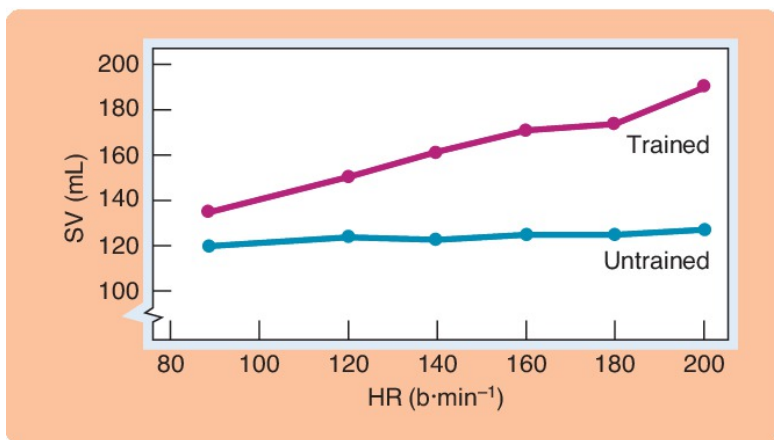


Figure 13.9 Stroke Volume Response in Trained and

Untrained Subjects.



Source: Modified with permission from Gledhill, N., D. Cox, & R. Jamnik: Endurance athletes' stroke volume does not plateau: Major advantage is diastolic function. *Medicine and Science in Sports and Exercise*. 26(9):1116–1121 (1994).

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Heart Rate (2c)

Resting heart rate is lower following endurance training (**Figure 13.7C**). Although bradycardia is technically defined as a resting heart rate less than 60 b·min⁻¹, the term is sometimes used to refer to the lower resting heart rate resulting from exercise training. Bradycardia is one of the classic and most easily assessed indicators of training adaptation. A reduced heart rate reflects a more efficient heart as the same amount of blood can be

pumped each minute (cardiac output) with fewer beats. Fewer heart beats are needed to achieve the same cardiac output because stroke volume is increased following training. The heart rate response to an absolute submaximal amount of work is significantly reduced following endurance training. Maximal heart rate is unchanged or slightly decreased ($2\text{--}3\text{ b}\cdot\text{min}^{-1}$) with endurance training (Ekblom et al., 1968; Saltin, 1969).

Maximal Oxygen Consumption (2d)

Maximal oxygen consumption ($\dot{V}O_{2\text{max}}$) is an integrated measure of cardiovascular function, and increases in $\dot{V}O_{2\text{max}}$ are one of the primary measures of cardiovascular health.

Increases in maximal oxygen consumption ($\dot{V}O_{2\text{max}}$) are one of the most recognized changes with endurance training (Figure 13.7D). The magnitude of the increase depends on the type of training program. Improvements of 5–30% are commonly reported, with improvements of 15% routinely found for training programs that meet the recommendations of the ACSM (2011).

$\dot{V}O_{2\text{max}}$ rapidly improves during the first 2 months of an endurance training program. Then improvements continue to occur, but at a slower rate. This pattern appears to be independent of sex and is consistent over a wide age range, although older individuals may take longer to adapt to endurance training (ACSM, 1998; Cunningham and Hill, 1975; Seals et al., 1984).

The improvement in $\dot{V}O_{2\text{max}}$ results from central and peripheral cardiovascular adaptations. Recall that $\dot{V}O_{2\text{max}}$ can be calculated as the product of cardiac output and arteriovenous oxygen difference ($a\text{-}vO_{2\text{diff}}$) (Equation 11.13). As previously discussed, maximal cardiac output increases as a result of endurance training, representing a central adaptation that supports the training-induced improvement in $\dot{V}O_{2\text{max}}$. The $a\text{-}vO_{2\text{diff}}$ reflects oxygen extraction by the working tissue and thus represents a peripheral adaptation that supports the improvement in $\dot{V}O_{2\text{max}}$ (see Chapter 10). Changes in cardiac output are a more consistent training adaptation than

changes in $a-vO_2\text{diff}$, and stroke volume appears to be the principal factor responsible for the increase in cardiac output.

Figure 13.10 uses compiled data to compare $\dot{V}O_{2\text{max}}$ of various athletic groups (Wilmore and Costill, 1988). Several conclusions can be drawn from this graph. First, even among athletes, a male-female difference occurs, with males generally having a greater $\dot{V}O_{2\text{max}}$ than females. Second, $\dot{V}O_{2\text{max}}$ varies considerably among athletes. Third, $\dot{V}O_{2\text{max}}$ is related to the demands of the sport. Athletes whose performance depends on the ability of the cardiovascular system to sustain dynamic exercise consistently have higher $\dot{V}O_{2\text{max}}$ values than athletes whose sport performance is based primarily on motor skills, such as baseball.

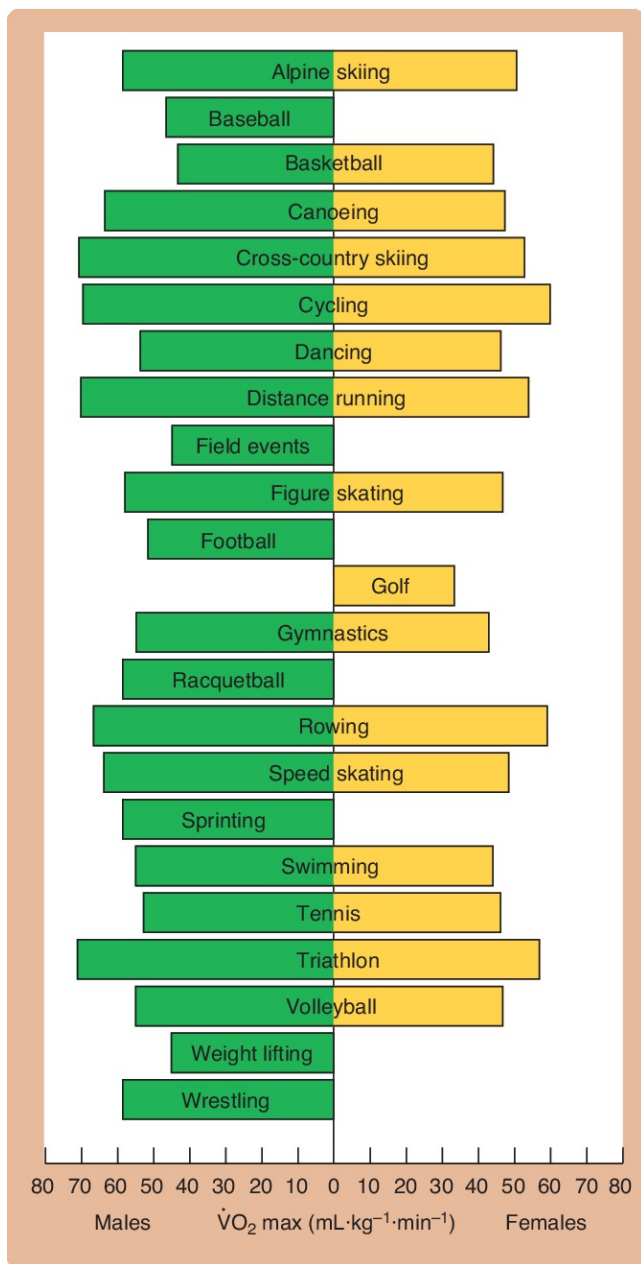


Figure 13.10 Average $\dot{V}O_2 \text{ max}$ of Male and Female

Athletes in Selected Sports.



Source: Based on data from [Wilmore and Costill \(1988\)](#).

Vascular Structure (3)

As described in [Chapter 11](#), blood vessel walls contain a layer of smooth muscle, the tunica media. Blood flow to a given region is determined by the pressure gradient and the resistance ($F = \Delta P / R$). By far, the greatest influence on resistance is the diameter of the vessel. Vessel diameter is determined by the actual size of the vessel and the relative degree of contraction of the smooth muscle in the tunica media. The greater the size of the vessel or the greater its ability to dilate, the greater the ability of the vasculature to provide increased blood flow to meet the needs of active tissue. Evidence shows that aerobic training can increase both the size of vessels and their ability to dilate.

Endurance training can lead to structural changes in the vasculature, including increased lumen size and decreased wall thickness, through the process of *arterial remodeling*. Strong evidence suggests that endurance athletes have enlarged arteries, both pericardial and those supplying skeletal muscle, thus demonstrating that aerobic exercise leads to structural changes in arteries that increase resting lumen diameter ([Dinunno et al., 2001](#); [Green et al., 2012](#); [Prior et al., 2003](#); [Schmidt-Trucksass et al., 2000](#)). [Naylor et al. \(2006\)](#) reported that the resting brachial artery diameter of elite rowers was significantly greater than that of untrained volunteers. Spence and colleagues reported that 6 months of endurance training (jogging/running) resulted in increased femoral artery diameter, but not brachial artery diameter indicating that exercise modality and muscles used impacts vascular structure ([Spence et al., 2013](#)). Certainly, an increased arterial diameter to working muscle represents a positive adaptation to exercise, but evidence also suggests that the coronary arteries, supplying blood to the working myocardium, are enlarged in highly trained athletes. Several studies (including the classic autopsy report of Clarence DeMar, winner of seven Boston marathons) have shown that habitual exercise is related to a larger cross-sectional arterial size. DeMar's arteries were reportedly two to three times the normal size ([Currens and White, 1961](#)). Research studies consistently find

that endurance athletes have larger arteries and many studies have reported a decreased wall thickness in trained athletes. These findings have led to the theory that there may be an “athlete’s artery” that is parallel to the “athlete’s heart” (Green et al., 2012).

Vascular Function (4)

Exercise training leads to an improved ability of arterial vessels to vasodilate; the increased vasodilatory potential is directly related to endothelium nitric oxide production. Thus, aerobic training improves endothelial function allowing the vessels to dilate and provide increased blood flow to working muscles. Improvements in endothelial function following aerobic exercise programs have been reported in healthy individuals with low risk for cardiac disease and in individuals with several risk factors as well as those with known cardiovascular disease (Green et al., 2003; Hambrecht et al., 1998; Niebauer and Cooke, 1996). Increasing evidence from animal studies shows that aerobic exercise leads to increased vasodilatory potential at several sites along the vascular tree, including the aorta, coronary arteries, and the brachial and femoral arteries (Jasperse and Laughlin, 2006).

Coronary vessels apparently have an increased vasodilatory response to exercise following exercise training. In a study that compared ultramarathoners to sedentary individuals, investigators found no difference in the internal diameter of the coronary arteries in the two groups at rest, but the capacity of the coronary arteries to dilate was two times greater in the marathoners than in the sedentary individuals (13.2 mm² vs. 6 mm²) (Haskell et al., 1993). The ability of arteries to dilate during exercise may be even more important than the resting diameter, because the myocardial demand for oxygen is low during rest and high during exercise, as evidenced by the low rate-pressure product (RPP) at rest and the high RPP during exercise.

The endothelium plays a critical role in determining vascular responses to acute exercise (as described in Chapter 12) and endurance exercise training improves endothelial function, specifically vasodilatory potential. Improved vascular function

has been reported in young men and women, overweight children, elderly men, and individuals with cardiovascular disease or disease risk factors (Clarkson et al., 1999; Hambrecht et al., 1998; Pedralli et al., 2020; Seals et al., 2019; Zhang et al., 2017).

Hemodynamics (5)

Aerobic training affects multiple hemodynamic variables that are associated with blood flow through the vascular system.

Blood Pressure (5a)

As indicated in **Figure 13.7E** and as most studies report, there is little or no change in arterial blood pressure (SBP, DBP, MAP) at rest, during submaximal exercise, or during maximal exercise in normotensive individuals after an endurance training program (Seals et al., 1984). However, because the maximal amount of work that can be done increases with exercise training, a trained individual is capable of doing more work. Thus, maximal systolic blood pressure may be higher for trained individuals at maximal exercise. This difference is usually small between sedentary and normally fit individuals.

Total Peripheral Resistance (5b)

Resistance is unchanged at rest or during an absolute submaximal workload following a training program (**Figure 13.7F**). However, total peripheral resistance is lower at maximal exercise following training, in part because the vessels supplying the working muscles are able to dilate to a greater extent. For this reason, trained individuals can generate significantly higher cardiac outputs at similar arterial pressures during maximal exercise. Much of the additional decrease in the total peripheral resistance at maximal exercise in trained individuals results from the increased capillarization of the skeletal muscle in these individuals (Blomqvist and Saltin, 1983).

Rate-Pressure Product (5c)

Rate-pressure product reflects the work of the heart. It is the product of HR and SBP, and thus adaptations in RPP could logically be described under both cardiac and vascular adaptations.

Myocardial oxygen consumption, indicated by the rate-pressure product, is lower at rest and during submaximal exercise following endurance training (**Figure 13.7G**). This result reflects the greater efficiency of the heart, since fewer contractions are necessary to eject the same amount of blood during submaximal exercise ([Mitchell and Raven, 1994](#)). Because maximal heart rate is unchanged and systolic blood pressure is either unchanged or increases slightly with exercise training, it follows that the maximal rate-pressure product is unchanged or increases slightly.

Blood Volume (6)

Blood volume increases as a result of endurance training. Highly trained endurance athletes have a 20–25% larger blood volume than untrained subjects. The increase in blood volume is primarily due to an expansion of plasma volume. This increase has been reported for both males and females and appears to be independent of age ([Convertino, 1991](#)). Increases in plasma volume occur soon after beginning an endurance training program, with changes between 8% and 10% occurring within the first week of training ([Convertino et al., 1980](#)) followed by a plateauing of plasma volume. For up to 10 days of training, an expansion of plasma volume accounts for increases in blood volume, with little or no change in red blood cell mass ([Convertino, 1991](#); [Convertino et al., 1980](#)).

Hematocrit and hemoglobin concentrations during this period are often lower, because the red blood cells and hemoglobin are diluted by the larger plasma volume. This condition has sometimes been called *sports anemia*, but this term is a misnomer because the number of red blood cells is almost the same or may actually be increased above pretraining levels. Thus, there is no reason for alarm about this condition; in fact, it may actually be beneficial. The lower hematocrit as a result of elevated plasma volume and normal or slightly elevated number of red blood cells means that the blood is less viscous, which decreases resistance to

flow and facilitates the transportation of oxygen.

After approximately 1 month of training, the increase in blood volume is distributed more equally between increases in plasma volume and red blood cell mass ([Convertino, 1991](#); [Convertino et al., 1991](#)). Blood volume and plasma volume return to pretraining levels when exercise is discontinued. **Figure 13.11** depicts these changes in blood volume, plasma volume, and red blood cell volume during 8 days of exercise training and after 7 days of cessation of exercise.

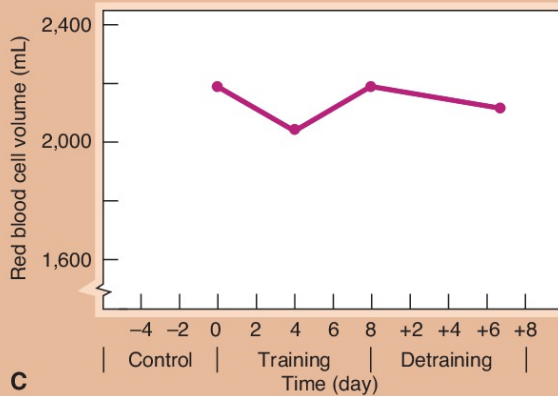
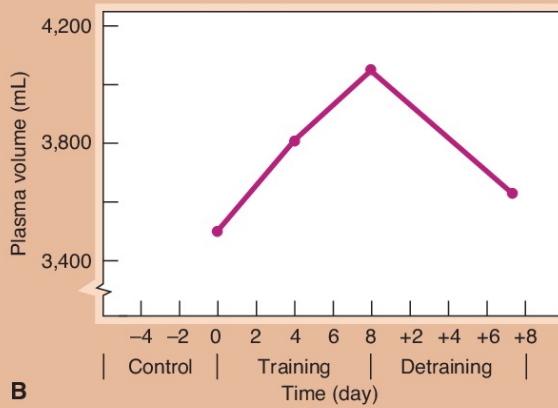
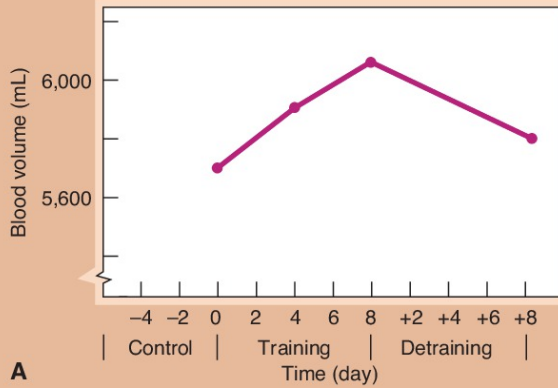


Figure 13.11 Changes in Blood Volume as a Result of Training and Detraining.

A. Blood volume. **B.** Plasma volume. **C.** Red blood cell

volume. **Source:** Reprinted with permission from Convertino, V. A., P. J. Brock, L. C. Keil, E. M. Bernauer, & J. E. Greenleaf: Exercise training–induced hypervolemia: Role of plasma albumin, renin, and vasopressin. *Journal of Applied Physiology*. 48(4):665–669 (1980). Copyright © 1980 The American Physiological Society. All rights reserved.

Clot Formation and Breakdown (7)

As discussed in [Chapter 11](#), a blood clot forms when needed to prevent blood loss from a damaged vessel. The body also breaks down clots (fibrinolysis) when they are no longer needed. Although blood clots are very useful when a vessel is damaged, unnecessary clots greatly increase the risk of heart attack and stroke.

Aerobic exercise training decreases the blood's tendency to clot and enhances the process of dissolving unnecessary clots (enhanced fibrinolytic activity), thus decreasing the risk for vascular clot formation. These are important mechanisms by which regular exercise decreases the risk of cardiovascular death. Moderate-intensity aerobic exercise alters coagulatory potential in part by depressing platelet aggregation (first step in clot formation) in healthy men and women ([Wang et al., 1995, 1997](#)). Since the endothelium releases factors that inhibit platelet aggregation, improved endothelial function with exercise may be related to the beneficial changes observed in platelets following a training program. In addition to suppressing platelet aggregation, evidence shows that moderate exercise training decreases clotting potential, thus decreasing the risk of coronary thrombus formation. Furthermore, exercise training is associated with an enhanced ability to break down clots ([Szymanski and Pate, 1994](#); [Womack et al., 2003](#)).

Table 13.7 summarizes the training adaptations that occur within the cardiovascular system as a result of a dynamic aerobic exercise program.



TABLE 13.7 Cardiovascular Adaptations to Dynamic Aerobic Exercise

| | Rest | Absolute Submaximal Exercise | Maximal Exercise |
|--------------|---------------------|------------------------------|------------------------------|
| \dot{Q} | Unchanged | Decreased or unchanged | Increased |
| SV | Increased | Increased | Increased |
| HR | Decreased | Decreased | Unchanged or slight decrease |
| SBP | Little or no change | Little or no change | Little increase or no change |
| DBP | Little or no change | Little or no change | Little decrease or no change |
| MAP | Little or no change | Little or no change | Little increase or no change |
| $\dot{V}O_2$ | — | — | Increased |
| TPR | Unchanged | Unchanged | Decreased |
| RPP | Decreased | Decreased | Unchanged or slight increase |

Cardiovascular Adaptations to HIIT

High-intensity interval training (HIIT) is becoming an attractive alternative to aerobic endurance training for individuals who want to improve fitness and health, in large part because it is a time-efficient. HIIT also mimics high bursts of energy required for many sports. Much less research has been conducted on the cardiovascular responses to HIIT than to endurance training, but most of the research that has been conducted indicates that HIIT brings about most of the same training adaptations as prolonged endurance training, and in many cases is more effective.

Cardiac Structure and Function

Even short periods of HIIT training can lead to changes in cardiac structure and function. [Saadatnia et al. \(2016\)](#) conducted a study in which young untrained men underwent 10 weeks of HIIT training. The training program resulted in an increase in left ventricular mass, end-diastolic ventricular volume, stroke volume, and ejection fraction.

Maximal Oxygen Consumption

Improvements in oxygen consumption with a training program reflect increased capacity of the cardiorespiratory system to take in, transport, and utilize oxygen. Aerobically trained individuals are identified by having high $\dot{V}O_{2\max}$ values. Several studies

have found the HIIT training is an effective way to improve $\dot{V}O_2\text{max}$. When studies compare moderate continuous training (MCT) to HIIT, and ensure that the same volume of work is done in both programs, greater improvements in $\dot{V}O_2\text{max}$ typically occur in HIIT programs (Sultana et al., 2019). When studies compare low-volume HIIT programs to MCT that have higher volume work, the HIIT programs still result in similar improvements, or greater improvements in $\dot{V}O_2\text{max}$ (Sultana et al., 2019). These findings are consistent with the important role of exercise intensity in determining improvements in $\dot{V}O_2\text{max}$ (ACSM, 2022).

Vascular Function

It appears that HIIT training leads to greater improvements in vascular function than MCT (Ramírez-Vélez et al., 2019; Ramos et al., 2015). Changes in hemodynamic variables during exercise leads to shear stress that stimulates the vascular endothelium. When comparing changes in maximal flow mediated dilation after a HIIT or MCT program, greater changes were reported after the HIIT program for most studies reviewed in a meta-analysis. While both types of training resulted in improvements in flow-mediated dilation, reflecting improved vascular function, on average, the HIIT programs resulted in 2.3% greater improvement in flow-mediated dilation than MCT (Ramos et al., 2015). The authors concluded that HIIT may be more effective at improving vascular function because of its ability to positively impact $\dot{V}O_2\text{max}$, cardiovascular disease risk factors, oxidative stress, inflammation, and insulin sensitivity.

Cardiovascular Adaptations to Dynamic Resistance Training

Low-volume dynamic resistance training (few repetitions and low weight) has not been shown to lead to any consistent or significant changes in cardiovascular variables. Thus, the changes

described in the following sections depend on high-volume (high total workload) dynamic resistance training programs (Stone et al., 1991).

Cardiac Structure

Dynamic resistance-trained athletes often have increased left ventricular wall and septal thicknesses, although this is not consistently seen in short-term training studies (Keul et al., 1981; Longhurst et al., 1981; Morganroth et al., 1975; Pluim et al., 2000; Spence et al., 2011). When the increase in wall thickness is reported relative to body surface area or lean body mass, the increase is greatly reduced or even nonexistent (Fleck, 1988a). The increase in wall thickness is thought to result from the work the heart must do to overcome the high arterial pressures (increased pressure afterload) encountered during resistance training (Morganroth et al., 1975); this depends on training intensity and volume.

Stroke Volume and Heart Rate

Resting stroke volume in highly trained dynamic resistance athletes has been reported to be both greater than normal and not different from normal (Effron, 1989; Fleck, 1988b). Because stroke volume is so seldom measured during resistance activities, changes that may occur in stroke volume from this type of training are not known (Sjogaard et al., 1988).

Highly trained dynamic resistance athletes have average or below-average resting heart rates (Stone et al., 1991). Heart rate at a specified submaximal dynamic resistance workload is lower following resistance training (Fleck and Dean, 1987).

Blood Pressure

Dynamic resistance-trained athletes do not have elevated resting blood pressures, provided that they are not chronically overtrained, do not have greatly increased muscle mass, and are not using anabolic steroids. This information contradicts the popular misconception that resistance-trained individuals have a

higher resting blood pressure than endurance-trained or untrained individuals. Indeed, most scientific investigations report that highly trained resistance athletes have average or lower-than-average systolic and diastolic blood pressures (Fleck, 1988b). Resistance-trained individuals also exhibit a lower blood pressure response to the same relative workload of resistance exercise than untrained individuals, even though the trained individuals are lifting a greater absolute load.

FOCUS ON RESEARCH

Benefits of Lifestyle versus Structured Exercise Training

Preprofessional students involved in athletics or high-intensity personal exercise training programs often find it difficult to accept that the level of activity originally recommended in the Surgeon General's Report (30 min d⁻¹, most days) can have any meaningful impact on measures of cardiorespiratory fitness or physiological variables. A study conducted at the Cooper Institute for Aerobics Research (and reported in these two articles) provides evidence for the effectiveness of this approach. Subjects were randomized into either a structured intervention program or lifestyle activity intervention program. Individuals in the structured group were given free memberships to the Cooper Fitness Center and trained with a designated exercise leader. Their program began with 30 minutes of walking 3 d·wk⁻¹, but after 3 weeks, they were allowed to select any available aerobic program and eventually progressed to 5 d·wk⁻¹. The lifestyle group received curricular material at weekly meetings centered around individual motivational readiness and behavioral motivation techniques. They were asked to accumulate no fewer than 30 minutes of at least moderate-intensity activity most days in any way that could be adapted to their individual lifestyle and to progress at their own rate. After 6 months, both groups were put on maintenance

programs, during which they were requested simply to continue their respective activities. Direct leadership and the number of group meetings were reduced. Selected cardiovascular results are presented in the accompanying table.

As anticipated, the greatest changes were made in the initial 6 months in both groups. Both interventions were effective in increasing physical activity, as indicated by the increases in energy expenditure and walking and the decreases in sitting. However, the structured group increased hard activity more than the lifestyle group and hence improved more than the lifestyle group in physical fitness. The improvement was measured by a greater decrease in HR during submaximal treadmill walking and a greater increase

in $\dot{V}O_2$ peak. In the ensuing 18 months, both groups decreased physical activity (energy expenditure) and physical

fitness ($\dot{V}O_2$ peak) from the 6-month level but maintained significant improvements over their initial values. Although the absolute magnitude of the changes is not great, it is important to realize that during the first 6 months, only 32 and 27% of the lifestyle and structure groups attained the level of activity suggested by the Surgeon General's Report. During the maintenance phase, these numbers were reduced to 20% in each group. Those in both groups who reported that they were active 70% or more of the weeks had at least twice as much improvement as those who did not.

The "take-home" messages from this study are that even under the conditions of well-designed and well-delivered external intervention, getting all individuals to include minimal but meaningful levels of activity into their lives is difficult. However, in previously sedentary healthy adult males and females, lifestyle intervention can be as effective as a structured exercise program in improving physical activity and cardiorespiratory fitness.

| | Lifestyle 6 mo | Lifestyle 24 mo | Structured 6 mo | Structured 24 mo |
|--|-------------------|--------------------|----------------------|---------------------|
| Activity energy expenditure (kcal·kg ⁻¹ ·d ⁻¹) | +1.53* | +0.84* | +1.34* | +0.69* |
| Achieved SG goal (2 kcal·kg ⁻¹ ·d ⁻¹) | 32% | 20% | 27% | 20% |
| Walking (min·d ⁻¹) | +19.80* | +13.07 | +16.52* | +26.75* |
| Sitting (hr·wk ⁻¹) | -5.27* | -1.18 | -6.88* | -6.85* [†] |
| Treadmill time (min) | +0.46* | +0.23* | +0.92* | +0.37* [†] |
| Submaximal HR (b·min ⁻¹) | -4.75* | -2.62* | -10.22* [†] | -4.88* |
| $\dot{V}O_{2peak}$ (mL·kg ⁻¹ ·min ⁻¹) | -1.58* | +0.77* | +3.64* [†] | +1.34* |
| SBP (mmHg) | | -3.63* | | -3.26* |
| DBP (mmHg) | | -5.38* | | -5.14* |
| Body fat (%) | | -2.39* | | -1.85* |

*Significant difference in each group compared to its own baseline.
[†]Significant difference between groups at 6 or 24 months.

Sources: Dunn, A. L., M. E. Garcia, B. H. Marcus, J. B. Kampert, H. D. Kohl, III, & S. N. Blair: Six-month physical activity and fitness changes in Project Active, a randomized trial. *Medicine & Science in Sports & Exercise*. 30(7):1076–1083 (1998); Dunn, A. L., B. H. Marcus, J. B. Kampert, M. E. Garcia, H. W. Kohl, III, & S. N. Blair: Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: A randomized trial. *Journal of the American Medical Association*. 281(4):327–334 (1999).

Dynamic resistance training has not been shown to consistently lower blood pressure in hypertensive individuals and, therefore, is not recommended as the only exercise modality for hypertensives except in the form of circuit training. Circuit training relies on high repetitions, low loads, and short rest periods in a series of stations. A supercircuit integrates aerobic endurance activities between the stations.

The rate-pressure product, which reflects myocardial oxygen consumption, is decreased at rest following strength training, during weight lifting or circuit training, and during aerobic exercise that includes a resistance component (such as holding hand weights while walking) (Fleck, 1988b; Stone et al., 1991). Researchers have suggested that these results occur because of a reduction in peripheral resistance.

Maximal Oxygen Consumption

Small increases (4–9%) in $\dot{V}O_{2\max}$ have been reported following circuit training and Olympic-style weight-lifting programs (Gettman, 1981; Stone et al., 1991). However, other studies have failed to identify any increase in $\dot{V}O_{2\max}$ with resistance training (Hurley et al., 1984). $\dot{V}O_{2\max}$ probably does not change much because of the low percentage of $\dot{V}O_{2\max}$ achieved during resistance training. Weight training may impact the central cardiovascular variables as described earlier (i.e., resulting in a reduced resting heart rate), but it does not enhance peripheral cardiovascular adaptations (i.e., a- $vO_{2\text{diff}}$). Thus, to improve cardiorespiratory fitness, individuals should not rely on resistance training programs but instead use dynamic resistance training in conjunction with aerobic endurance training.

FOCUS ON RESEARCH | *Clinically Relevant*

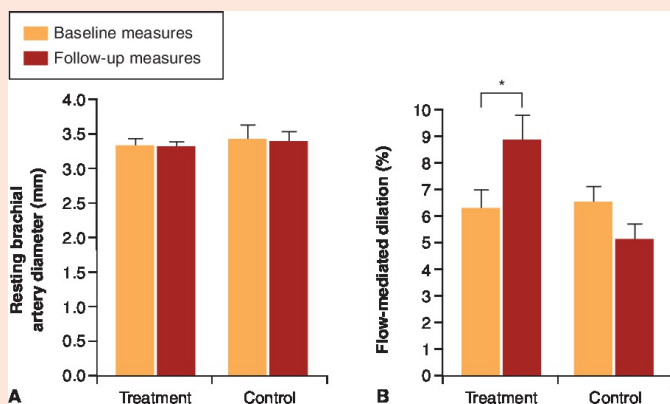
Resistance Training Improves Vascular Function in Overweight Women

Aerobic exercise is known to improve endothelial function. Aerobic exercise significantly elevates blood flow under moderately high pressure for a prolonged period of time. This increase in shear stress on the endothelium is thought to increase nitric oxide production leading to enhanced vasodilation. A recent study, however, hypothesized that resistance training, which elevates blood flow for shorter periods but under higher pressure, would also provide a stress stimulus on the endothelium resulting in improved vascular function.

The study included 30 overweight women, 15 of whom engaged in a 1-year resistance training program and 15 who served as controls. The researchers measured the resting diameter of the brachial artery before and after training.

They also measured the artery's ability to vasodilate after 3 minutes of occlusion, which is known to cause an increase in blood flow; this phenomenon is known as reactive hyperemia. The brachial diameter during the reactive hyperemia was reported as peak flow-mediated dilation and expressed as a percent.

This study found that resistance training positively affects vascular function in overweight women. This finding suggests that resistance training has important cardiovascular benefits and provides further support for the recommendation of including resistance training in an overall fitness program. However, given the small sample size and the narrow population studied, additional research into the effects of resistance training on vascular structure and function is warranted.



A. Resting baseline diameter of the brachial artery in the resistance-trained and control groups. **B.** Peak flow-mediated dilation of the brachial artery in the resistance-trained and control groups. Data are presented as mean \pm SEM. * $P < 0.05$ for within-group analysis.

Source: Adapted with permission from Olson, T. P., D. R. Dengel, A. S. Leon, & K. H. Schmitz: Moderate resistance training and vascular health in overweight women. *Medicine & Science in Sports & Exercise*. 38(9):1558–1564 (2006).

The Influence of Age and Sex on Cardiovascular Training Adaptations

Few data are available regarding the influence of age and sex on cardiovascular adaptations to dynamic resistance exercise. Therefore, this section addresses only cardiovascular adaptations to aerobic endurance exercise.

Male-Female Differences in Adaptations

Research evidence suggests no differences between the sexes in central or peripheral adaptations to aerobic endurance training. Both sexes exhibit similar cardiovascular adaptations at rest, during submaximal exercise, and at maximal exercise (Drinkwater, 1984; Mitchell et al., 1992). Maximal cardiac output is higher in both sexes because of the increased stroke volume following training; however, the absolute value achieved by a woman is less than that attained by a similarly trained man.

When males and females of similar fitness level train at the same frequency, intensity, and duration, they show no differences

in the relative increase in $\dot{V}O_2\text{max}$ (Lewis et al., 1986; Mitchell et al., 1992). As shown earlier in Figure 12.20,

$\dot{V}O_2\text{max}$ overlaps considerably between the sexes. Thus, a well-trained female may have a higher $\dot{V}O_2\text{max}$ than a sedentary or even normally active male; however, a female will always have a lower $\dot{V}O_2\text{max}$ than a similarly trained male.

The blood pressure (SBP, DBP, MAP) response to exercise is unchanged in both sexes following endurance training. Males and females show the same adaptations in total peripheral resistance and rate-pressure product. The effects of endurance training on cardiovascular variables at maximal exercise are reported in Table 13.8 for both sexes. In summary, the trainability of females does not differ from that of males, and similar benefits can and should be gained from regular activity by both sexes (Hanson and

Nedde, 1974). However, the absolute values achieved for maximal oxygen consumption, cardiac output, and stroke volume are generally lower in females because of their smaller body and heart size.

TABLE 13.8 Comparison of Cardiovascular Responses to Maximal Exercise in Sedentary and Trained Young Adults (20–30 years)

| Variable | Men | | Women | |
|--|-----------|---------|-----------|---------|
| | Sedentary | Trained | Sedentary | Trained |
| \dot{Q}_{\max} (L·min ⁻¹) | 22 | 30 | 16 | 20 |
| SV _{max} (mL·b ⁻¹) | 115 | 155 | 80 | 105 |
| HR _{max} (b·min ⁻¹) | 195 | 195 | 195 | 195 |
| $\dot{V}O_{2\max}$ (mL·kg ⁻¹ ·min ⁻¹) | 50 | 65 | 37 | 52 |
| SBP (mmHg) | 200 | 200 | 190 | 190 |
| DBP (mmHg) | 70 | 70 | 66 | 66 |
| MAP (mmHg) | 135 | 135 | 128 | 128 |
| TPR (units) | 6.1 | 4.5 | 8.0 | 6.4 |
| RPP (units) | 390 | 390 | 370 | 370 |

Adaptations in Children and Adolescents

Endurance training has been documented to result in increased left ventricular mass and heart volume in children, as it does in adults (Bar-Or, 1983; Greenen et al., 1982). The increase in heart size is associated with an increased resting stroke volume (Gutin et al., 1988) and a decreased resting heart rate but not with any change in cardiac output (Eriksson and Koch, 1973). Research also suggests an increased blood volume and hemoglobin level in young endurance athletes compared with sedentary children (Eriksson and Koch, 1973; Koch and Rocher, 1980; Zauner et al., 1989), but possibly not as much as in adults. Information about changes in capillary density with training in children is not available (Rowland, 2005).

At submaximal levels of exercise, cardiac output is unchanged or slightly decreased in youngsters after endurance training (Bar-Or, 1983; Soto et al., 1983) as a result of increased submaximal stroke volume and decreased heart rate (Bar-Or, 1983; Lussier

and Buskirk, 1977). Neither systolic nor diastolic blood pressure changes significantly as a result of endurance training during submaximal work (Lussier and Buskirk, 1977).

At maximal work, cardiac output increases in children and adolescents as a result of endurance training. This is caused by an increased maximal stroke volume and stable maximal heart rate (Eriksson and Koch, 1973; Lussier and Buskirk, 1977).

Children and adolescents can participate in a wide variety of training programs in school or community settings (Figure 13.12). Research has consistently shown improvements in endurance performance as a result of exercise training. Such improvements have occurred when endurance performance was measured as an increase in the workload performed (longer treadmill times or distances run, more distance covered in a set time, higher work output on a cycle ergometer, or longer rides at the same load) or as a faster time for a given distance (Cooper et al., 1975; Daniels and Oldridge, 1971; Daniels et al., 1978; Duncan et al., 1983; Dwyer et al., 1983; Goode et al., 1976; Graunke et al., 1990; Mosellin and Wasmund, 1973; Siegel and Manfredi, 1984). Given the lack of association between

endurance performance and $\dot{V}O_2\text{max}$ in children, it is not surprising that endurance performance improvements are not always accompanied by a comparable improvement in

$\dot{V}O_2\text{max}$ (Daniels and Oldridge, 1971; Daniels et al., 1978). Although children and adolescents who participate in organized

athletic activities have higher $\dot{V}O_2\text{max}$ values than those who do not, the relationship between measures of physical activity (such as self-report questionnaires, heart rate monitoring, and

motion detection devices) and measures of $\dot{V}O_2\text{max}$ is generally only low to moderate (Morrow and Freedson, 1994; Rowland, 2005; Vaccaro and Mahon, 1987). The most consistent finding of cardiovascular adaptations in prepubertal children is a diminished level of aerobic trainability compared to adults (Rowland, 2005). This occurs even in those studies in which the training meets the standards of intensity, duration, and frequency that result in substantial improvements in adults. Thus, where an adult (or postpubertal adolescent) might show a 25–30% increase, this is more likely to be 10–15% in prepubertal children.

It has been suggested ([Rowland, 2009b](#)) that this difference may be the result of metabolic differences between children and adults (as discussed in [Chapter 5](#)). Clarification requires further research.



Figure 13.12 Children Performing Soccer Drills.

Adaptations in Older Adults

Older men and women respond to endurance exercise training with adaptations similar to those in younger adults ([Hagberg et al., 1989](#); [Heath et al., 1981](#); [Ogawa et al., 1992](#)). Left ventricular wall thickness and myocardial mass are greater in elderly athletes than in elderly sedentary individuals, although these training adaptations may not be as pronounced or as quickly achieved as in younger adults ([Green and Crouse, 1993](#); [Heath et al., 1981](#); [Ogawa et al., 1992](#)).

Left ventricular end-diastolic volume and ejection fraction increase as a result of endurance training in older individuals. These changes enhance myocardial contractile function, especially the Frank-Starling mechanism, and help maintain cardiac output in the active elderly ([Green and Crouse, 1993](#)).

Resting cardiac output is unchanged as a result of endurance training in the elderly. Elderly athletes with an extensive history of endurance training consistently show lower resting heart rates than sedentary older adults. However, short-term training programs sometimes cause the expected decrease in resting heart rates and sometimes do not. Resting stroke volume typically increases, but the increase is generally small ([Green and Crouse, 1993](#)).

As in normotensive individuals of other ages, endurance training does not affect systolic blood pressure, diastolic blood pressure, or mean arterial blood pressure at rest in elderly people. Both hemoglobin levels and blood volume increase in the elderly as a result of endurance training, as does the density of capillaries supplying blood to the active musculature ([Green and Crouse, 1993](#)).

Most training studies show no change in cardiac output during any given submaximal workload. The components of cardiac output, however, often change reciprocally, with the expected decrease in heart rate and increase in stroke volume. Again, the stroke volume changes tend to be small and do not always reach statistical significance. Submaximal values for systolic blood pressure, mean arterial blood pressure, and total peripheral

resistance are lower in elderly athletes than in nonathletes and decrease with endurance training (Green and Crouse, 1993).

Maximal cardiac output may be increased by exercise training in older individuals. This increase is completely accounted for by the increased maximal stroke volume, since maximal heart rate is unchanged. The reported effects of endurance training on blood pressures and systematic vascular resistance are inconsistent, although most evidence suggests no change in these variables (Green and Crouse, 1993).

The results of training status on cardiovascular responses to maximal exercise in the elderly, including $\dot{V}O_{2\max}$, are shown in **Table 13.9** for both men and women. $\dot{V}O_{2\max}$ is higher in trained than in untrained elderly. Thus, training programs can result in increases in $\dot{V}O_{2\max}$ in older individuals. The magnitude of this increase depends on the individual's initial fitness level and the training program. Research suggests, however, that healthy, older untrained males and females can improve their $\dot{V}O_{2\max}$ by 15–30% with training (Hagberg et al., 1989; Ogawa et al., 1992; Seals et al., 1984).

TABLE 13.9 Comparison of Cardiovascular Responses to Maximal Exercise in Sedentary and Trained Elderly Individuals (60–70 years)

| Variable | Men | | Women | |
|--|-----------|---------|-----------|---------|
| | Sedentary | Trained | Sedentary | Trained |
| \dot{Q}_{\max} (L·min ⁻¹) | 16 | 19.4 | 12 | 15 |
| SV _{max} (mL·b ⁻¹) | 100 | 125 | 75 | 90 |
| HR _{max} (b·min ⁻¹) | 155 | 155 | 155 | 155 |
| $\dot{V}O_{2\max}$ (mL·kg ⁻¹ ·min ⁻¹) | 28 | 48 | 22 | 35 |
| SBP (mmHg) | 190 | 190 | 190 | 190 |
| DBP (mmHg) | 84 | 84 | 84 | 84 |
| MAP (mmHg) | 138 | 138 | 138 | 138 |
| TPR (units) | 8.6 | 7.3 | 11.5 | 9.2 |
| RPP (units) | 290 | 290 | 290 | 290 |

One study using a short-duration exercise program (9 weeks) of endurance training reported that a low-intensity exercise prescription (30–45% HRR) was as effective as a high-intensity exercise prescription (60–75% HRR) in eliciting improvements in $\dot{V}O_2\text{max}$ (Badenhop et al., 1983). However, a 1-year training program found that 6 months of training at low intensities (40% HRR) resulted in only a 10.5% improvement in $\dot{V}O_2\text{max}$ in elderly subjects. When the training program was progressively changed to a high-intensity program (85% HRR) and the duration extended, their $\dot{V}O_2\text{max}$ increased by another 16.5%. High-intensity interval training (HIIT) has also been shown to be effective in older individuals. A 6-week program that include HIIT for 5 days a week resulted in clinically relevant improvements in blood pressure and an increase in $\dot{V}O_2\text{max}$ in sedentary and athletic older adults (Grace et al., 2018). This research suggests that older individuals respond to exercise training in much the same way as younger individuals.

When starting a training program for older people, it is important to begin at low intensities to avoid injury. Significant improvements in function can be gained from low-intensity programs. After individuals become accustomed to the program, the training can be upgraded to a more intense level if desired. Note that the rate of adaptation may be slower in older individuals (ACSM, 2022).

Although older athletes are more similar to younger individuals than to their sedentary counterparts, and training programs tend to show the same beneficial changes in older as in younger subjects, exercise training does not stop the effects of aging on the cardiovascular system. At best, exercise training can only lessen normal age-related losses in cardiovascular function. This conclusion is exemplified in **Figure 13.13**, where the average rate of decline in $\dot{V}O_2\text{max}$ is shown for both an active, highly fit (HF) group of females and a relatively sedentary, low fitness (LF) comparison group (Plowman et al., 1979). The first thing to notice is that the HF group had higher $\dot{V}O_2\text{max}$ values than the LF group in every decade. Indeed, the $\dot{V}O_2\text{max}$ values of active 45-year-olds equaled those of

inactive 20-year-olds. Second, $\dot{V}O_2\text{max}$ expressed per kilogram of body weight declined with age, and the rate of decline was similar in the two groups. More recent data suggest that the rate of decline in peak $\dot{V}O_2\text{max}$ in healthy adults is not constant across the age span but accelerates markedly with each successive decade, regardless of physical activity habits (Fleg et al., 2005). It appears that declining forced expiratory volume (FEV1) and maximal exercise heart rates account for much of the “aging effect” on aerobic capacity (Hollenberg et al., 2006). The decline of peak aerobic capacity has substantial implications with regard to functional independence and quality of life for older adults.

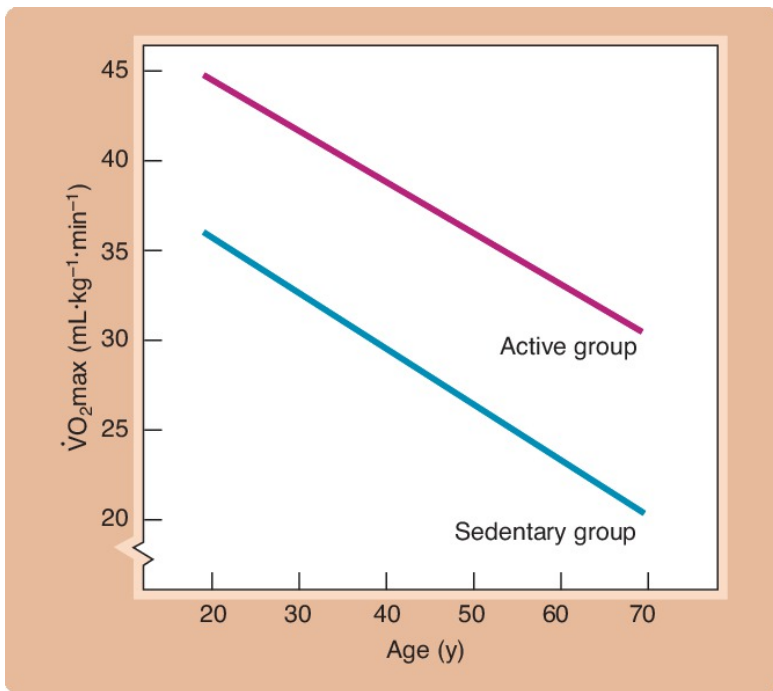


Figure 13.13 Age-Related Decline in $\dot{V}O_2\text{max}$ in Highly

Active and Sedentary Women.



Source: From Plowman, S. A., B. L. Drinkwater, & S. M. Horvath: Age and aerobic power in women: A longitudinal

Summary

1. A cardiovascular training program depends on the individual's age, health status, and the program's goals.
2. Any activity involving large muscle groups for a prolonged time has the potential to increase cardiovascular fitness. The choice of exercise modalities should be based on interest, availability, and a low risk of injury.
3. Training using different exercise modalities causes the same overall benefits with central cardiovascular adaptations, but peripheral cardiovascular adaptations are specific to the muscles being exercised.
4. Intensity is very important for improving maximal oxygen consumption ($\dot{V}O_{2\max}$) primarily in conjunction with duration, which determines training volume. Intensity can be prescribed in relation to heart rate, oxygen consumption, or rating of perceived exertion (RPE). Training intensity is the most important factor for maintaining cardiovascular fitness.
5. The ACSM recommends the following training goals to develop and maintain cardiorespiratory fitness in healthy adults: frequency of 3–5 d·wk⁻¹, intensity of 64–95% HRmax, 40–89% $\dot{V}O_{2R}$ or %HRR, and duration of 20–60 minutes of continuous aerobic activity.
6. The Canadian Society for Exercise Science recommends a total of 150 min·wk⁻¹ of moderate to vigorous physical activity more activity leads to more health benefits.
7. Children and adolescents should participate in at least 60 min·d⁻¹ of moderate to vigorous physical activity that is age appropriate while minimizing sedentary activity the rest of the day.
8. The absolute and relative increases in $\dot{V}O_{2\max}$ and the

health benefits thereof are inversely related to the individual's initial fitness level. The greatest improvements in fitness and health occur when very sedentary individuals begin a regular, low-to-moderate endurance exercise program. Meaningful health benefits can be achieved with minimal increases in activity or fitness by those who need it most.

9. Endurance training results in increased cardiac dimensions and mass.
10. Cardiac output at rest and at an absolute submaximal workload is not changed by an endurance training program. However, cardiac output at the same relative workload and at maximal exercise is greater with endurance training.
11. Stroke volume is greater at rest, at submaximal exercise (absolute and relative workloads), and at maximal exercise with endurance training.
12. Heart rate is lower at rest and during an absolute submaximal workload with endurance training. It is unchanged at the same relative submaximal workload and at maximal exercise.
13. $\dot{V}O_{2\max}$ increases with endurance training; improvements of 15% are routinely reported with training programs that meet the recommendations of ACSM.
14. Endurance training leads to positive structural and functional adaptations in the vasculature because of vascular remodeling and improved endothelial function.
15. Blood pressure changes little or not at all at rest, during submaximal exercise, or during maximal exercise in normotensive individuals with endurance training.
16. Endurance training results in increased blood volume, with highly trained endurance athletes having 20–25% greater volume than untrained subjects. Changes in plasma volume occur early in a training program, with an 8–10% change occurring within the first week. Early changes (at 1 month) are due almost entirely to increases in plasma volume, whereas increases in red blood cells and hemoglobin occur later.
17. Endurance training results in changes in blood formation

and clot breakdown that decrease the likelihood of unnecessary clot formation.

Review Questions

1. How is overload manipulated to bring about cardiorespiratory adaptation? Consider exercise recommendations for fitness and physical activity guidelines for health benefit in your response.
2. Differentiate between central and peripheral cardiovascular adaptations.
3. Compare and contrast adaptations in cardiac output, stroke volume, heart rate, and blood pressure with endurance training at rest and during submaximal and maximal exercise.
4. Discuss the relevance of an individual's initial fitness level for expected improvements in fitness and health-related benefits.
5. Describe the physiological benefits of a warm-up and a cooldown period.
6. Explain the changes in blood volume that result from endurance training.
7. Describe changes in cardiac dimensions that result from endurance training, and explain how these structural changes support improved cardiac function.
8. Describe changes in blood clotting and breakdown that result from endurance training, and explain how these physiological changes support improved cardiovascular health.
9. Compare and contrast cardiovascular adaptations to dynamic endurance and dynamic resistance training.
10. Discuss the cardiovascular adaptations to HIIT.

Literature Search

1. Cardiovascular adaptations to exercise are a powerful reason for improved exercise performance and increased aerobic fitness. To better understand the research that has been done on this important issue, do a literature search using a search engine such as PubMed, Google scholar, or Web of Science.
 - a. Search adaptations to endurance training. This search will yield an enormous number of articles.
 - b. Refine your search using key terms that may reflect your interest in this area. For example:
 - i. Cardiac adaptations to resistance training
 - ii. Endothelial responses to HIIT
 - iii. Sex differences in ventricular diameter following endurance training
 - iv. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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14 Thermoregulation



Chapter Outline

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Summary

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OBJECTIVES

After studying the chapter, you should be able to:

- Articulate the importance of an exercise professional having a strong understanding of the influence of heat stress on safety and performance.
- Identify environmental factors that affect thermoregulation and be able to use indices of heat stress and windchill to assess the risks associated with exercise under various conditions.
- Describe thermal balance and discuss factors that contribute to heat gain and heat loss.

- Define the mechanisms by which heat is lost from the body, and describe how they differ under exercise conditions.
- Describe the body's regulatory system for temperature control in terms of the sensory input, neural integration, and effector responses to increase or decrease heat loss.
- Identify the factors that influence heat exchange between an individual and the environment.
- Describe the challenges to the cardiovascular system during exercise in a hot environment and in a cold environment.
- Describe the goals for fluid ingestion before, during, and after exercise.
- Differentiate among the types of heat illness in terms of severity and symptoms.
- Identify ways in which an exercise leader can prevent heat and cold injuries and illness.

Introduction

Many athletic competitions and recreational activities occur in settings in which hot or cold environmental conditions affect or may threaten physical performance, health, and even life. **Thermoregulation** is the process whereby body temperature is maintained or controlled under a wide range of such environmental conditions. In human beings, body temperature is maintained within a fairly narrow range by mechanisms that match heat production to heat loss. Human thermoregulatory responses rely heavily on the cardiovascular system to maintain body temperature. This chapter addresses issues related to exercise in environmental extremes, emphasizing the role of the cardiovascular system in mediating the body's responses to exercise under such conditions.

Thermoregulation The process whereby body temperature is maintained or controlled under a wide range of environmental conditions.

Exercise in conditions of environmental extremes can present

a serious challenge to the thermoregulatory and cardiovascular systems of the body. If the cardiovascular system cannot meet the concurrent demands of supplying adequate blood to the muscles and maintaining thermal balance in a hot environment, *exertional heat illness (EHI)* may ensue. Heat illness includes a spectrum of disorders from heat cramps to life-threatening heatstroke. **Heat stress** refers to physical work and environmental components that combine to create heat load on an individual. The physiological responses and resulting thermoregulatory processes that combat this heat stress are known as **heat strain**. A relatively stable core temperature is achieved through the interaction of behavioral and physiological reactions to thermal stimuli. Cold conditions can also pose problems. If an exerciser is unprepared or inadequately clothed for exercise in a cold environment, heat loss can exceed heat production, leading to cold-induced injury.

Heat Stress The physical work and environmental components that combine to create heat load on an individual.

Heat Strain The physiological responses and resulting thermoregulatory processes to combat heat stress.

Exercise professionals have a responsibility to understand the problems associated with exercise in extreme environmental conditions because they may affect an individual's performance or place an exerciser at risk for injury or even death. Understanding the body's responses to extreme environmental conditions is necessary for minimizing performance decrements and avoiding injury or illness in those who train and compete in adverse conditions. Understanding the body's responses to exercise in different environments begins with basic environmental measures and the measurement of body temperature.

Measurement of Environmental

Conditions

Human thermoregulation is affected by several environmental conditions, namely, ambient temperature (T_{amb}), relative humidity, and wind speed. *Ambient temperatures* are often measured with a mercury or digital thermometer and can vary greatly in areas that are in shade or direct sunlight. **Relative humidity** is a measure of the moisture in the air relative to how much moisture, or water vapor, can be held by the air at a given ambient temperature. Thus, 70% humidity means that the air contains 70% of the moisture that it can hold at that temperature.

Relative Humidity The moisture in the air relative to how much moisture (water vapor) can be held by the air at any given ambient temperature.

Specific scales are used to assess thermal heat load imposed by the environment. Wet bulb globe temperature (WBGT), developed by the military, is often used in industrial settings and athletic situations. The WBGT is calculated based on a formula that includes measures of air temperature, radiant heat load (measured by a thermometer in a small black globe that absorbs radiant heat), and relative humidity (measured by a thermometer covered with a wet cotton wick). Recommendations about the risk of heat stress at various WBGT levels are available in the American College of Sports Medicine (ACSM) Position Stand on exertional heat illness ([ACSM, 2007a](#)). Included in this publication are guidelines for modifying or canceling high-intensity or long-duration exercise when WBGT conditions are a risk for adults and children. In many cases, however, WBGT measurements are not available, and a simpler measure of environmental heat stress—the heat index—can be used to assess the risk. The **heat index** is used to estimate the risk of heat stress based on the ambient temperature and relative humidity (**Figure 14.1**).

Heat Index A scale used to determine the risk of heat stress

from measures of ambient temperature and relative humidity.

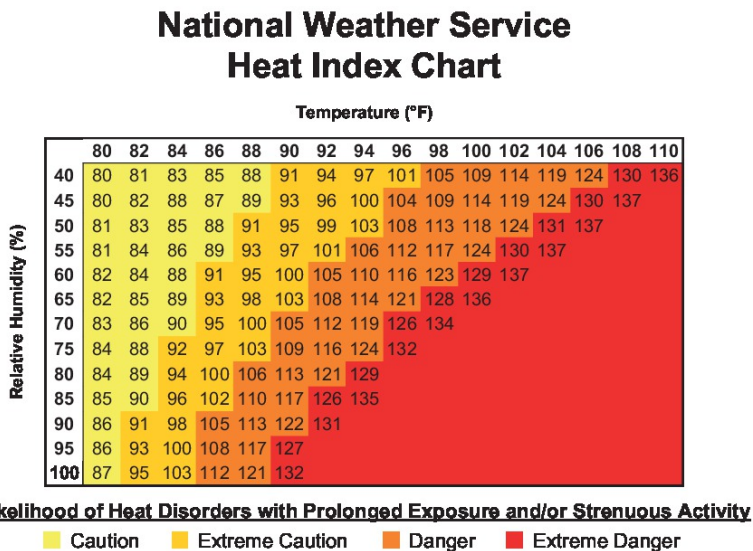


Figure 14.1 Heat Index Chart.

Low risk: use discretion, especially if unconditioned or unacclimatized; little danger of heat stress for acclimatized individuals who hydrate adequately. Moderate risk: heat-sensitive and unacclimatized individuals may suffer; avoid strenuous activity in the sun; take adequate rest periods and replace fluids. High risk: extreme heat stress conditions exist; consider canceling all exercise. **Source:** Modified from [Armstrong and Hubbard \(1985\)](#); U.S. National Weather Service.

Wind speed affects the amount of heat lost from the body and is used to calculate the windchill factor. [Table 14.1](#) presents the windchill chart adopted by the U.S. National Weather Service. This chart uses the wind velocity measured at a height of 1.3 m (5 ft). The windchill chart was developed as a public health tool to help prevent frostbite and cold-induced injuries by providing information for choosing appropriate clothing and activities based on available environmental data.

TABLE 14.1 Windchill Index

| Wind Speed (mi·hr ⁻¹) | Thermometer Reading (°F)* | | | | | | | | | | | | | | | | |
|--|---------------------------|----|----|----|----|----|-----|---------------|-----|-----|-----|-----------|-----|-----|-----|-----|-----|
| | 40 | 35 | 30 | 25 | 20 | 15 | 10 | 5 | 0 | -5 | -10 | -15 | -20 | -25 | -30 | -35 | -40 |
| 5 | 36 | 31 | 25 | 19 | 13 | 7 | 1 | -5 | -11 | -16 | -22 | -28 | -34 | -40 | -46 | -52 | -57 |
| 10 | 34 | 27 | 21 | 15 | 9 | 3 | -4 | -10 | -16 | -22 | -28 | -35 | -41 | -47 | -53 | -59 | -66 |
| 15 | 32 | 25 | 19 | 13 | 6 | 0 | -7 | -13 | -19 | -26 | -32 | -39 | -45 | -51 | -58 | -64 | -71 |
| 20 | 30 | 24 | 17 | 11 | 4 | -2 | -9 | -15 | -22 | -29 | -35 | -42 | -48 | -55 | -61 | -68 | -74 |
| 25 | 29 | 23 | 16 | 9 | 3 | -4 | -11 | -17 | -24 | -31 | -37 | -44 | -51 | -58 | -64 | -71 | -78 |
| 30 | 28 | 22 | 15 | 8 | 1 | -5 | -12 | -19 | -26 | -33 | -39 | -46 | -53 | -60 | -67 | -73 | -80 |
| 35 | 28 | 21 | 14 | 7 | 0 | -7 | -14 | -21 | -27 | -34 | -41 | -48 | -55 | -62 | -69 | -76 | -82 |
| 40 | 27 | 20 | 13 | 6 | -1 | -8 | -15 | -22 | -29 | -36 | -43 | -50 | -57 | -64 | -71 | -78 | -84 |
| 45 | 26 | 20 | 12 | 5 | -2 | -9 | -16 | -23 | -30 | -37 | -44 | -51 | -58 | -65 | -72 | -79 | -86 |
| Low risk | | | | | | | | Moderate risk | | | | High risk | | | | | |
| Low risk: use discretion; little danger, if properly clothed. | | | | | | | | | | | | | | | | | |
| Moderate risk: postpone exercise, if possible. Proper clothing is essential. Individuals at risk should take added precautions against overexposure. | | | | | | | | | | | | | | | | | |
| High risk: There is great danger from cold exposure; consider canceling all exercise. | | | | | | | | | | | | | | | | | |

*Note that this table uses °F; see Appendix A for conversion.

Source: U.S. Weather Service.

Measurement of Body Temperature

Exercise physiologists differentiate among temperatures in different body sites; most commonly used are core temperature (T_{co}) and skin temperature (T_{sk}). Even this distinction is simplistic, however, because core and skin temperature both vary among different specific sites. Core temperature is normally maintained within fairly narrow limits of approximately 36.1–37.8°C (97–100°F) in the resting individual (Marieb and Hoehn, 2019). Skin temperature is considerably cooler, averaging approximately 33.3°C (91.4°F). Skin temperature is more variable than core temperature because it is greatly influenced by environmental conditions.

Body temperature is commonly measured with a thermometer placed in the mouth. However, this method is affected by many factors, including breathing rate and recent fluid ingestion. Heavy breathing through the mouth and the ingestion of cold fluids result in artificially low oral temperatures, whereas the ingestion of hot liquids can artificially raise oral temperatures. For these reasons, oral measurement is not the method of choice among physiologists.

Core temperature is most accurately assessed by measuring the temperature of the blood as it enters the right atrium or measuring esophageal temperature. These measurements are invasive procedures, however, and are not practical for routinely measuring core temperature. Therefore, rectal temperature (T_{re}) or gastrointestinal (TGI) temperature (via an ingested radio transmitter—see “Focus on Research”) is often used in laboratory and research settings to measure core body temperature.

Although rectal and GI temperature measurements are accurate and reliable, they are not feasible for mass testing, nor are they routinely used to assess temperatures in exercise participants or athletes. Despite the importance of assessing body temperature for preventing and treating heat illness, there is no readily available, accurate, and convenient way of assessing core temperature in many situations such as athletic events. Often practitioners must rely on oral temperature measurements despite problems associated with this method. When appropriate, medical personnel often obtain rectal temperatures. Tympanic membrane (ear) temperatures (T_{tym}) are sometimes used to measure body temperature, but these instruments do not accurately detect exercise-induced changes in body temperature and thus should not be used to assess exertional heat stress ([Casa and Armstrong, 2003](#)).

Skin temperature is not routinely measured in field settings, but it is important because it affects the amount of heat that can be exchanged with the environment. Heat moves down a thermal gradient (both between the core and the skin and between the skin and the environment). Therefore, more heat is lost from the body when the skin is considerably hotter than the environment (larger gradient) than when the two temperatures are similar (smaller gradient). In the same way, more heat is gained by the body when the environment is considerably hotter than the skin. Skin temperatures are measured with thermocouples attached to the skin.

A great deal of effort has gone into the development of wearable physiological monitors to assess heat strain in athletes and workers using physiological measures. Typically, these monitors rely on algorithms to predict core temperature or to derive an index of physiological strain. However, there remain

barriers to widespread use of these devices, including the lack of a clear cut-off value for when an individual may be at risk of heat illness ([Morrissey et al., 2021](#); [Notley et al., 2018](#)).

Thermal Balance

Body temperature results from a balance between heat gain and heat loss (**Figure 14.2**). Although heat can be gained from the environment, most heat is typically produced in the body by metabolic activity. Heat is a by-product of cellular respiration; at rest, the body liberates approximately 60–80% of the energy from aerobic metabolism as heat (see Figure 2.1). The minimum energy required to meet the metabolic demands of the body at rest is called basal metabolic rate or resting metabolic rate; this accounts for a large proportion of the body's heat production.

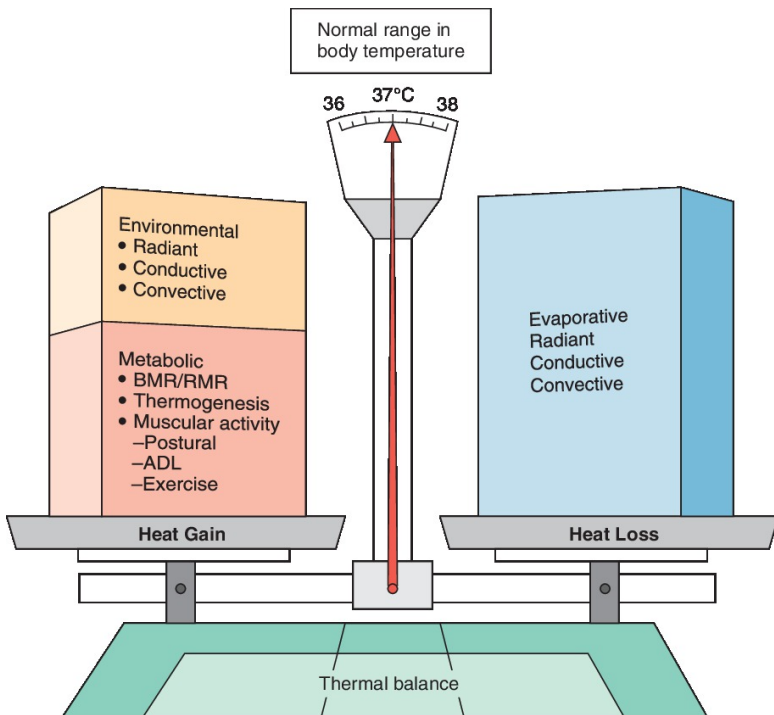


Figure 14.2 Thermal Balance.

Body temperature is maintained within a narrow range by a balance of heat gain and heat loss mechanisms.

The ingestion of food increases the body's production of heat. This is known as *thermogenesis* (see [Chapter 8](#)). Muscular activity also increases heat production, including activity related to muscle tone and posture; activities of daily living (ADL), such as bathing, dressing, and meal preparation; and planned exercise. Because metabolism increases greatly during physical activity, heat production also increases dramatically.

Heat can be exchanged (gained or lost) from the body through four processes: radiation, conduction, convection, and evaporation. The extent of heat gain or loss through these processes depends on environmental conditions: ambient temperature, relative humidity, and wind speed.

Radiant heat loss occurs through the emission of electromagnetic heat waves to the environment. It depends on the thermal gradient between the body and the environment. When the environmental temperature equals the skin temperature, no heat is lost through radiation. If the environmental temperature exceeds the skin temperature, radiation adds to the heat load of the body.

Conduction involves the direct transfer of heat from one molecule in contact with another. Conduction in humans primarily involves contact between the skin and the molecules of air and other substances in contact with the skin. The extent of conductive heat loss depends on the thermal gradient between the skin and the molecules in contact with the skin and on the thermal properties of the molecules in contact with the skin. Because water absorbs and conducts heat much better than air, submersion in cool water can more rapidly lower body temperature.

FOCUS ON RESEARCH

Core Temperature during a Half Marathon

Technology permits temperature to be measured relatively noninvasively by swallowing a vitamin-sized telemetric temperature sensor. Core body temperature in the lower GI tract is then transmitted to a small recorder (see Photo).

This technology was used to continuously measure core temperature of male soldiers participating in a half marathon (21 km or 13.1 mi) in a tropical environment. The soldiers were heat acclimatized and regularly participated in fitness training. The soldiers consumed an average of 1.18 L of fluid before and during the race and lost an average of 2.89 L of sweat—meaning on average they replaced only about 42% of sweat loss. The accompanying graphs show their individual core temperatures by race finish time (panel A, 105–111 minutes; panel B, 111–117 minutes; panel C, 122–146 minutes).

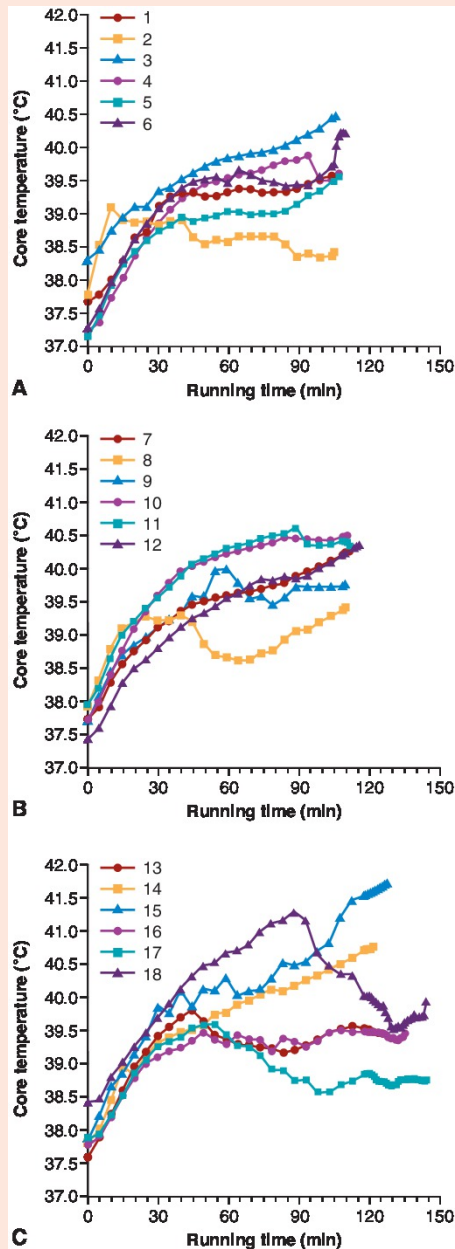


CorTemp Data Recorder and CorTemp Ingestible Core Body Temperature Sensor (photos courtesy of HQ, Inc.).

These measurements highlight the considerable variability in core temperature response even in a relatively homogeneous group of young, trained, acclimatized soldiers.

Also seen here is the magnitude of core temperature rise that these runners voluntarily achieved during a distance run in a hot, humid environment without medical consequence.

It is important to recognize that the core temperatures reported in this study do not indicate “safe” levels of core temperature for all individuals. Indeed, many unfit or unacclimatized individuals would suffer from heat illness at much lower core temperatures.



Individual Core Temperature during Running. Individual core temperature responses of 18 runners during the half marathon, presented in order of finishing time: 105–111 minutes, $N = 6$ (A); 111–117 minutes, $N = 6$

(B); 122–146 minutes, $N = 6$ (C).

Source: Reprinted with permission from Byrne, C., J. K. Lee, S. A. Chew, C. L. Lim, & E. Y. Tan: Continuous thermoregulatory responses to mass-participation distance running in the heat. *Medicine & Science in Sports & Exercise*. 38(5): 803–810 (2006). Copyright ©2006 The American College of Sports Medicine.

Convective heat loss depends on the movement of the molecules in contact with the skin. When there is a breeze, heat loss is greater because the warmer molecules are moved away from the skin. Thus, the thermal gradient is maintained, and more heat is lost through conduction.

Evaporation is the conversion of liquid into vapor. The evaporation of unnoticed water from the skin, called *insensible perspiration*, contributes to heat dissipation under resting and exercise conditions. However, the evaporation of sweat is the major mechanism for cooling the body under exercise conditions. Sweat is 99% water derived from plasma and released from eccrine glands. These glands are located throughout the body but are more concentrated on the forehead, hands, and feet ([Marieb and Hoehn, 2019](#)). The remaining 1% includes the electrolytes sodium (Na^+), chloride (Cl^-), and potassium (K^+) and traces of amino acids, bicarbonate (HCO_3^-), carbon dioxide (CO_2), copper, glucose, hormones, iron, lactic acid, magnesium (Mg^{2+}), nitrogen (N), phosphates (PO_4^-), urea, vitamins, and zinc ([Murray, 1987](#)). The exact proportion of these elements in sweat varies among individuals and within the same individual under different conditions; it is also influenced by the individual's fitness level ([Haymes and Wells, 1986](#)).

When the body is in *thermal balance*, the amount of heat lost equals the amount of heat produced, and body temperature remains constant. In this situation, when all heat exchange processes are added, the sum is equal to zero. This can be shown by the following formula ([Winslow et al., 1939](#)):

14.1 $M \pm R \pm C \pm K - E = 0$

where M is metabolic heat production, R is radiant heat exchange, C is convective heat exchange, K is conductive heat exchange, and E is evaporative heat loss.

The \pm sign for radiant, convective, and conductive processes indicates that heat can be lost or gained by the body through these mechanisms. When the environment is hotter than the skin temperature, heat is gained by the body (a $+$ sign in the equation). When skin temperature is higher than the environment temperature, heat is lost from the body (a $-$ sign in the equation). Evaporation cannot add to the heat load of the body. This mechanism can only dissipate heat; thus, there is only a negative sign in the equation for evaporation.

Heat Exchange

The exchange (transfer) of heat between the body and the environment occurs by the mechanisms just described: conduction, convection, radiation, and evaporation. Heat exchange is represented schematically in **Figure 14.3**. These four mechanisms are important for dissipating heat to the environment under most conditions. However, when ambient temperatures are high, conduction, convection, and radiation may actually add heat to the body.

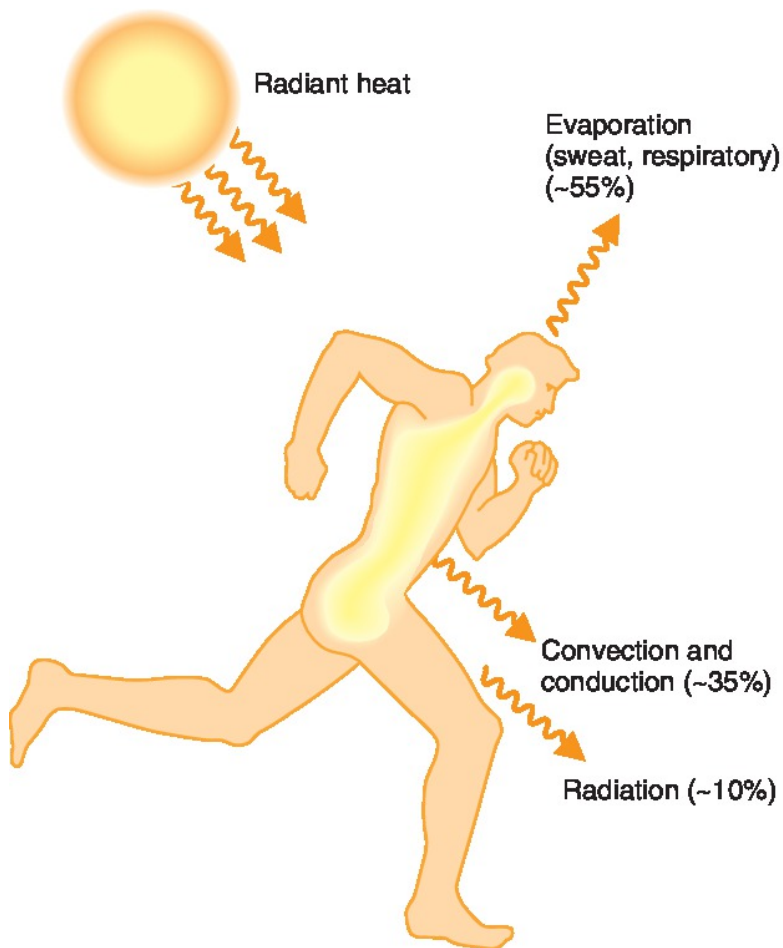


Figure 14.3 Mechanisms of Heat Exchange and Percentage of Heat Loss.

Source: Modified from [Gisolfi and Wenger \(1984\)](#).

The effectiveness of heat exchange between an individual and the environment is affected by five factors:

1. The thermal gradient
2. The relative humidity
3. Air movement
4. The degree of direct sunlight

5. The clothing worn

The greater the difference between two temperatures—called the thermal gradient—the greater the heat loss is from the warmer of the two. Typically, the body is warmer than the environment, so heat moves down its thermal gradient to the environment. More heat is lost in cooler environments because the thermal gradient is greater.

High humidity decreases evaporative heat loss from the body because the air is already largely saturated with water vapor. Relative humidity is the primary determinant of the extent of evaporative cooling; on humid days, evaporative cooling is limited. Although an exerciser may sweat profusely when humidity is high, the sweat does not evaporate as effectively.

Air movement increases convective heat loss from the skin to the environment. Thus, on windy days, more heat is lost from the body.

Direct sunlight can add considerably to the radiant heat load of an individual. Conversely, shade or cloud cover can often provide significant relief from heat.

Clothing also influences the extent of heat transfer with the environment. In cold weather, clothing protects against excessive heat loss. For example, long sleeve undershirts and tights are often added to football uniforms to help retain heat in cold weather outdoor games. Conversely, clothing can interfere with heat dissipation in hot weather by decreasing convective heat loss. Clothing that is lightweight, nonrestrictive, and light-colored promotes heat loss; heavy, dark clothing interferes with the dissipation of heat. Football uniforms, especially the protective padding, are an example of heavy, restrictive clothing that has been shown to increase physiological strain ([Armstrong et al., 2010](#)). Helmets, in particular, limit heat loss by encapsulating the head. The color of the clothing is also a consideration. Dark colors absorb light and thereby add to the radiant heat load; light colors reflect light. Thus, dark-colored uniforms can contribute to the heat stress of athletes as they play on a synthetic field that radiates heat in the hot sun. Short shirts and the use of mesh materials are attempts to aid in heat dissipation under these conditions.

Thermoregulation

The body regulates its internal body temperature within a narrow range despite wide variations in environmental temperatures. The process by which the body regulates body temperature is termed thermoregulation.

Normal Body Temperature

The human body typically regulates its temperature within approximately 1°C near 37°C (98.6°F) (**Figure 14.4**). Maintaining temperature within this range is important because changes in body temperature dramatically affect biological function by altering chemical reactions and ultimately directly damaging body tissue. Although body temperature is carefully regulated, it is not consistent throughout the body. The core is tightly regulated to maintain a temperature near 37°C . The shell (skin), on the other hand, varies greatly in temperature because it is strongly influenced by environmental conditions.

Thermal Regulation

Condition

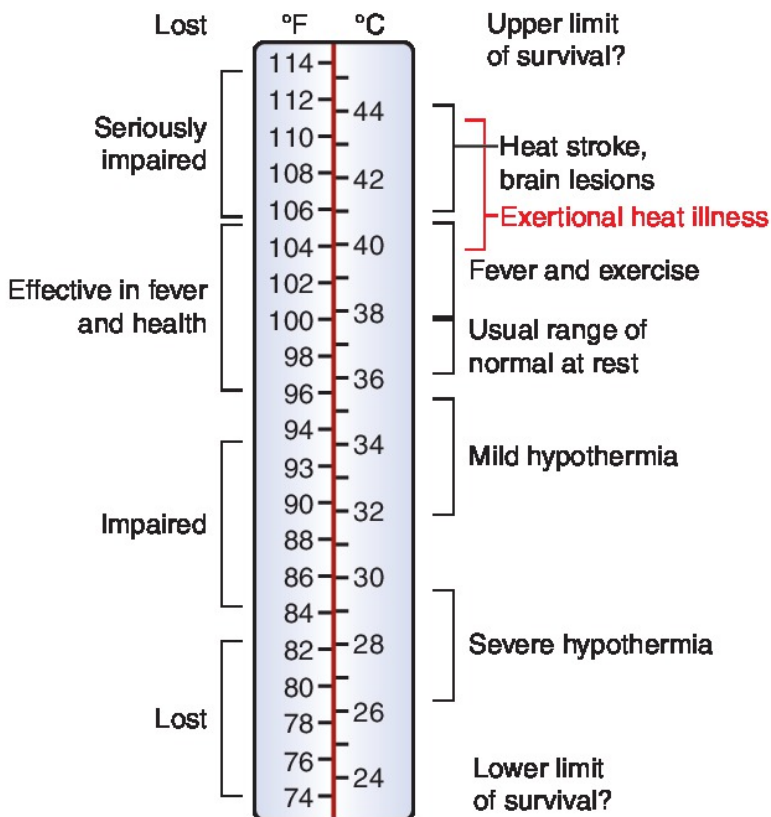


Figure 14.4 Range of Core Temperature and Associated Conditions.

A range of temperatures is associated with each condition. A given temperature may fall into more than one category depending on individual characteristics. **Source:** Modified from [Wenger and Hardy \(1990\)](#).

Core body temperature fluctuates throughout the day (circadian rhythm) and is typically 0.7–0.8°C higher in the late afternoon than in the early morning hours. Core temperature can also vary throughout the menstrual cycle, increasing by 0.5–0.75°C at ovulation ([Stitt, 1993](#)).

Behavioral and Physiological Thermoregulation

Core body temperature is controlled by regulatory mechanisms broadly categorized as behavioral thermoregulation and physiological thermoregulation. Behavioral thermoregulation involves conscious efforts to regulate body temperature such as adding clothing or increasing activity to stay warm. Physiological thermoregulation involves the reflex control of effector organs (blood vessels, sweat glands, and skeletal muscle) to maintain temperature within homeostatic limits. **Figure 14.5** summarizes the thermoregulatory system that maintains core body temperature (Hall and Hall, 2021; Sawka and Wenger, 1988).

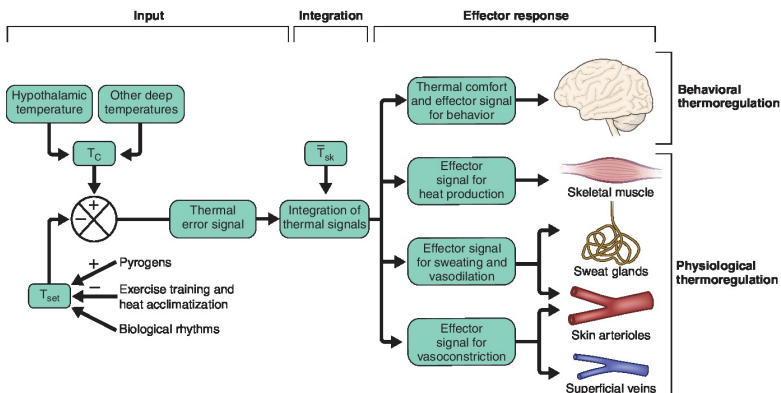


Figure 14.5 Schematic Diagram of Behavioral and Physiological Thermoregulation.

Several factors (biological rhythms, training and acclimatization, and circulating pyrogens) influence the core temperature set point (T_{set}). Core temperature is compared to the T_{set} , which is integrated with thermal input from the skin to produce effector signals for behaviors and physiological thermoregulatory responses. **Sources:** Modified from Sawka, M. N., & C. B. Wenger: Physiological responses to acute exercise-heat stress. In Pandolf, K. B., M. N. Sawka, & R. R. Gonzalez (eds.): *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Dubuque, IA: Brown & Benchmark, 97–151 (1988).

Reprinted with permission from Michael N. Sawka, MD; Hall, J. E. & M. E. Hall: *Guyton and Hall Textbook of Medical Physiology* (14th ed.). Philadelphia, PA: Elsevier (2021). Philadelphia, PA: Elsevier (2021).

The hypothalamus is the primary integration center for the control of body temperature. The hypothalamus receives input from sensors that detect core body temperature and skin (shell) body temperature. These signals are compared to the body's "set point" for temperature, and, if necessary, effector responses are initiated to increase heat production or facilitate heat loss. The primary physiological effector organs for heat dissipation are the sweat glands that produce sweat that can cool the body via evaporative heat loss, and skin arterioles that can vasodilate to increase heat loss or vasoconstrict to preserve body heat. Skeletal muscle activity is a major contributor to body temperature even at rest because muscle tissue is metabolically active and produces heat as a by-product. Importantly, during exercise, increased skeletal muscle activity is the primary factor that increases core temperature, which causes the hypothalamus to provide effector signals to increase heat dissipation through sweating and vasodilation. **Figure 14.6** reveals how the primary heat dissipation mechanisms, sweating (**Figure 14.6A**) and vasodilation, shown in terms of blood flow (**Figure 14.6B**), are affected by changes in core temperature. As core temperature increases, sweating increases. Furthermore, sweating is greater at any given core temperature when the skin is warmer. Blood flow increases with increasing temperature when the skin is warm (35.5°C), but when the skin is cool (30.3°C), forearm blood flow remains steady until core temperature reaches approximately 37.5°C, at which time blood flow increases.

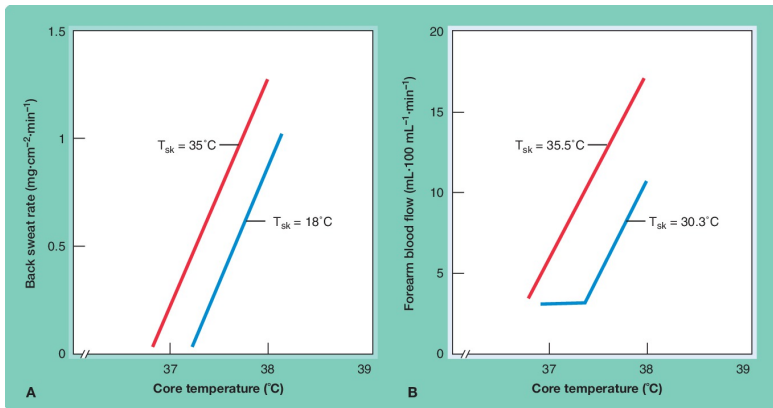


Figure 14.6 Effect of Core and Skin Temperature on Sweating Rate and Forearm Blood Flow.

At any given core temperature, sweating (A) is greater when the skin is warmer. Forearm blood flow (B) is constant with an increase of core temperature to approximately 37.5°C, after which blood flow increases with rising core temperature when skin temperature is approximately 30°C. When the skin is warm (35.5°C), any increase in core temperature is associated with increased blood flow.

Sources: Panel A modified with permission from Sawka, M. N., R. R. Gonzalez, L. L. Drolet, & K. B. Pandolf: Heat exchange during upper- and lower-body exercise. *Journal of Applied Physiology*. 57(4):1050–1054 (1984). Copyright © 1984 The American Physiological Society. All rights reserved. Panel B modified with permission from Wenger, C. B., M. F. Roberts, J. A. J. Stolwijk, & E. R. Nadel: Forearm blood flow during body temperature transients produced by leg exercise. *Journal of Applied Physiology*. 38(1): 58–63 (1975). Copyright © 1975 The American Physiological Society. All rights reserved.

Exercise in the Heat

Many sporting events and recreational activities occur in hot

environments. As just described, the body's thermoregulatory system dissipates excess body heat resulting from muscle activity and environmental heat. However, strenuous exercise and environmental extremes can combine to overwhelm the thermoregulatory capacity of the body.

Body Temperature during Exercise in the Heat

Body temperature increases during prolonged exercise because heat production exceeds heat dissipation. As shown in **Figure 14.7**, the increase in core temperature is proportional to the metabolic power output performed (measured in watts) and is largely independent of environmental temperature over a fairly wide range of temperatures. This suggests that the heat-dissipating mechanisms of the body can compensate for the increased metabolic heat production and stabilize body temperature. The term “*prescriptive zone*” is used to describe the combination of environmental conditions and work intensities at which thermoregulatory mechanisms are effective in preventing dangerous rises in body temperature during prolonged work ([Lind, 1963](#)). More generally, this zone might also be described as a *compensable zone*, in which the thermoregulatory system can effectively compensate for increased metabolic heat production by increasing heat dissipation so that body temperature does not continue to rise, even though it reaches a steady state that is greater than resting body temperature.

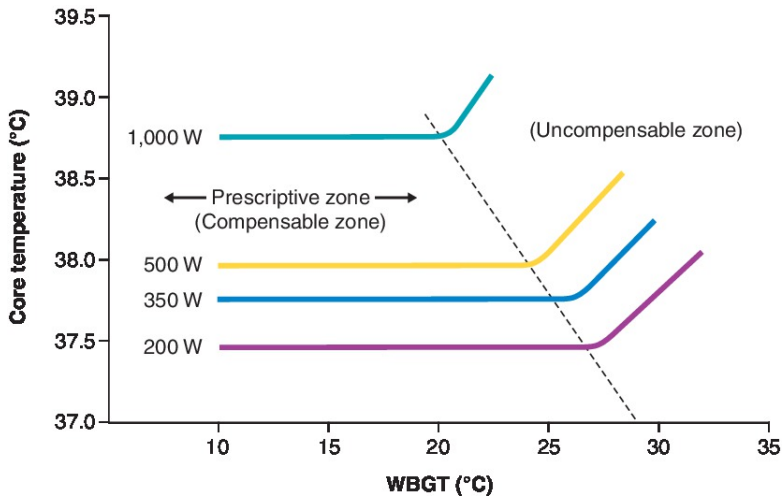


Figure 14.7 Core Temperature Responses during Exercise of Various Intensities (200–1,000 W) in Different Environmental Conditions.

In the compensable zone, core temperature is elevated due to increased metabolic heat production but reaches a steady state because the body is able to dissipate the heat that is being generated. In the uncompensable zone, the heat-dissipating mechanism of the body cannot fully compensate for the increased heat production and body temperature continues to rise—sometimes dangerously. **Source:** Modified from [Lind \(1963\)](#).

When the body cannot dissipate the metabolic heat generated during exercise, body temperature continues to rise (see **Figure 14.7**, area to the right of dashed line). **Uncompensable heat stress** is a condition in which the evaporative cooling that is needed is greater than the evaporative cooling permitted by the environment ([Montain et al., 1994](#); [Sawka and Pandolf, 2001](#)). During uncompensable heat stress, steady-state core temperature cannot be achieved, and body temperature continues to rise until exhaustion occurs. Uncompensable heat stress is associated with exhaustion from heat strain at relatively low core temperatures ([Montain et al., 1994](#); [Sawka and Pandolf, 2001](#)).

Uncompensable Heat Stress A condition in which the evaporative cooling that is needed is greater than the evaporative cooling permitted by the environment.

Obviously, the change in core temperature with exercise is affected by environmental temperature and exercise intensity and duration. But, even when the same exercise is performed in the same environmental conditions, repeated bouts of moderate- to high-intensity intermittent exercise in the heat can result in greater heat strain during a second exercise session on the same day (separated by 2 hours) and on the following day. Researchers had young, recreationally active men perform a 20-minute exercise protocol (including jogging, running, walking, running, jogging, running, rest) 6 times for a total of 2 hours of exercise. All participants could complete the first session, but only 35% of the participants could complete the second session that was performed 2 hours later. When the exercise session was repeated 24 hours after the first session, 71% of participants were able to complete the entire 2 hours of exercise (Pryor et al., 2019). Although these findings are largely consistent with expectations, the reason for the differences is not entirely clear. Participants had similar immediate postexercise HRs and core temperatures in all three conditions, and starting HR and core temperature were similar between the first session on day 1 and the session on day 2; though HR was about 20 beats higher at the start of the second session on day 2 than the first session and core temperature was about 0.3°C higher during the second session. The authors concluded that high intensity exercise in the heat negatively impacts exercise during subsequent exercise sessions. The end exercise physiological and perceptual measures were similar despite the fact that exercise was conducted for a shorter period on average in subsequent trials. The authors recommend that multiple exercise sessions should be avoided on the first day of physical activity in the heat as this may increase the risk of heat illness (Pryor et al., 2019).

Heat Exchange during Exercise

Heat production and heat transfer occur by the same mechanisms

during exercise as they do at rest. However, during exercise, the total body metabolism may increase to 15–20 times the resting rate (Sawka and Pandolf, 1990). In this situation, metabolic heat production may increase to a greater extent than heat dissipation; thus, the body stores heat and body temperature increases. The increase in body temperature with exercise is termed **hyperthermia**.

Hyperthermia The increase in body temperature with exercise.

Metabolic heat produced in the muscles is transported to the core of the body and skin by the blood. Heat is also transferred to the skin and exchanged with the environment by conduction, convection, radiation, and evaporation. Two physiological mechanisms allow the body to dissipate heat in an attempt to maintain thermal balance during exercise: an increase in sweating rate and vasodilation of the cutaneous (skin) vessels. Evaporative cooling of sweat is the primary mechanism by which the body cools itself during exercise in warm temperatures. Vasodilation of cutaneous vessels brings the warm blood close to the body's surface so that heat can be dissipated to the environment via conduction, radiation, and convection, assuming that the ambient temperature is cooler than the body.

Figure 14.8 depicts the relative importance of heat exchange mechanisms during 60 minutes of cycling exercise ($900 \text{ kpm} \cdot \text{min}^{-1}$) at various ambient temperatures. Total heat loss (THL) remains relatively constant across a wide range of ambient temperatures. The fact that the metabolic heat production (M) exceeds the THL accounts for the increased temperature that occurs with exercise. **Figure 14.8** reinforces the earlier observation that the increase in body temperature (heat storage) is relatively constant over a wide range of ambient temperatures as long as the work rate is constant. This is true because the metabolic heat production depends on the amount of work being done, regardless of temperature. The THL also remains the same. Thus, the difference between metabolic heat production and THL—that is, heat storage—is constant. Although THL remains relatively constant, it occurs through different processes at

different ambient temperatures. At high temperatures, evaporative heat loss is primary, and radiant and convective heat losses are less effective. This combination is effective, however, only when the humidity is low enough to allow sweat to evaporate. In high humidity, evaporative heat loss is less effective, and heat storage (body temperature) increases. This explains why temperature and humidity must be considered together (using the heat index in [Figure 14.1](#), or the WBGT) for determining when activity is safe.

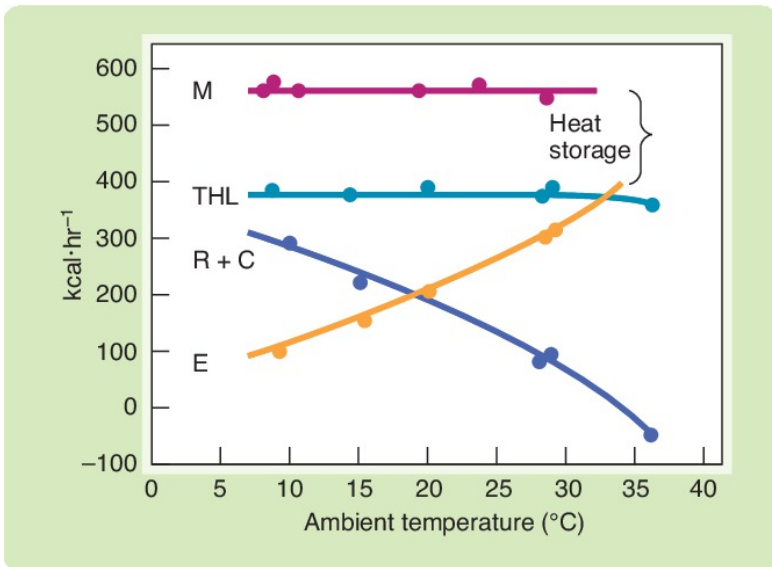


Figure 14.8 Mechanisms of Heat Loss during Exercise at Different Ambient Temperatures.

M, metabolic heat production; THL, total heat loss; R + C, heat loss by radiation and conduction; E, heat loss by evaporation. Exercise was performed for 60 minutes at the work rate of $900 \text{ kpm} \cdot \text{min}^{-1}$ at each ambient temperature. Notice that the metabolic heat produced and the THL (and thus the heat storage) are constant over a wide range of temperatures ($\sim 10\text{--}36^{\circ}\text{C}$; $50\text{--}97^{\circ}\text{F}$), although they are achieved via different mechanisms. Evaporative heat loss becomes increasingly important as ambient temperature increases. **Source:** Reprinted with permission from Gisolfi,

During heavy exercise, the sweat rate can increase dramatically. Sweat rates vary considerably among individuals, depending on genetics and fitness level. A fit person begins sweating at a lower body temperature and sweats more profusely. For any one individual, sweat rate depends on environmental conditions, exercise intensity, fitness level, degree of acclimatization, and hydration status. **Figure 14.9** estimates hourly sweating rates for running at various speeds in different environmental conditions ([Sawka and Pandolf, 1990](#)). Notice how common it is for sweating rates to exceed $1 \text{ L}\cdot\text{hr}^{-1}$ of sweat. Clearly, such a water loss will lead to a decrease in total body water and plasma volume and will have deleterious effects on cardiovascular function if fluid is not replaced. The evaporation of sweat is the primary defense against heat stress. Sweating itself does not cool the body; the sweat must evaporate. The evaporation of sweat produces a cooling of the body because energy is needed to convert the liquid sweat into a vapor, and this energy is extracted from the immediate surroundings. The amount of energy needed for the evaporation of sweat can be quantified: 580 kcal of heat energy is released for each liter of water that is vaporized ([Hall and Hall, 2021](#)).

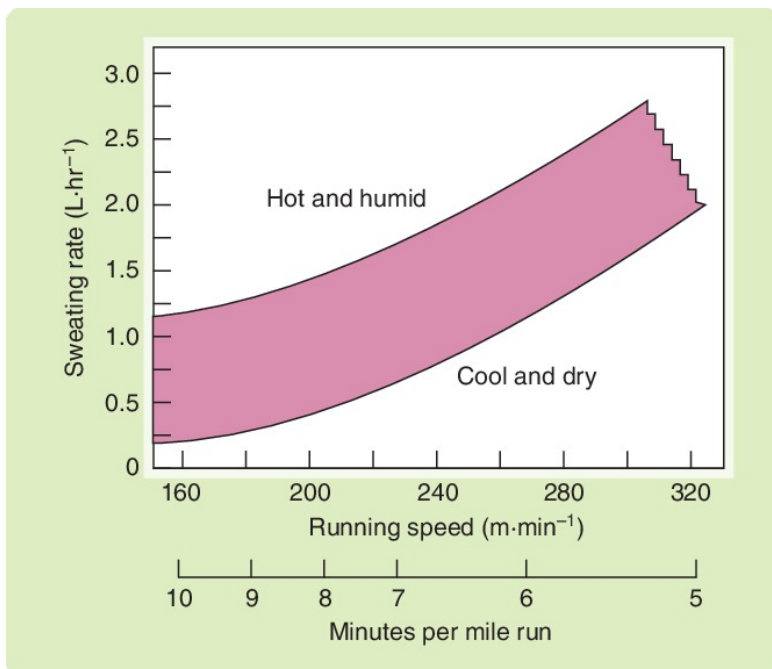


Figure 14.9 Estimated Sweating Rate at Various Running Speeds.

Source: From Sawka, M. N., & K. B. Pandolf: Effects of body water loss on physiological function and exercise performance. In Gisolfi, C. V., & D. R. Lamb (eds.): *Perspectives in Exercise Science and Sports Medicine*. Vol. 3: *Fluid Homeostasis During Exercise*. Indianapolis, IN: Benchmark Press, 1–38 (1990). Reprinted with permission from Michael N. Sawka, MD.

Cardiovascular Demands of Exercise in the Heat

As discussed fully in previous chapters, the cardiovascular system responds to exercise by increasing blood flow to the active tissue to support energy production. The cardiovascular system is also directly involved in thermoregulation and heat dissipation in

particular. The cardiovascular system faces several interrelated challenges when exercise is performed in warm or hot conditions:

1. The skin and the muscles compete for blood flow. The muscles need increased blood flow to meet the demands of metabolic activity, and the skin needs increased blood flow to dissipate heat from the body's core.
2. Vasodilation in cutaneous vessels effectively decreases venous return, thus decreasing stroke volume. Therefore, cardiac output may be reduced at a time when the demands for flow are greatest (Rowell, 1986).
3. Sweating results in a reduced plasma volume, contributing to the reduction in stroke volume and, therefore, cardiac output.
4. Adequate blood pressure must be maintained to perfuse the vital organs, including the brain, kidney, and liver. The ability to maintain blood pressure is challenged by widespread vasodilation in the skeletal muscle beds and cutaneous vessels, which decreases total peripheral resistance.

Figure 14.10 presents cardiovascular responses to various intensities of exercise (light, moderate, and heavy) under hot and thermoneutral conditions. As seen in **Figure 14.10A**, during short-term, light submaximal exercise, cardiac output increases to a similar degree in both hot and thermoneutral environments (Rowell, 1974). However, cardiac output in a hot environment is achieved by a higher heart rate (**Figure 14.10C**) and a lower stroke volume (**Figure 14.10B**) than in a thermoneutral environment. This reduced stroke volume occurs during hot conditions because of sweating and vasodilation in the cutaneous vessels, which decreases central venous volume. Mean arterial pressure is similar to or only slightly lower in hot environments than in thermoneutral environments because of vasoconstriction in the kidneys and digestive tract.

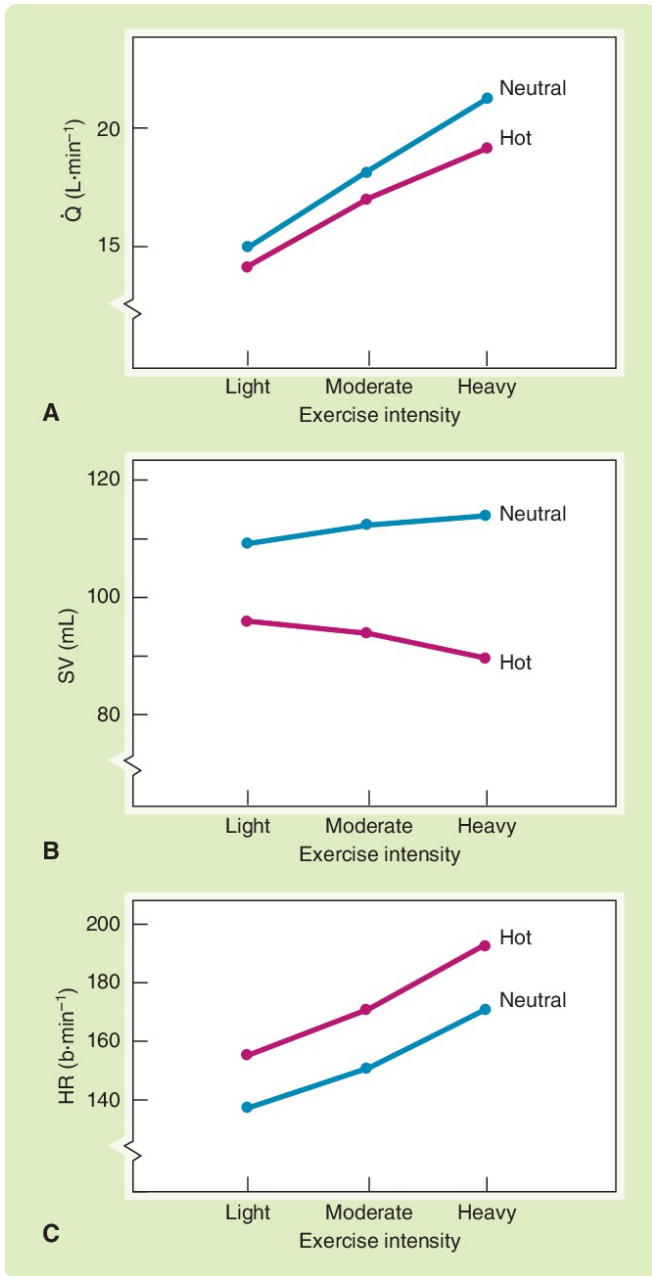


Figure 14.10 Cardiovascular Responses to Hot or Thermoneutral Conditions.

A. Cardiac output (\dot{Q}). B. Stroke volume (SV). C. Heart rate

(HR). **Source:** Based on Rowell (1974).

During prolonged, heavy submaximal exercise in the heat, cardiac output increases less than in a thermoneutral environment (**Figure 14.10A**). Cardiac output in hot environments fails to reach levels attained under thermoneutral conditions during heavy exercise because stroke volume declines progressively as the intensity of exercise increases. Although the heart rate is higher, it cannot compensate fully for the reduced stroke volume during heavy exercise in hot conditions. Thus, cardiac output is lower in hot conditions than in thermoneutral conditions.

During long-term, heavy submaximal exercise in the heat, vasoconstriction occurs in the digestive and renal areas in an attempt to maintain mean arterial blood pressure. Vasoconstriction may result in ischemia and even tissue damage in extreme conditions (Rowell, 1986). The regulatory mechanisms that maintain blood pressure are stressed by the excessive water loss that occurs with profuse sweating. If this fluid is not replaced, stroke volume, cardiac output, and blood pressure will decrease. Additionally, performance will suffer, and heat illness becomes increasingly likely.

Maximal cardiac output during incremental exercise to maximum is lower when performed in hot conditions than in thermoneutral conditions. This decreased cardiac output results from a lower stroke volume, because maximal heart rate is unchanged, although maximal heart rate may occur earlier in the heat. The estimated distribution of cardiac output during an incremental exercise test performed under neutral and hot conditions is presented in **Figure 14.11**.

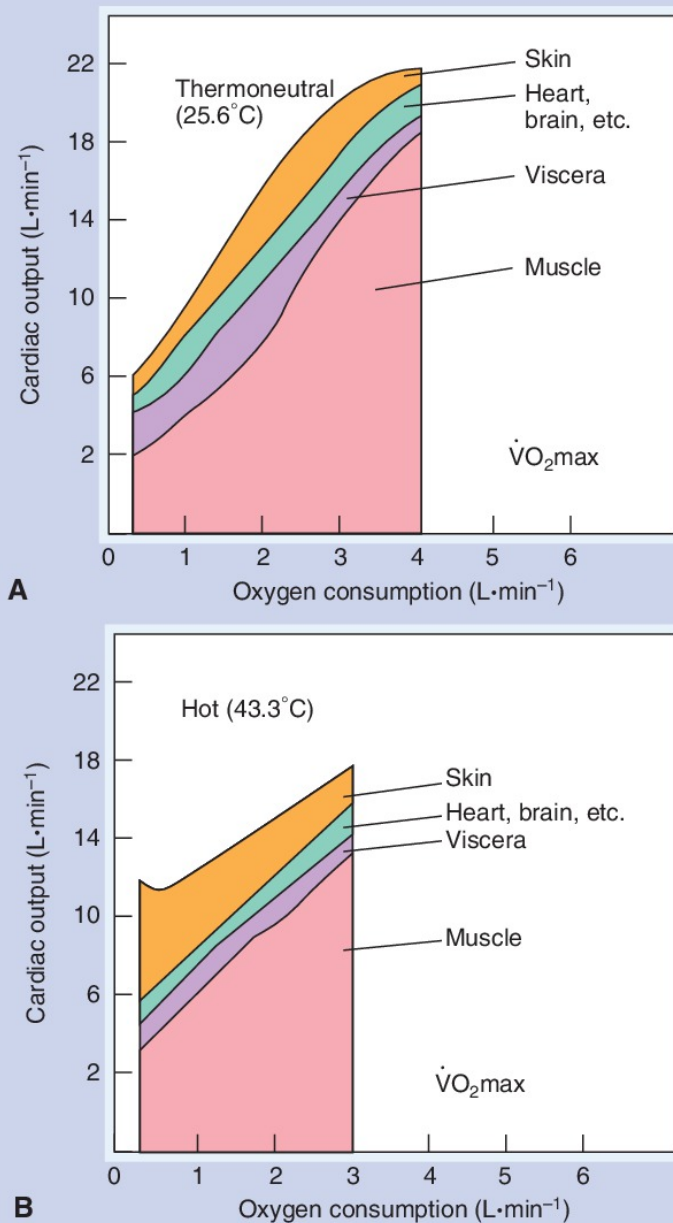


Figure 14.11 Distribution of Cardiac Output during Incremental Exercise in (A) Thermoneutral (25.6°C; 78°F) and (B) Hot (43.3°C; 110°F) Conditions.

Source: Modified with permission of Oxford University Press from Rowell, L. B.: Human Circulation Regulation during Physical Stress. New York, NY: Oxford University Press (1986); permission conveyed through Copyright Clearance Center, Inc.

When exercise is performed in a hot environment, it cannot be performed as long as in cooler conditions. Consequently,

$\dot{V}O_2 \text{ max}$ is lower in a hot environment (**Figure 14.11B**). Cardiac output increases during exercise in both conditions.

However, cardiac output at $\dot{V}O_2 \text{ max}$ is less in the hot environment, presumably because blood is displaced in cutaneous veins, thus decreasing venous return and stroke volume. Blood flow to the active skeletal muscles increases throughout exercise in both conditions. However, blood flow to skeletal muscle is a smaller proportion of total blood flow in a hot environment than in a thermoneutral environment, because skin blood flow accounts for a larger portion of the blood flow in the hot

environment. At $\dot{V}O_2 \text{ max}$ in the hot condition, visceral blood flow is severely reduced in an effort to support the muscles with adequate blood flow and maintain blood pressure. The lower cardiac output and the decrease in blood flow when maximal exercise is performed in a hot environment both contribute to a

lower $\dot{V}O_2 \text{ max}$ and an earlier onset of fatigue.

Factors Affecting Cardiovascular Response to Exercise in the Heat

Several factors affect an individual's response to exercise in the heat. The following sections discuss five key factors: exercise intensity, acclimatization, cardiovascular fitness, body composition, and hydration level.

Exercise Intensity

Intuitively it makes sense the more exercise (muscular work) that is performed, the greater the thermal strain. Importantly, the

changes in core temperature, and in sweating rate, are most closely associated with the absolute amount of work that is performed, and hence the amount of heat produced, than the relative amount of work. This was shown in a classic study when [Jay et al. \(2014\)](#) examined change in core temperature and sweat rates during exercise at a fixed percentage of $\dot{V}O_2 \text{ max}$ and at a fixed absolute workload in both fit and unfit men. The changes in core body temperature and sweat rate were greater in the fit group than in the unfit group during exercise at 60% of $\dot{V}O_2 \text{ max}$, because the fit group had performed more muscular work. In contrast, changes in core temperature and sweating rate were similar between the fit and unfit men during exercise at a fixed exercise intensity [Jay et al. \(2014\)](#).

Acclimatization

Acclimatization refers to adaptive changes in an individual who undergoes prolonged or repeated exposure to a stressful environment; these changes reduce the physiological strain produced by such an environment. Although acclimatization and acclimation are often used interchangeably, scientists distinguish between *acclimation*—which is a short-term adaptation (i.e., one that occurs in days or weeks) that is induced in a laboratory—and *acclimatization*—which is an adaptation that occurs over a longer time period and occurs in a natural climate. Acclimatization to heat results from repeated exposure to heat sufficient to increase core body temperature and elicit moderate to profuse sweating ([Werner, 1993](#)). Repeated exercise in hot climatic conditions results in improved efficiency of the thermoregulatory responses, augmented cardiovascular function, and enhanced endurance performance based on many, but not all studies ([Rahimi et al., 2019](#); [Tipton et al., 2008](#)). Acclimation also appears to attenuate the sensation of fatigue during exercise heat stress ([Willmott et al., 2019](#)). Light to moderate exercise in the heat for 1–2 hours a day can lead to positive adaptations within a few days although many acclimatization programs use 10–14 days to optimize adaptations.

Acclimatization The adaptive changes that occur when an

individual undergoes prolonged or repeated exposure to a stressful environment; these changes reduce the physiological strain produced by such an environment.

Acclimatization to heat involves several underlying mechanisms:

1. An expansion of plasma volume that leads to an enhanced cardiac output during exercise and a decrease in heart rate and cardiovascular strain at a given level of exercise in the heat (Lorenzo et al., 2010; Nielsen et al., 1993).
2. Sweating begins earlier and at a lower body temperature.
3. The sweating rate for a given core temperature is higher and can be maintained longer (Tipton et al., 2008; Wenger, 1988).

Figure 14.12 compares changes in rectal temperature, heart rate, and sweating rate during 4 hours of aerobic exercise before and after acclimatization (Wyndham et al., 1964). These data support the benefits of heat acclimatization in lowering thermal and cardiovascular strain. Proper acclimatization ensures that individuals can perform longer and more safely in the heat.

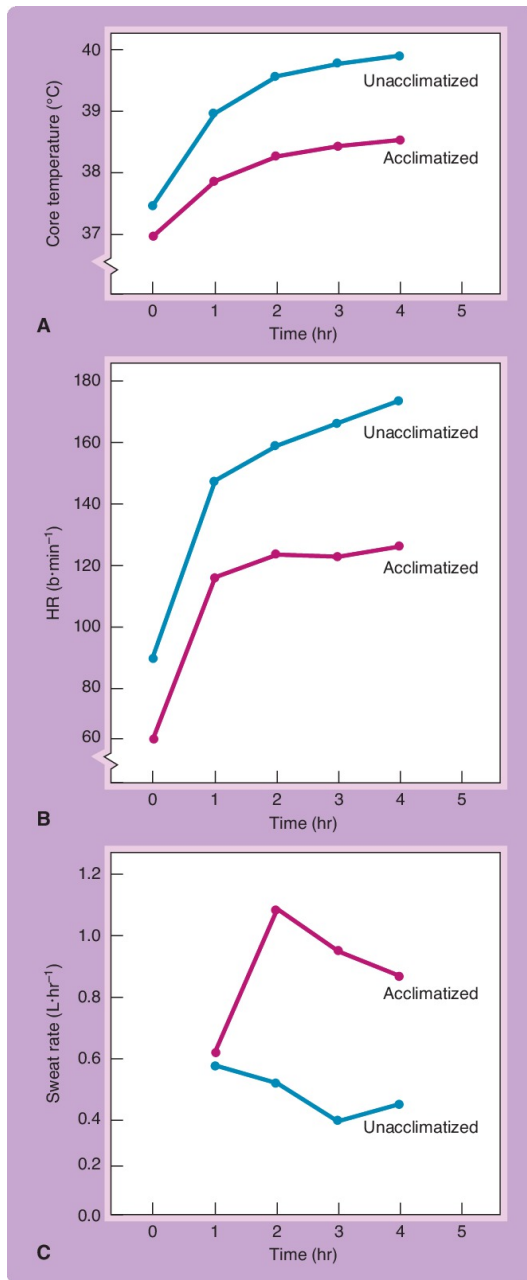


Figure 14.12 Comparison of Core (Rectal) Temperature (A), Heart Rate (B), and Sweat Rate (C) in Acclimatized and Unacclimatized Individuals during Long-Term,

Moderate to Heavy Submaximal Exercise.

Source: Based on data in [Wyndham et al. \(1964\)](#).

Fitness Level

Aerobic fitness is often reported to improve an individual's thermoregulatory function and heat tolerance. Endurance training results in a lower resting core temperature, a larger plasma volume, an earlier onset of sweating, and a smaller decrease in plasma volume during exercise ([Drinkwater, 1984](#); [Werner, 1993](#)). Therefore, individuals who are aerobically fit are better able to handle the cardiovascular demands, particularly the increased sweating, of exercise in hot conditions than sedentary individuals. It is also likely that exercise training leads to perceptual changes that make individuals more willing to exercise in the heat. High-intensity interval training in thermoneutral conditions has been reported to decrease the time required for acclimatization to exercise in a hot environment ([Cohen and Gisolfi, 1982](#)). In fact, many of the changes in cardiovascular function that are seen when repeated bouts of exercise are used to induce acclimatization may primarily reflect a training adaptation ([Tschakovsky and Pyke, 2008](#)).

Body Composition

Excessive body fat is a liability for thermoregulation during exercise in the heat. Greater adiposity contributes to heat stress by two primary mechanisms. First, adipose tissue interferes with the dissipation of heat; body fat acts to insulate the core. Second, body fat adds to the metabolic cost of activity by adding weight to the body that must be moved. Heat illness is more common in overweight individuals and is more likely to be fatal to these individuals ([Bedno et al., 2010](#); [Henshell, 1967](#)).

Hydration Level

An individual's level of hydration has a large impact on exercise tolerance and on the cardiovascular responses to long-term exercise in the heat. A decrease in body water, termed dehydration or hypohydration, leads to several negative effects

on physiological function, including cardiovascular, thermoregulatory, metabolic, and central nervous system impairments that become increasingly greater as dehydration worsens (Murray, 2007). Dehydration increases physiological strain and perceived effort for the same absolute amount of exercise, and this effect is exacerbated when exercise is performed in the heat (ACSM, 2007a). Not only does dehydration lead to greater physiological strain, but research demonstrates that dehydration also leads to impaired performance. Although there is still considerable debate on precisely how much body water can be lost before performance is affected, there is strong evidence that dehydration greater than 2% of body weight leads to impaired performance in endurance events (Murray, 2007). Short-term anaerobic events and muscular power events appear to be less affected by modest dehydration.

Body water accounts for 65–70% of the total body mass of an average adult. Water balance is generally well regulated on a daily basis as long as water and food are readily available (Sawka et al., 2005). However, during long-term exercise in the heat, if fluid is not replaced, profuse sweating leads to large acute losses of total body water and a reduction in plasma volume (Wade and Freund, 1990). The reduced plasma volume, in turn, leads to decreased stroke volume and an increased heart rate during prolonged exercise. Therefore, it is recommended that fluid ingestion during exercise be sufficient to prevent excessive dehydration (>2% of body weight) and excessive loss of electrolytes to prevent compromised performance and health risks. Individuals vary considerably in the amount of sweat lost during activity, because of both external factors (such as the exercise intensity and duration, environmental conditions, clothing) and individual factors (such as genetic makeup, heat acclimatization, initial hydration status, and training and health status). Thus, unfortunately, general recommendations about fluid ingestion are not feasible. Instead, it is recommended that individuals develop a personalized fluid replacement plan to ensure that dehydration does not account for more than 2% of their body weight during exercise (ACSM, 2007a). Careful monitoring of body weight following an exercise bout is necessary to determine an individual's typical sweat loss with exercise and the required fluid intake to offset this loss.

Thirst is an inadequate stimulus for adequately replacing the fluid lost during exercise. Even with unlimited access to water, most individuals do not voluntarily consume enough water to replace the water lost during physical activity, resulting in a relative state of dehydration. **Voluntary dehydration** refers to exercise-induced dehydration that develops despite an individual's unlimited access to water. **Figure 14.13** shows the sweat loss and voluntary fluid intake of men and women in various sports. Clearly, voluntary fluid intake does not fully replace sweat loss. Thus, the coach or exercise leader needs to encourage participants to consume fluid beyond what thirst dictates. Experts recommend that proper hydration be achieved by paying attention to prehydration, fluid ingestion during exercise, and rehydration following exercise ([ACSM, 2007a](#)). See the Focus on Application box for additional details.

Voluntary Dehydration Exercise-induced dehydration that develops despite an individual's access to unlimited water.

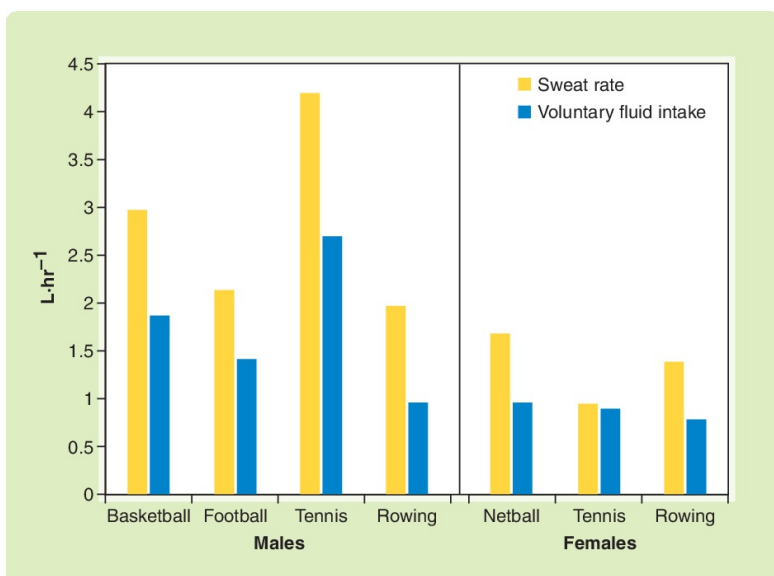


Figure 14.13 Sweating Rate and Voluntary Fluid Intake for Males and Females in Various Sports.

Source: Based on data in [American College of Sports Medicine \(2007a\)](#).

Hydration status is particularly important in sports where competition occurs by weight class and many athletes attempt to “make weight” by getting rid of fluid. Although the NCAA and many U.S. high school federations attempt to regulate weight throughout the season for wrestlers (see [Chapter 8](#)), this is not the case throughout the world, for all combat sport participants, nor for major events such as the Olympics. As a result, hypohydration is a problem. A recent study conducted on elite Swedish wrestlers, judokas, boxers, and taekwondo athletes found that 42% of both male and female participants (wrestling and taekwondo), despite weighing in the evening before had serious hypohydration the following morning—the morning of competition. Fully 50% of the judokas and boxers who weighed in the morning of competition were similarly seriously hypohydrated. Thus, neither a longer time for rehydration with an evening before weigh-in nor a shorter timed weigh-in prior to competition that was assumed would discourage extreme weight changes was able to prevent hypohydration before competition ([Pettersson and Berg, 2014](#)).

Fluid Ingestion during and after Exercise

During heavy exercise, metabolic heat production in active muscles increases up to 100 times that of inactive muscle. If this heat were not dissipated, the internal temperature would rise 1°C every 5–8 minutes, resulting in overheating (hyperthermia) and collapse of the individual within 15–20 minutes. Thus, sweating under these conditions is absolutely essential ([Nadel, 1988](#)). However, sweating does not occur without a price—the removal of fluid from the body ([Maughan, 1991](#)). A 70-kg, 2:30 marathoner can lose 5 L of body water, or 1–2 L·hr⁻¹. As previously described, sweat is not pure water but contains a variety of substances. The most important electrolytes are sodium (Na⁺), chloride (Cl⁻), and potassium (K⁺).

Although shifts in internal water compartments provide the

liquid portion of sweat, ultimately some of the water lost through sweating comes from blood plasma. If this water is not replaced, the individual may perform poorly and suffer from heat injuries. If fluid loss during exercise is adequately and appropriately replaced, cardiorespiratory variables can be maintained or undergo smaller disruptions from homeostasis ([Moreno et al., 2013](#)).

Type of Fluid Ingested

Whether to supplement with plain water or a carbohydrate and/or electrolyte beverage (sports drink) has been much debated. For the vast majority of sports or fitness workouts, plain water is the beverage of choice (**Figure 14.14**), but endurance events may be the exception ([American Dietetic Association, 1987](#); [Coyle, 2004](#)).



Figure 14.14 Water is an Appropriate Beverage to Replace Fluid Loss during Most Types of Physical Activity.

The rate at which any ingested fluid enters the body's water supply depends on the rate at which it leaves the stomach (*gastric*

emptying) and the rate at which it is absorbed across the intestinal membrane (*intestinal absorption*). Both gastric emptying and intestinal absorption are influenced by the composition of the ingested fluid.

Three factors affect gastric emptying. First, the gastric-emptying rate decreases as the caloric content of the ingested fluid increases. However, the differences in the gastric-emptying rate of solutions of 2.5–10% carbohydrate are negligible and are similar to that of plain water (Coleman, 1988; Maughan, 1991). Second, the rate of gastric emptying is exponentially related to the volume of fluid in the stomach (Maughan, 1991; Murray, 1987). That is, the amount of fluid emptied from the stomach is relatively large in the first minutes and then the rate slows down. Therefore, frequent ingestion of small amounts of fluid is preferable to infrequent ingestion of large amounts; 200–400 mL of fluid should be ingested every 15–30 minutes, because approximately 400 mL can be cleared in 15 minutes (American Dietetic Association, 1987; Nieman, 1990). Third, the temperature of the fluid may be relevant. Although it has been suggested that cold sports drinks enhance gastric emptying, the evidence suggests that the gastric-emptying rates of hot and cold beverages are the same (Maughan, 1991). The real advantage of a cold drink may simply be that it tastes better to a hot, sweaty athlete, thereby encouraging more drinking. In addition, a cold fluid does not add heat to the body, as a hot fluid would. Fluid temperatures of 15–22°C (59–72°F) are recommended (ACSM, 2007a).

FOCUS ON APPLICATION

Exercise and Fluid Replacement

The ACSM publishes a wide variety of Position Stands on issues related to Exercise, Physical Activity, and Health (go to Lippincott Connect for a complete list). These pronouncements written by members of the ACSM represent research-based consensus views from leading experts in the

area. The Position Stand titled “Exercise and Fluid Replacement” provides important recommendations about fluid and electrolyte requirements during physical activity and a review of research documenting the effect of hydration status on performance, physiology, and health. The accompanying tables summarize the goals and recommendations for prehydration, fluid intake during physical activity, and postexercise rehydration and estimate the approximate fluid intake needed to compensate for the sweating rate of individuals of different body sizes who are running at different speeds in different environmental conditions. It is recommended that changes in body weight be directly measured by individuals to personalize their fluid replacement regimes.

Please see the Position Stand in its entirety for additional recommendations on fluid and electrolyte replacement.

Approximate Fluid Intake (L) Needs Based on Body Weight and Running Speed in Cool and Hot Conditions

| Cool Temperature (18°C; 64.4°F) | | | |
|---------------------------------|---|---|---|
| Body Weight (kg) | 10 km·hr ⁻¹ (~6.3 mi·hr ⁻¹) | 12.5 km·hr ⁻¹ (~7.9 mi·hr ⁻¹) | 15 km·hr ⁻¹ (~9.5 mi·hr ⁻¹) |
| 50 | 0.53 | 0.69 | 0.86 |
| 70 | 0.79 | 1.02 | 1.25 |
| 90 | 1.04 | 1.34 | 1.64 |
| Warm temperature (28°C; 82.4°F) | | | |
| Body weight (kg) | 10 km·hr ⁻¹ (~6.3 mi·hr ⁻¹) | 12.5 km·hr ⁻¹ (~7.9 mi·hr ⁻¹) | 15 km·hr ⁻¹ (~9.5 mi·hr ⁻¹) |
| 50 | 0.62 | 0.79 | 0.96 |
| 70 | 0.89 | 1.12 | 1.36 |
| 90 | 1.15 | 1.46 | 1.76 |

Source: Reprinted with permission from American College of Sports Medicine (ACSM): Position stand: Exercise and fluid replacement. *Medicine & Science in Sports & Exercise*. 39(2):377–390 (2007). Copyright ©2007 The American College of Sports Medicine.

Fluid Intake Goals and Recommendations

| Time Period | Goal | Recommended Intake |
|-------------------------------------|--|---|
| Prephysical activity (prehydration) | Begin activity euhydrated—with normal electrolytes | If not already adequately hydrated drink ~5–7 mL·kg ⁻¹ BW 4 hr prior and, if still not hydrated, drink ~3–5 mL·kg ⁻¹ BW 2 hr prior to the event |
| During activity | Prevent excessive (>2% loss of body weight) dehydration and excessive change in electrolyte balance to avert compromised performance | Customize based on type of exercise, clothing, weather, and individual factors (genetics, acclimatization, training, and health status) (Use the table above to approximate necessary fluid intake) |
| Postphysical activity (rehydration) | Replace any fluid and electrolyte deficit | <ul style="list-style-type: none"> • If recovery time permits, this can be achieved with increased water and food containing enough Na⁺ to replace Na⁺ sweat loss • If recovery time is short, exercisers should ingest 1.5 L of fluid that includes electrolytes for each kg of body weight lost |

Intestinal absorption is not influenced by as many factors as gastric emptying. However, research has shown that glucose combined with sodium greatly increases intestinal fluid absorption compared to plain water. Thus, small percentages of glucose do not inhibit gastric emptying but enhance intestinal absorption if accompanied by sodium.

Although it has been claimed that electrolytes make sports drinks more palatable and hence encourage drinking, evidence indicates that sodium is the only physiologically beneficial electrolyte consumed during exercise (Maughan, 1991; Murray, 1987). Evidence does not indicate that matching the content of sweat—even if this were possible, considering its extreme variability—is necessary in replacement fluid (Haymes and Wells, 1986). Despite considerable attention given to potassium loss, its loss in sweat is low and of little consequence during exercise. The general consensus is that electrolyte losses will be replaced through normal food intake after exercise, with the possible exception of sodium. Sodium appears to be important in postexercise beverages for reasons other than intestinal absorption. After exercise, if drinks with little or no sodium are ingested, the plasma becomes diluted, stimulating urine production and fluid excretion. This also shuts off the thirst drive, delaying rehydration (ACSM, 2007a; Maughan, 1991; Nose et al., 1988). Once again, however, if some is good, more is not better. Salt tablets should never be ingested because highly concentrated amounts of salt can lead to GI discomfort, dehydration, and electrolyte loss (Steen, 1994).

Because most commercially available sports drinks (see Table 6.4 in Chapter 6) contain varying amounts of electrolytes other than sodium, some have wondered whether these other electrolytes may be harmful. Studies have not provided evidence one way or the other. However, no cases of problems arising from the ingestion of commercial sports drinks have been reported in the literature (Nose et al., 1988). One would assume, given the ongoing debate about such products, that such incidences would have been publicized had they occurred. It is important to note, however, that sports drinks differ significantly from “energy” drinks. Sports drinks are designed to aid in rehydration, whereas energy drinks are designed to increase alertness or prevent fatigue. Energy drinks should not be used to rehydrate. In fact,

caution is warranted in using energy drinks, which contain high levels of caffeine and other stimulants, before or during physical activity because of reports that these drinks can worsen heat stress and dehydration and are related to deaths in some cases (Seifert et al., 2011).

In situations where fluid replacement is more important than energy substrate supplementation (such as in a relatively short endurance event of 1–2 hours or an activity in high heat and humidity), the carbohydrate concentration of the sports drink should be low (2.5–8%) and the sodium content moderately high (30–110 mg). To put this amount of sodium in context, an 8-oz glass of 2% milk or tomato juice contains over 100 mg of sodium. Conversely, in those situations where substrate provision is needed (a long endurance event of over 2 hours in temperate environmental conditions), a more concentrated carbohydrate solution (6–10%) with sodium should be ingested (Maughan, 1991; Murray, 1987).

Exercise-Associated Hyponatremia (EAH)

While it is clear that dehydration can lead to impaired performance and serious health problems, it is also clear that excessive fluid ingestion can also be dangerous—even fatal. If large quantities of fluids are ingested in events lasting longer than 4 hours, hyponatremia (low sodium, sometimes called “water intoxication”) may occur. **Exercise-associated hyponatremia (EAH)** is the occurrence of hyponatremia during or up to 24 hours after prolonged physical activity; it is diagnosed by a plasma sodium concentration below normal values (usually $135 \text{ mmol}\cdot\text{L}^{-1}$) (Hew-Butler et al., 2008). Hyponatremia not only affects performance but can lead to brain damage and death. Death due to hyponatremia has been reported following marathons, ultraendurance events, and military training (Nolte et al., 2015). The early detection of EAH is important in initiating treatment and is associated with better outcomes. However, early recognition is often delayed because EAH has overlapping signs and symptoms with heat exhaustion and exertional heat stroke. The primary cause of hyponatremia during exercise is sustained overdrinking, in which fluid ingestion, particularly hypotonic fluids such as water, exceeds fluid loss through sweating and

urination. Elevated ambient temperatures, longer exercising times, and nausea are also associated with increased risk of EAH (Bennett et al., 2020). Some research has suggested that the use of nonsteroidal anti-inflammatory drugs increases the risk of developing EAH (ACSM, 2007a; Wharam et al., 2006) but this finding remains controversial. Thus, in ultraendurance events, athletes are urged to drink electrolyte beverages in addition to water and to avoid fluid consumption in excess of fluid loss.

Exercise-Associated Hyponatremia (EAH) The occurrence of hyponatremia during or up to 24 hours after prolonged physical activity; it is diagnosed by a plasma sodium concentration below normal values (usually $135 \text{ mmol}\cdot\text{L}^{-1}$).

Influence of Sex and Age on the Exercise Response in Heat

Male-Female Differences in Exercise Response in Heat

Most scientific evidence suggests that the response of individuals to exercise in a hot environment depends more on the state of their cardiovascular system and their hydration status than on their sex (Drinkwater, 1984; Stephenson and Kolka, 1988, 1993). A confounding factor, however, is the phase of the menstrual cycle. Core temperature is greater, at rest and during exercise, in the luteal phase (days 14–28) than in the follicular phase (days 1–13) of the menstrual cycle (Pivarnik et al., 1992; Stephenson and Kolka, 1993). Studies that have matched subjects for fitness level, body size, body fat, and degree of heat acclimatization have found few thermoregulatory differences between the sexes, particularly if the phase of the menstrual cycle was controlled for (Stephenson and Kolka, 1988). An attempt to map regional sweat rates and then predict whole body sweating rate found some regional differences in sweat rates between men and women but no statistical difference in predicted whole-body sweat rate (Baker et al., 2018).

There does appear to be a difference between the sexes in sweating response. Females generally sweat less and rely more on convection and conduction to dissipate heat than men. Males begin sweating at a lower core temperature and have a greater sweat rate and loss of electrolytes in humid conditions than similarly trained females (ACSM, 2007a; Avellini et al., 1980a; Drinkwater, 1984). Whether this difference is an asset or a liability to males is unknown. Some researchers have suggested that the higher sweat production of males is inefficient and wasteful and that females have the advantage of sweating less and thus decreasing blood volume to a lesser extent (Avellini et al., 1980b; Wyndham et al., 1965). Others counter that higher sweat rates might be advantageous in situations where evaporative heat loss is important, such as in a drier environment (Frye and Kamon, 1981). Although the sweating response is slightly different in males and females, heart rate and core temperature responses to exercise in the heat are similar (Frye and Kamon, 1983). Heat acclimatization also produces similar results in both sexes (Frye and Kamon, 1983).

Exercise Response of Children and Adolescents in the Heat

Thermoregulatory responses are different between adults and children. Heat is produced by active muscle mass but dissipated by exposed surface area. Despite a lower muscle mass, children produce more metabolic heat during the same amount of weight-bearing exercise (walking, running) than adults. On the other hand, the surface area-to-mass ratio is greater in children than in adults. This progressively decreases during growth and maturation until the adolescent reaches adult size. Therefore, in thermoneutral or moderate heat situations, the higher metabolic heat production of children is compensated for, to a large extent, by a relatively larger body surface area over which to dissipate this heat by dry heat exchange (radiation, convection, conduction). Thus, children rely less on evaporative cooling than adults. However, in high heat situations, this variation becomes a liability. Not only does the higher metabolic heat production continue but the body absorbs heat from the surroundings over

this larger surface area, and evaporative cooling may not be able to sufficiently compensate (Falk, 1998).

With high ambient temperatures, sweat evaporation is the main process for heat dissipation, and this requires the involvement of sweat glands. Children have the same total number of sweat glands as adults by the age of 2 or 3 years, and thus, children have a higher number of sweat glands per unit of area than adolescents or adults. However, sweat gland size and sensitivity appear directly related to age. Sensitivity is lower in children, meaning that the threshold at which sweating begins is higher; it takes longer for sweating to start and the rate of sweating may be different in children and adolescents than in adults. Sweat rate is the best indicator for evaporative heat loss. Sweat rates are lower (by as much as 40%) in boys than in young adult males. The sweat rate increases progressively in young males both per gland and per surface area as they progress from prepubertal to midpubertal and late pubertal stages of maturation. Girls appear to sweat less than boys, but most studies have not found any difference between prepubertal girls and adult females in sweat rate (Falk, 1998; Meyer et al., 1992; Rowland, 2005).

The effectiveness of thermoregulation is typically judged by the stability of core temperature. In children, the body can prevent a detrimental rise in core temperature with exercise in the heat as well as adults under moderate conditions. However, when the ambient temperature is very high or exceeds skin temperature by 5–10°C, symptoms of fatigue and intolerance are more common in children than in adults (Rowland, 2005).

There is no clear evidence that exercise affects the level of dehydration or circulatory function more in children than in adults. The size of voluntary dehydration does not appear to differ across the age span (Rowland, 2005), although voluntary dehydration is typically not a problem in activities lasting less than 45 minutes for children. However, the same degree of dehydration, corrected for body weight, results in a greater core temperature rise in children than adults during exercise in the heat. The levels of sodium and chloride in children's sweat during exercise in a hot environment are lower, and the level of potassium is higher, compared with adults. However, these

differences do not require most currently available sports drinks to be diluted for children. Evidence suggests that adding flavoring and enriching water with sodium chloride (NaCl) and carbohydrates (as in sports drinks) increases voluntary ingestion in children and adolescents and, when freely available, can prevent dehydration ([Falk, 1998](#); [Kenney and Chiu, 2001](#)).

Aerobic training improves heat tolerance and thermoregulatory effectiveness in adults but has only a minor effect in children ([Bar-Or and Rowland, 2004](#)). However, children, adolescents, and adults all appear to successfully acclimatize to heat. The main difference is the rate of acclimatization, although the time period needed for acclimatization may not vary. For example, at the end of 2 weeks, both children and adults may have acclimatized, but throughout the process, adult adaptations (sweat rate, core temperatures, etc.) are ahead of the children's ([Falk, 1998](#)). Children should undergo a long and gradual acclimatization program to ensure that they are physiologically prepared for exercise in the heat ([Rowland, 1990](#)). Children subjectively feel acclimatized before physiological acclimatization has occurred, and thus, they are likely to try to do too much too soon ([Falk, 1998](#)).

Exertional heat stroke is one of the leading causes of sudden death in sports and requires special attention by exercise professionals who have responsibilities for children. In many cases, secondary schools do not have as many resources devoted to the prevention and care of exertional heat stress. Many experts recommend environmental monitoring, appropriate guidelines for activity, and ability to rapidly cool athletes competing at the secondary school level to ensure the safety of these athletes ([Adams, 2019](#); [Hosokawa et al., 2021](#)).

Exercise Response of Older Adults in the Heat

Older individuals have a lower sweat rate than younger adults during environmental heat stress leading to a reduction in evaporative heat loss ([Balmann et al., 2018](#)). Older individuals are also more likely to have health issues that impair thermoregulatory responses. Thus, heat strain is of concern for

older individuals both at rest, particularly with the increasing number and severity of heat waves, and during exercise. Cardiac output increases above resting levels during exercise in the heat for both young and older individuals. However, at higher workloads, the increase in cardiac output is not as great when exercise is performed in the heat as when it is performed under thermoneutral conditions (see **Figure 14.10**). The increase in cardiac output during exercise in a hot environment is less in older persons than in younger individuals of similar fitness levels (Kenney and Anderson, 1988). Furthermore, it appears that young adults increase cardiac output by augmenting heart rate, but older individuals increase cardiac output by increasing stroke volume (Kenney and Anderson, 1988). This helps explain why differences exist in cardiac output at higher workloads; apparently, there is a limit to the extent to which older individuals can increase stroke volume.

FOCUS ON RESEARCH

Heat Dissipation and Age

The two primary physiological mechanisms to dissipate heat are an increased sweat rate and an increased skin blood flow. It has long been known that the ability to dissipate heat during exercise in hot environments declines with increasing age. However, for many years, it was unclear what physiological mechanisms caused the alterations in sweat rate and skin blood flow that occur as a person ages. The obvious way to explore an answer to this question is to design a study that compares younger adults with their older counterparts. However, one inherent difficulty in such studies is that older adults typically have a lower $\dot{V}O_2 \text{ max}$. It is known that an increase in $\dot{V}O_2 \text{ max}$ is associated with an earlier onset of sweating, presumably because in a trained individual, the body has adapted to maintain thermal balance more effectively. Thus, it has been unclear whether aging itself or the age-related decline in $\dot{V}O_2 \text{ max}$ is responsible for the

decreased sweating and reduced heat loss in older exercisers.

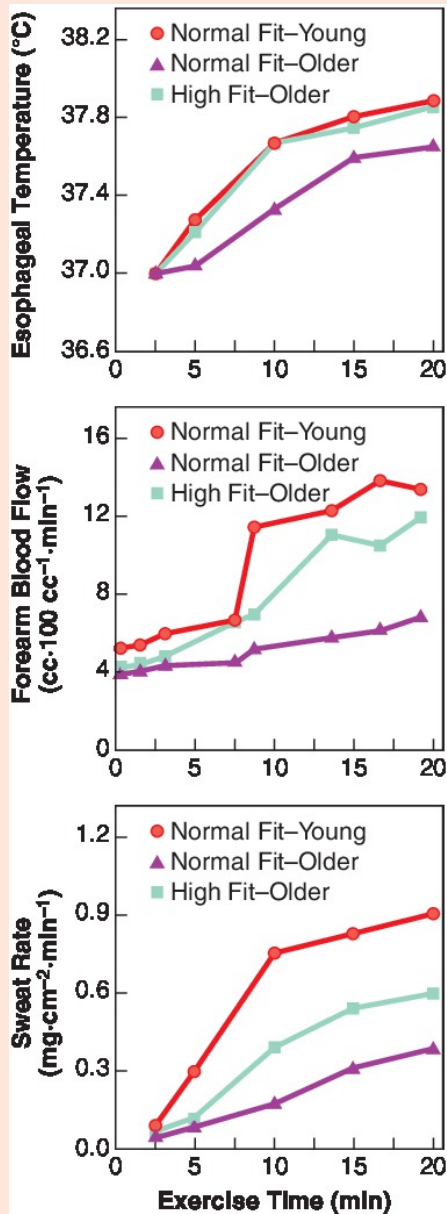
To overcome these difficulties, Tankersley et al., 1991 designed a study that compared young normally fit individuals to a group of older normally fit individuals and to a group of older highly fit individuals during 20 minutes of submaximal exercise (67.5% of $\dot{V}O_2 \text{ max}$). Thus, the younger group could be compared to an older group with a lower $\dot{V}O_2 \text{ max}$ (the normally fit older group) and an older group with a similar $\dot{V}O_2 \text{ max}$ (the highly fit older group). The accompanying graphs present the esophageal temperature, chest sweating rate, and forearm blood flow in the three groups.

The graphs indicate the following:

1. Esophageal temperature during the exercise was not different among the three groups working at the same relative workload. However, because the normally fit older group had the lowest $\dot{V}O_2 \text{ max}$, they were working at a lower absolute workload and thus should be producing less metabolic heat. Therefore, the fact that the increased temperature among the three groups was similar may indicate an impaired ability to dissipate heat in the normally fit older group compared to the two groups with a higher $\dot{V}O_2 \text{ max}$.
2. In general, the average forearm blood flow and chest sweating rates for the highly fit older individuals were intermediate to those in the normally fit older group and the normally fit younger group.
3. The normally fit older individuals had a lower forearm blood flow and lower sweating response during most of the exercise protocol compared to the normally fit younger individuals.

These results suggest that younger and older subjects matched for similar $\dot{V}O_2 \text{ max}$ have similar heat loss responses to submaximal exercise. Therefore, it appears that

the decreased $\dot{V}O_2 \text{ max}$ that typically accompanies aging is primarily responsible for the changes in sweating and blood flow reported in older adults. A person who maintains a high fitness level as he or she ages is not likely to experience these detrimental changes in the thermoregulatory response to aging.



Source: Reprinted with permission from Tankersley, C. G., J. Smolander, W. L. Kenney, & S. M. Fortney: Sweating and skin blood flow during exercise: The effects of age and maximal oxygen uptake. *Journal of Applied Physiology*. 71(1): 236–242

Exercise-induced reductions in plasma volume are greater in older individuals than in young individuals when they exercise in hot and humid conditions. When exercising at the same relative workload, young and older individuals demonstrate similar changes in systolic, diastolic, and mean arterial blood pressure. The accompanying Focus on Research box explores the influence of age and fitness level on thermoregulatory (temperature, forearm blood flow, and sweating) responses to exercise. As noted in the Focus on Research box, there is no difference in core temperature between young and healthy older individuals when they exercise in the heat when the individuals are matched for fitness level. Furthermore, acclimatization to heat results in similar changes in healthy older individuals and in young people ([Best et al., 2014](#); [Pandolf et al., 1988](#)).

It has been shown that whole body heat loss is significantly reduced in older versus younger individuals when doing physical activity in the heat. This impairment in heat dissipation is evident as early as age 40 years ([Kenny et al., 2015](#); [Larose et al., 2013](#)). See the Focus on Research box for a discussion of this exercise response.

Heat Illness

The magnitude of cardiovascular stress placed on the body by exercise in the heat was addressed in compelling language by Loring Rowell over 30 years ago:

Probably the greatest stress ever imposed on the human cardiovascular system (except for hemorrhage) is the combination of exercise and hyperthermia. Together, these stresses can present life-threatening challenges, especially in highly motivated athletes who drive themselves to extremes in hot environments ([Rowell, 1986](#)).

When the cardiovascular system cannot meet the

thermoregulatory and metabolic demands of the body, heat illness ensues. **Exertional heat illness (EHI)** involves a range of disorders resulting specifically from the combined stresses of exertion and heat stress. This is the type of heat illness that is most likely to be encountered by exercise professionals. EHI can be defined as a range of multisystem illnesses related to elevated body core temperature (hyperthermia) and the cardiovascular and metabolic responses that result from exercise and the body's attempts at thermoregulation. EHI varies greatly in severity and includes exertional dehydration, heat cramps, heat exhaustion, heat injury, and heatstroke. One of the greatest challenges of EHI is that it is often difficult or impossible to distinguish among these disorders because they often overlap and frequently evolve into a different form over time ([Gardner and Kark, 2001](#)).

Exertional Heat Illness (EHI) A range of multisystem illnesses related to elevated body core temperature and the cardiovascular and metabolic processes that result from exercise and the body's thermoregulatory response.

EHI syndromes involve the physiological disruption of several organs or systems and can vary in severity. **Figure 14.15** depicts the major EHI syndromes in terms of physiological disruption and severity. Within each category along the EHI range (minor to serious), there can be different levels of physiological disruption, and each illness may differ in severity and consequence. Furthermore, the shading within the figure reinforces the considerable overlap among the EHS syndromes and suggests that clear distinctions among them are not always possible ([Gardner and Kark, 2001](#)). Although clinicians have long sought to categorize heat illnesses in discrete categories and have attempted to use core temperature as a way to define thresholds for defining categories, current thought is that such categorization is not possible and that body temperature alone does not accurately reveal the severity of heat illness.

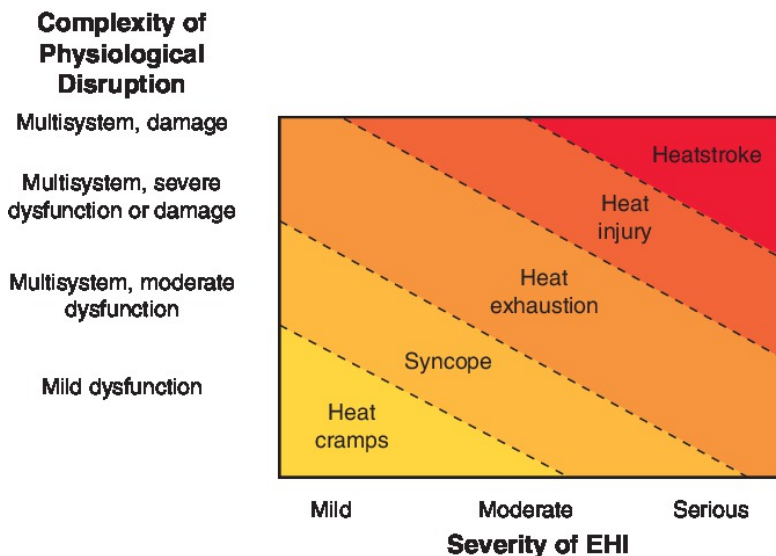
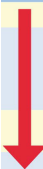


Figure 14.15 Spectrum of EHIs.

Minor Exertional Heat Illness

Minor EHI includes mild dehydration, heat cramps, and heat syncope ([Carter et al., 2006](#); [Gardner and Kark, 2001](#)). Symptoms of heat stress may be nonspecific, particularly in the early stages of heat illness. As heat illness progresses, so does the severity of orthostatic signs and symptoms ([Table 14.2](#)).

TABLE 14.2 Symptoms of Exertional Heat Illness (EHI)

| Nonspecific Symptoms | | | |
|---|-----------------|--|--|
| Thirst | Weakness | Headache | Nausea |
| Hyperventilation | Fatigue | Poor concentration | Vomiting |
| Tachycardia | Cramps | Impaired judgment | Diarrhea |
| | | Anxiety | |
| | | Hysteria | |
| Progressive Orthostatic Signs and Symptoms | | | |
| Severity | Sign or Symptom | | Action |
|  | Mild | Faintness | Cool body, rehydrate |
| | | Dizziness | |
| | | Wobbly legs | |
| | | Stumbling gait | |
| | Moderate | Blurred vision, tunnel vision, blackout | Seek medical help |
| | Severe | Collapse (without loss of consciousness) | Seek medical help Medical emergency |
| | | Collapse—brief (<3 min), loss of consciousness | |
| | | Sustained hypotension | |
| | | Shock, cardiovascular collapse | |

Heat cramps are an acute disorder consisting of brief, recurrent, and excruciating pain in the voluntary muscles of the legs, arms, or abdomen. Typically, the muscles have recently been engaged in intense physical activity and are fatigued. Heat cramps may result from a fluid-electrolyte imbalance. Profuse sweaters who lose large quantities of sodium may be more susceptible than others. Skeletal muscle heat cramps frequently occur in football and tennis players competing in late summer or early fall heat and humidity (ACSM, 2007a, 2007b), but other athletes are not immune. A combination of rest, stretching, and ingestion of fluids or foods containing NaCl typically leads to recovery. *Heat syncope* (fainting) is characterized by vertigo (dizziness) and weakness during or following standing or with a rapid change to the upright posture during heat stress (Carter et al., 2006). Syncope is often related to low blood pressure, which is corrected as the person becomes supine. Of course, fainting can also result in injury if the person hits their head as they fall. Syncope can also be indicative of more serious heat-related injuries.

Serious Exertional Heat Illnesses

Serious EHIs include heat exhaustion, heat injury, and heatstroke (Carter et al., 2006). **Exertional heat exhaustion** is characterized by an inability to maintain adequate cardiac output and moderate (38.5°C; 101.3°F) to high (>40°C; 104°F) body temperatures. The person experiences a rapid and weak pulse, fatigue, weakness, profuse sweating, psychological disorientation,

and fainting. Heat exhaustion is caused by an acute fluid loss and the inability of the cardiovascular system to adequately compensate for the concurrent demands of muscle and skin blood flow.

Exertional Heat Exhaustion A moderate illness characterized by an inability to maintain adequate cardiac output at moderate (38.5°C) to high (> 40°C) body temperatures.

Children may be more susceptible to heat exhaustion because they have a smaller plasma pool from which to draw fluid. Thus, they have a potential greater deficiency of peripheral blood supply during strenuous activity in the heat than adults (Zwiren, 1992).

Individuals suffering from heat exhaustion should be moved to a cool place, given fluids, and encouraged to lie down. In severe cases of heat exhaustion, a person may require medical intervention including the intravenous administration of fluids and electrolytes.

Exertional heat injury is a moderate to severe progressive multisystem disorder, with hyperthermia accompanied by organ damage or severe dysfunction. Muscle cell breakdown (rhabdomyolysis) occurs when heat decomposes the muscle cell membrane, which in turn may cause liver and kidney damages (Carter et al., 2006; Gardner and Kark, 2001). Rhabdomyolysis is associated with and possibly caused by excessive repetitive exercise, especially eccentric exercise. Heat stress and dehydration exacerbate this condition (Szczepanik et al., 2014). Exertional heat injury is more severe than heat exhaustion but less severe than heatstroke. Exertional heat injury may progress into heatstroke, however.

Exertional Heat Injury A moderate to severe progressive multisystem disorder, with hyperthermia accompanied by organ damage or severe dysfunction.

Exertional heatstroke is a life-threatening illness

characterized by central nervous system dysfunction (Carter et al., 2006; Leon and Helwig, 2010) and has become the third leading cause of death in high school athletes (Allen and Cross, 2014). Heatstroke is a progressive multisystem disease that can lead to death due to multiorgan failure (Leon and Helwig, 2010). It often involves severe muscle and liver cell deaths, cardiovascular collapse, and seizures (Gardner and Kark, 2001). It can be accompanied by elevated skin and core temperatures in excess of 40.5°C, tachycardia (rapid heart rate), vomiting, diarrhea, hallucinations, and coma. Heatstroke involves a failure of the thermoregulatory mechanisms. If heatstroke is suspected, the individual should be cooled as quickly as possible (preferably by cold water immersion) and medical personnel notified immediately. Rapid cooling is critical to limiting the amount of time that individuals have a critically elevated body temperature (Armstrong et al., 2007). Cooling should be initiated quickly and not delayed for transport. Unfortunately, a study found that although athletic trainers know that cold water immersion is the most effective way to treat this condition, only about half (49.7%) of those surveyed have adopted this method of cooling for their cooling treatment in cases of suspected heatstroke (Mazerolle et al., 2010).

Exertional Heatstroke A life-threatening illness characterized by high body temperature and central nervous system dysfunction.

Complete the [Check Your Comprehension 1—Case Study 1](#) to determine your understanding of exertional heat illness.

FOCUS ON APPLICATION

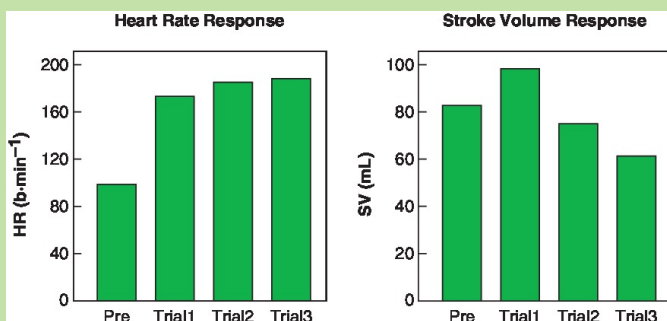
Working under Environmental Extremes

Many occupational workers, such as construction workers, miners, and hazardous material crews, are routinely required

to perform muscular work under extreme environmental conditions. Probably no workers are exposed to higher thermal environments than are firefighters. Firefighters produce large amounts of metabolic heat and are often exposed to very high temperatures. Firefighters wear vapor-impermeable gear weighing approximately 20 kg and perform heavy muscular work, often in temperatures ranging from 100 to 400°C. Thus, it is not surprising that firefighters experience severe cardiac strain. In fact, the leading cause of death in the line of duty among firefighters is myocardial infarction. It accounts for approximately 50% of the line-of-duty deaths each year—far more than deaths due to burn injuries (<10% in most years).

To better understand the magnitude of the cardiovascular stress, [Smith et al. \(2001\)](#) had firefighters perform three trials of a standardized set of firefighting tasks in a training building that contained live fires. Each set of drills took approximately 7 minutes to complete. A 10-minute rest period was provided between the second and third trials, during which firefighters removed their helmets, face masks, and coats in an attempt to cool the body and were strongly encouraged to consume cold water. As seen in the accompanying graphs, their heart rates increased quickly during the first trial and reached age-predicted maximal values by the end of the third trial. Stroke volume increased following the first trial but was significantly below resting values by the end of the third trial. Stroke volume was lower following several minutes of firefighting due to profuse sweating and vasodilation of cutaneous vessels. This occurred despite the attempt to cool the body and replace fluid during the 10-minute rest period.

Because of the severe cardiovascular strain associated with firefighting and the risk of myocardial infarction, many fire departments are initiating fitness programs for their personnel. Aerobic fitness programs help lessen the cardiovascular strain associated with firefighting by increasing plasma volume and improving heart function.



Source: [Smith et al. \(2001\)](#).

CHECK YOUR COMPREHENSION 1—CASE STUDY

Martha is a 34-year-old mother of three who resides in Pennsylvania. Martha signed up for half marathon in June in Florida so that her children could travel with her and enjoy a trip to Disney. Race day was unseasonably warm (85°F). Martha completed the run in 2 hours and 11 minutes, although she had been finishing her training runs in less than 2 hours. As she was looking for her running partners in the postrace, she felt an unaccustomed weakness, stumbled several times, and had difficulty remember details such as the planned meeting

place. What was the likely cause of these symptoms? What steps should be taken to alleviate these symptoms?

Check your answer in Appendix C.

Prevention of Exertional Heat Illness

Although exercise professionals must be able to recognize and respond to heat illness, preferably they should prevent heat injuries by using sound judgment and observing basic recommendations. The ACSM position stand entitled “The Prevention of Thermal Injuries During Distance Running” (1987) outlines strategies to decrease thermal injuries during road races. Following are basic recommendations for people who exercise in hot environments:

1. Allow adequate time for acclimatization (10–14 days).
2. Exercise during cooler parts of the day (early morning or evening).
3. Limit or defer exercise if the heat index is in the high-risk zone (see **Figure 14.1**).
4. Use appropriate periodization and progression.
5. Adequately hydrate before exercise and replace fluid loss during exercise. Monitor daily body weight changes closely, because they reflect acute water loss.
6. Wear light-colored, loose-fitting clothing that exposes large areas of skin to the air to enhance evaporation.

Complete the [Check your Comprehension 2](#) scenario to test your understanding strategies to prevent heat illnesses.

CHECK YOUR COMPREHENSION 2

Coach Brown, a high school lacrosse coach, is preparing for the start of the fall season in Charleston, SC. When practice begins in late August, the coach expects a mix of returning players and new players trying out for the team.

What environmental and physical factors should he consider

in relation to heat stress on his players?

What individual characteristics should he consider in relation to heat stress on his players?

What can he do to lessen the risk of heat illness among his players?

Check your answer in Appendix C.

Exercise in the Cold

Cold weather can cause significant injuries to individuals who are unprepared or inadequately equipped for exercise training or competition. Although cold-related injuries are less common than heat-related injuries, they can be serious and even life threatening. Most commonly, exercise in the cold involves sporting events or wilderness experiences. Sporting events include winter sports, such as skiing and ice skating, and athletic contests, such as football. In these activities, hypothermia (low body temperature) is typically not a threat because individuals usually wear proper clothing, produce much metabolic heat, and have access to shelter when necessary. Wilderness activities, such as hiking and backpacking, present more risk because exposure can be prolonged (**Figure 14.16**).



Figure 14.16 Prolonged Activities in Cold Environments Can Pose a Risk of Hypothermia or Frost Bite.

Exposure to cold results in several physiological responses that alter thermal balance. Heat production increases, and heat loss is minimized by vasoconstriction. Heat production increases through nonshivering thermogenesis, shivering thermogenesis, and exercise metabolism. *Nonshivering thermogenesis* refers to increased metabolic heat production other than through muscular contraction. Circulating hormones—catecholamines, glucocorticoids, and thyroxine—increase metabolic rate during cold exposure (Toner and McArdle, 1988). Muscular tensing without noticeable shivering accounts for over 30% of the increase in heat production in response to cold exposure (Toner and McArdle, 1988). This response is sometimes called preshivering. Shivering can also contribute significantly to heat production. Finally, although it varies with intensity and duration, exercise can be the greatest contributor to increased metabolic heat in the cold. Conversely, cool temperatures are often useful for dissipating the large amount of heat produced by exercise.

Very often, individuals who are exposed to the cold will voluntarily increase muscular activity as a means to increase heat production and thus feel more comfortable. Heat loss is

minimized by widespread vasoconstriction, which decreases blood flow to the periphery in an attempt to maintain core temperature.

Despite the compensatory mechanisms, in some situations, being in a cold environment leads to heat loss that exceeds heat production, and body temperature drops. When an individual is exposed to the cold and heat loss is greater than heat production, serious injuries can result. The two most common cold-induced injuries of concern to exercise professionals are hypothermia and frostbite.

Cold-Induced Injuries

Hypothermia, defined as a core temperature less than 35°C (95°F), is a lowering of the body temperature to the point that it affects normal function (Bar-Or and Baranowski, 1994). This condition is potentially fatal. Body temperature drops when heat loss exceeds heat production. The magnitude of heat loss is affected by temperature, wind speed, and whether the individual becomes wet. The windchill chart (Table 14.1) takes into account the combined effects of temperature and wind speed and should be consulted before activity in the cold.

Hypothermia A core temperature less than 35°C (95°F), resulting in the loss of normal function.

Because heat production increases greatly during exercise, hypothermia is seldom a concern for an exerciser who is properly clothed and not exposed to the environment for a prolonged time. However, during long-term events, such as a marathon, hypothermia may occur as a result of reduced heat production and increased heat loss. For instance, many runners run the second half of a race at a slower pace, resulting in less heat production. At this time, the runner may also have removed some clothing because of greater heat production earlier in the run and may be wet due to accumulated sweat, thus increasing the rate of conductive and evaporative heat loss.

As body temperature decreases, physical signs of hypothermia

can be observed. Early signs include depressed heart rate, respiration, and reflexes. Mild hypothermia is marked by a loss of judgment and reduced ability to reason. The person often complains of being cold and is focused on getting warm. As hypothermia progresses, fine motor skills are affected and speech may become slurred. Severe hypothermia is characterized by agitation and inappropriate behavior and may progress to unconsciousness and coma. Mild hypothermia can be managed by warming the individual with blankets and warm beverages. However, moderate and severe cases of hypothermia should be treated by medical personnel. It is important to replace wet clothing and to protect a person with hypothermia from wind so that additional heat from the body is not lost. Because a person suffering from hypothermia often has an impaired ability to reason, the person should not be left alone.

Frostbite results from water crystallization within tissues that causes cellular dehydration and leads to tissue destruction. Frostbite usually occurs in insufficiently insulated skin. Severe frostbite can cause permanent circulatory damage, potentially leading to a need for amputation. Moving quickly (such as when running, skiing, and cycling) can create a windchill condition that increases the likelihood of frostbite.

Prevention of Cold-Induced Injuries

Injury prevention is preferable to injury treatment. To avoid cold injuries, individuals should exercise in appropriate clothing. Windproof and water-repellent outer garments are necessary. Layering loose-fitting clothing is more advantageous than wearing a single layer of heavy, bulky clothes. Clothing next to the skin should be composed of a material that wicks moisture away from the skin. Cotton should not be worn next to the skin because it holds moisture, which will draw heat away from the body.

Exercisers should consider warming up indoors, but only to the point at which they begin to sweat, so that they are not wet when they go outside. If possible, runners should run against the wind first and return with the wind at their back. Exercisers

should also avoid periods of decreased metabolic heat production unless they can compensate by adding clothing or leaving the cold environment. The cooldown period at the end of exercise is potentially dangerous because heat production has decreased although heat loss remains high. Additionally, the exerciser may be fatigued, which further exacerbates the condition. The exerciser should ideally cool down inside also. Exercising in cold environments, not just hot ones, results in water losses. There appears to be a blunted thirst response, so attention should be paid to fluid intake ([Mears and Shirreffs, 2014](#)).

Influence of Sex and Age on Cold Tolerance

Male-Female Differences

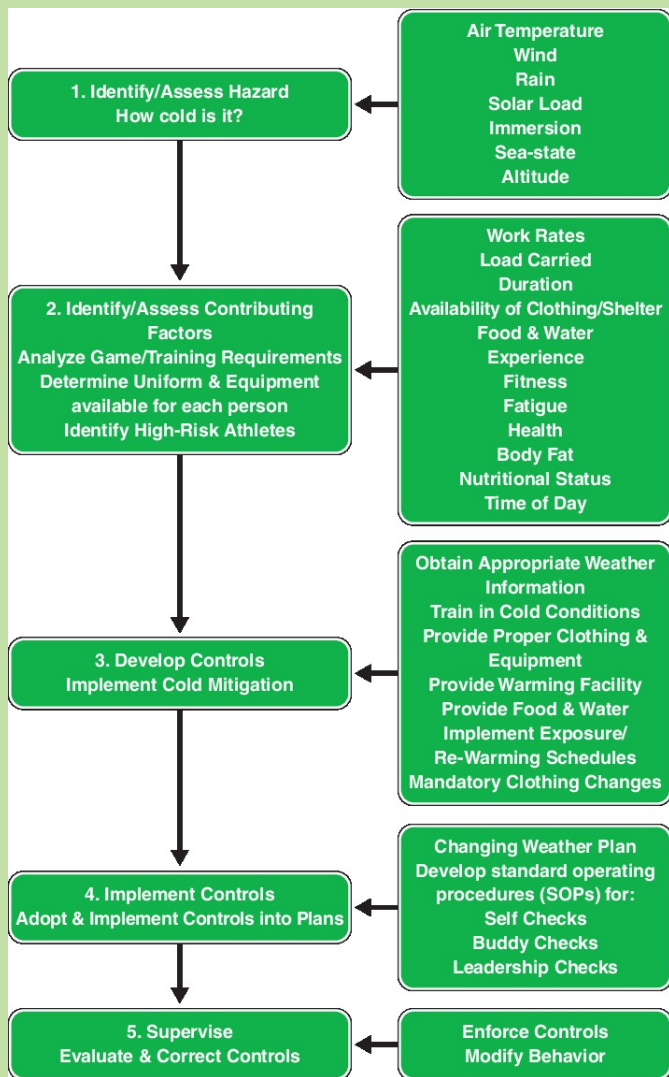
Body size and composition are important influences on an individual's response to cold exposure. Body fat protects against heat loss and can thus be advantageous in the cold ([Toner and McArdle, 1988](#)). Because women on average have a greater percentage of body fat than men, they may tolerate cold better.

FOCUS ON APPLICATION

Preventing Cold Injuries during Exercise

The American College of Sports Medicine has published a Position Statement titled "Prevention of Cold Injuries during Exercise" to increase the safety of individuals performing work or exercise in cold environments. The adjacent flowchart provides an overall strategy for identifying and managing potential threats to performance and safety in cold conditions.

Please see the Position Stand in its entirety for additional recommendations for preventing cold injuries during exercise.



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Children and Adolescents

Because children have a larger surface area per kilogram of body weight, they lose heat faster in the cold than adults (Falk, 1998). As compensation, however, peripheral vasoconstriction occurs to a greater extent in children to protect core temperature. This reduced blood flow lowers skin temperature, however, and increases the risk of frostbite in the extremities (Bar-Or and Baranowski, 1994). Children also cool faster in water than adults because of their relatively larger surface area and smaller amount of subcutaneous fat. Special attention should be given, therefore, when children are swimming on cool days or when water temperatures are cool.

Older Adults

Older adults have a greater risk of hypothermia due to blunted physiological and behavioral responses to the cold. In older adults, vasoconstriction to conserve heat is reduced from that in young adults. Because older adults also have a lower exercise capacity, they will become fatigued earlier when performing the same absolute workload. An older person is more likely to produce less heat (due to fatigue and discontinuing exercise) and may be less able to limit heat loss (due to reduced vasoconstriction), placing them at greater risk for hypothermia. Evidence suggests that older adults often have a blunted sensitivity to cold, such that they may be slower to make appropriate behavioral changes in a cold environment (ACSM, 2006).

Summary

1. Environmental conditions that affect human thermoregulation are ambient temperature (T_{amb}), relative humidity, and wind speed. The heat index assesses the risk of thermal injury from measures of ambient temperature and relative humidity. The windchill index assesses the risk of cold-induced injury from wind speed and ambient temperature.

2. Body temperature results from a balance of heat gain and heat loss. Most heat gain results from the body's metabolic heat production. Heat can be lost from the body through radiation, conduction, convection, and evaporation.
3. Sweat evaporation is the primary defense against heat stress. For each liter of sweat vaporized, 580 kcal of heat energy is released.
4. The extent of heat exchange depends on the thermal gradient, relative humidity, air movement, degree of direct sunlight, and clothing worn.
4. Several interrelated problems challenge the cardiovascular system under hyperthermic conditions.
 - a. The skin and the muscles compete for blood flow.
 - b. Vasodilation in the cutaneous vessels effectively decreases venous return and thus stroke volume.
 - c. Adequate blood pressure must be maintained to perfuse the vital organs.
 - e. Plasma volume is reduced, contributing to reduced cardiac output.
6. An individual's response to exercise in the heat is influenced by the degree of acclimatization, cardiovascular fitness, body composition, and hydration level.
7. Water balance is generally well maintained, but athletes need to pay particular attention to hydration before, during, and after exercise in the heat.
8. The response of males and females to exercise in a hot environment is similar, although males appear to have a greater sweat rate.
9. In children and adolescents, thermoregulation is slightly different from that in adults, and they may be at greater risk of heat stress when exercising in hot environments.
10. When the cardiovascular system cannot meet the body's thermoregulatory and metabolic demands, heat illness ensues. Heat illness is a range of disorders that vary in intensity, complexity, and severity. Exertional heat illnesses include heat exhaustion, heat injury, and heatstroke.
11. During exercise in the cold, heat loss may exceed heat

production, potentially resulting in serious injuries: hypothermia and frostbite.

Review Questions

1. Diagram the thermal balance that is typically maintained at rest. Indicate how this balance is altered during exercise in hot and cold environments.
2. Identify the factors that influence heat exchange, and discuss how each facilitates or impedes the transfer of heat to and from the body.
3. Describe the cardiovascular responses to incremental exercise in a hot environment compared to a thermoneutral environment, and explain why these occur.
4. What is the importance of acclimatization? How much time is needed for acclimatization to occur?
5. Explain the influence of hydration level on an individual's response to exercise in the heat. What is necessary to maintain adequate hydration?
6. Discuss the effects of fitness level and body composition on an individual's response to exercise in the heat.
7. Provide a definition, cause, and treatment for exertional heat cramps, heat syncope, heat exhaustion, heat injury, and heatstroke.
8. Identify ways to minimize the risk of heat illness.
9. Explain the underlying cause of hypothermia and frostbite. Suggest ways in which these conditions can be prevented.

Literature Search

1. Exertional heat illnesses represent a serious risk when exercise is performed in the heat. To better understand the risks associated with this important issue and what can be done to mitigate the risk, do a literature search using a search engine such as PubMed, Google scholar, or Web of

Science.

- a. Do a search for “Exertional Heat Stress.” This search will yield many articles. Note how many of them are related to military operations, occupational work, and to sport and athletic participation.
- b. Refine your search using key terms that may reflect your interest in this area. You may be interested in a specific type of heat illness, a particular population, or in the pathophysiology or treatment of heat-related illness. For example,
 - i. Treatment for exertional heat stroke
 - ii. Prevention of exertional heat exhaustion in construction workers
 - iii. Prevention and management of heat stroke in secondary school athletics

For further review and study tools, visit Lippincott Connect.

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15 Cardiovascular Disease Risk Factors and Physical Activity



Chapter Outline

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OBJECTIVES

After studying the chapter, you should be able to:

- Discuss the prevalence of cardiovascular disease and its impact in terms of mortality and economic costs.
- Describe the progression of atherosclerosis and indicate how atherosclerosis can lead to a myocardial infarction (heart attack).
- Identify the cardiovascular risk factors and classify them as risk factors that cannot be modified, major modifiable risk factors, or contributing (nontraditional) risk factors.
- Describe the relationship between each risk factor and cardiovascular disease in terms of the degree of risk and the underlying pathology.
- Identify how exercise/physical activity, exercise training, and physical fitness impact each of the risk factors.

- Track the cardiovascular risk factors from childhood to adulthood.

Introduction

Earlier in this unit, a distinction was made between the application of the training principles for the achievement of health and for fitness. The health factor of primary concern in this chapter is cardiovascular disease (CVD). Cardiovascular disease (CVD) affects an estimated 85 million Americans (one in three) and has been the leading cause of death in the United States every year since 1900, except 1918. Cardiovascular disease is the underlying cause of over 30% of all deaths in the United States, which is more than cancer and chronic lung disease combined (Virani et al., 2021). Worldwide, cardiovascular disease is also increasing as a cause of death and disability.

Several terms have a very specific meaning in regard to statistics about disease. **Prevalence** refers to the number of cases of a disease in a specific population at a given time. **Incidence** is the rate of new cases of a disease in a specific population. **Mortality** is the number of deaths in a population. **Morbidity** refers to the number of people with a sickness or disease in a population.

Prevalence The number of cases of a disease in a specific population at a given time.

Incidence The rate of new cases of a disease in a specific population.

Mortality The number of deaths in a population.

Morbidity The number of people with a sickness or disease in a population.

Figure 15.1 depicts the total number of CVD deaths for men and women in the United States from 1980–2020 (Virani et al., 2021). CVD is a broad category of diseases affecting the heart and the vasculature. Coronary heart disease (arteriosclerosis in coronary arteries) continues to account for the highest percentage of total CVD deaths (42%). Stroke is responsible for approximately 17% of CVD deaths, high blood pressure for 11% of deaths, heart failure about 10% of deaths, and a broad array of other cardiovascular causes account for the remaining 20%.

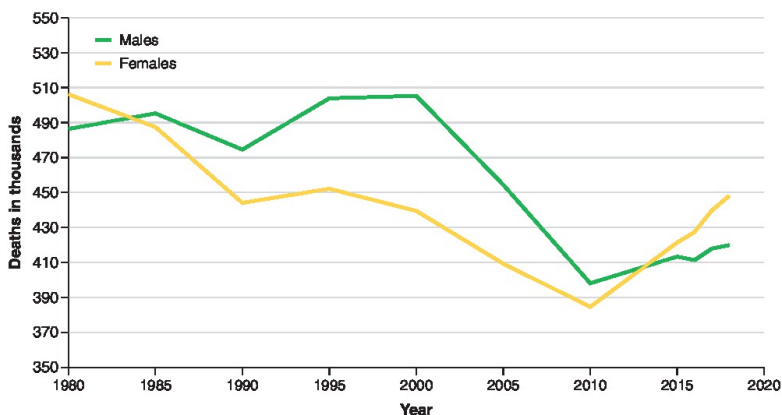


Figure 15.1 Cardiovascular Disease Mortality Trends for U.S. Adults.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021).
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In addition to the emotional tragedy of lives cut short, the economic cost of CVD is staggering. In 2017, the direct and

indirect costs of CVD were estimated at \$363 billion, with the cost of hospital stays accounting for almost \$100 billion (Virani et al., 2021). The cost of treatment is likely to continue to climb, and demographic data indicate an aging America; thus, health care costs associated with CVD are a major national concern. One way to address this cost, and the human burden of disease, is through more widespread and effective preventive strategies including increased physical fitness and physical activity.

Coronary heart disease (CHD), also called coronary artery disease (CAD), is the most common form of CVD and often coexists with other forms of CVD (most often with hypertension). Coronary heart disease is characterized by plaque buildup in the coronary arteries and can lead to ischemia, decreased oxygen to heart tissue resulting from reduced blood flow, or to a myocardial infarction (a heart attack) due to a blood clot forming on ruptured plaque.

Figure 15.2 presents the incidence of myocardial infarction resulting from CHD with advancing age in black and white males and females. Aging increases the risk of death from myocardial infarction, but being young is not an automatic protection from impairment or death. This figure also illustrates the important point that significant disparities exist between racial groups and the burden of CHD. Given the tremendous toll of cardiovascular disease, and the role of physical fitness and physical activity in preventing and treating these diseases, it is important to understand how CHD progresses. While this text cannot fully describe the pathology of atherosclerosis, a basic comprehension of the evolution of atherosclerotic plaque formation is needed to understand how cardiovascular risk factors are related to disease and how exercise training can effectively reduce CVD.

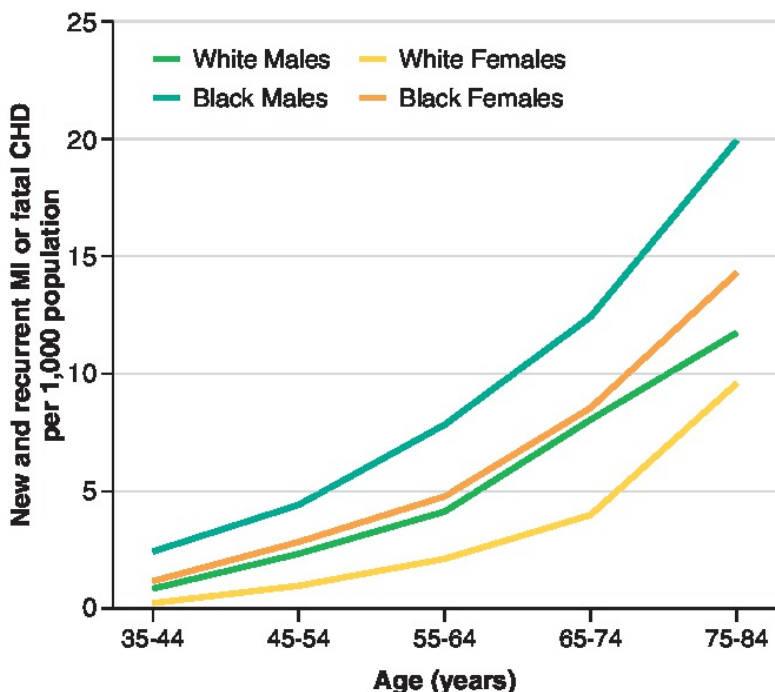


Figure 15.2 Incidence of Myocardial Infarction by Age, Race, and Sex.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021). Copyright © 2021 American Heart Association, Inc.

Progression of Coronary Heart Disease

Coronary heart disease (CHD) results from damage to the

coronary arteries supplying blood flow to the heart muscle (myocardium). Coronary heart disease leads to narrowing (stenosis) of an artery as atherosclerotic plaque is developed in the coronary artery wall. A rupture of this plaque provokes a powerful clotting signal, which can lead to a myocardial infarction, which may or may not be fatal. **Atherosclerosis** is an inflammatory disease arising in response to endothelial cell injury or dysfunction and characterized by lipid accumulation (plaque) in the arterial wall (Libby and Theroux, 2005; Libby et al., 2019). In contrast to atherosclerosis, **arteriosclerosis** encompasses the natural aging changes that occur in blood vessels—namely, thickening of the wall, loss of elastic connective tissue, and hardening of the vessel wall.

Coronary heart disease (CHD) Also called coronary artery disease or ischemic heart disease, CHD results from damage to the coronary arteries supplying the heart muscle (myocardium).

Atherosclerosis A pathological process that results in the buildup of plaque inside blood vessels.

Arteriosclerosis The natural aging changes that occur in blood vessels, including thickening of the walls, loss of elastic connective tissue, and hardening of the vessel wall.

The process of atherosclerosis begins with damage to the endothelium of the artery. The initial injury to the endothelial cells of the arterial wall may result from many factors, including chemical irritants in tobacco smoke; the turbulent blood flow resulting from hypertension, hyperglycemia, high cholesterol (specifically low-density lipoprotein cholesterol—LDL-C) levels; immune complexes; vasoconstrictor substances; homocysteine (a proclotting factor); and viral or bacterial infection (Libby, 2006; Libby et al., 2019). Damaged endothelium allows LDL-C to enter the arterial wall and become oxidized (modified) by free radicals

(see the Focus on Application box in [Chapter 5](#)). Oxidized LDL-C in the arterial wall initiates an inflammatory response that attracts immune cells (macrophages) into the arterial wall. An inflammatory response by the immune system is a normal reaction to such an injury (see [Figure 22.3](#)), but in the case of atherosclerosis, the immune response and the progression of the plaque leads to narrowing of the arteries and the potential for a proclotting condition, which can be life threatening. Macrophages ingest (phagocytize) the LDL-C and release chemicals that stimulate smooth muscle cells in the tunica media of the vessel wall to divide and migrate into the intima. The result of this complex process is the formation of an atherosclerotic plaque (or lesion) inside the blood vessels composed of connective tissue, smooth muscle cells, cellular debris, cholesterol, and, in advanced cases, calcium. The calcification of plaque signals advanced atherosclerosis and is the basis for the calcium scan and other testing that detects atherosclerosis in the coronary arteries. The pattern of calcification may predict which plaques are stable and which are most prone to rupture ([Mori et al., 2018](#)). The progression of atherosclerosis leads to an increased thickness of the tunica intima layer and can seriously decrease the internal diameter of the coronary artery. [Figure 15.3](#) summarizes the process of atherosclerotic plaque development.

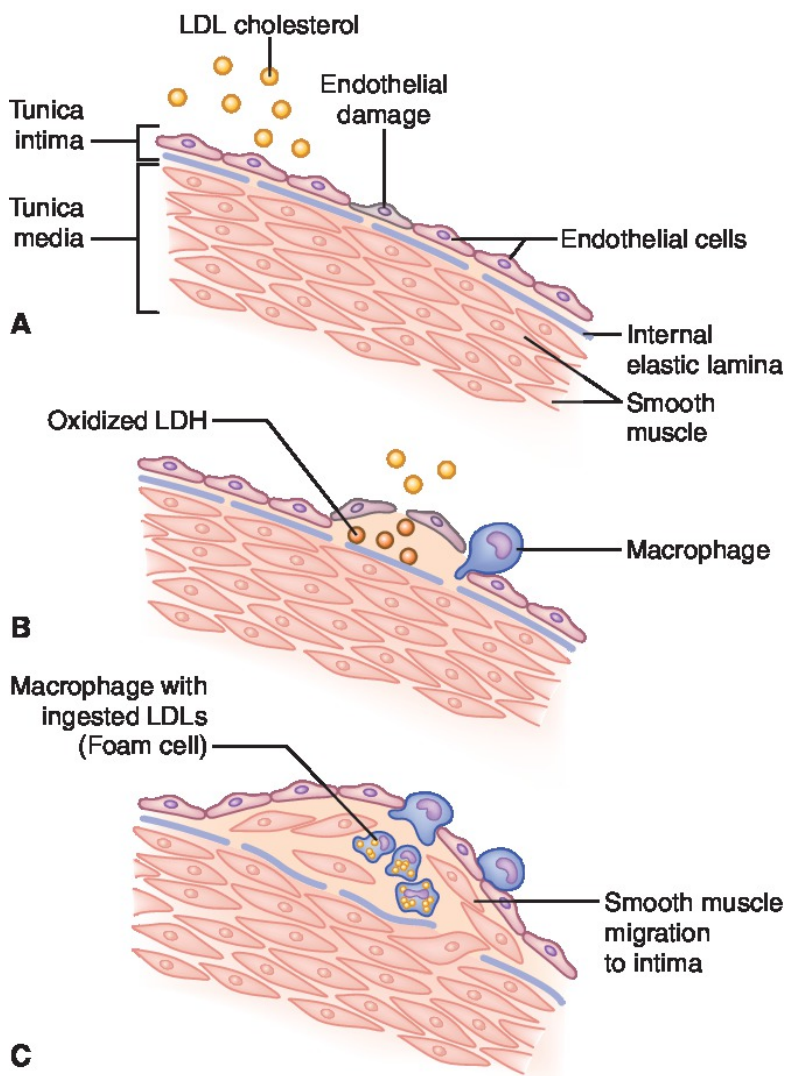


Figure 15.3 Progression of Atherosclerotic Plaque.

A. Atherosclerosis is initiated by damage to the endothelial lining of the vessel wall allowing LDL-C to enter the vessel wall. **B.** LDL-C in the intimal layer of the arterial wall is oxidized, leading to an inflammatory response, which is evident by the entry of macrophages into the arterial wall. **C.** Macrophages ingest the oxidized lipids and release chemical signals that enhance the inflammatory response

and signal smooth muscle division and migration into the tunica intima, resulting in an atherosclerotic plaque that increases intimal thickness.

The growing atherosclerotic plaque can interfere with arterial function in many ways. Early in the progression of atherosclerosis, there is evidence of endothelial dysfunction (an impaired ability to vasodilate in response to increased need for blood flow). As the atherosclerotic plaque progresses, it can cause narrowing of the artery to the extent that blood flow cannot increase sufficiently during periods of increased work (causing pain called *angina pectoris* if in the heart or *claudication* if in the legs). This may be called *demand ischemia* because there is adequate blood flow at rest, but when work is done and there is an increased need for blood flow, the blood flow cannot increase to the extent needed due to narrowing. Atherosclerotic plaque can also cause turbulent blood flow that may lead to the rupture of an aneurism or further injury to the endothelium. Finally, advanced atherosclerotic plaque is highly thrombotic, meaning that if the plaque ruptures, the material in the plaque will lead to the formation of a blood clot that could completely occlude a coronary artery, leading to a myocardial infarction (or a stroke if an artery to the brain is occluded). If the affected area of the myocardium is small, the individual may recover from the infarction. However, if the affected area of the myocardium is large, the heart can no longer pump blood, and the infarction will be fatal. The process of atherosclerotic plaque development may begin in childhood and progress for many years before any clinical symptoms of disease occur. In fact, in many cases, a cardiac event is the first symptom of atherosclerosis. This explains why so much attention is paid to identify risk factors that increase the likelihood of disease and adopting preventive strategies to decrease risk factors and the development of atherosclerosis.

Describing Cardiovascular Health and Disease

As discussed in the section above, atherosclerotic plaque development occurs over many decades, starting with endothelial injury, an inflammatory state, and endothelial dysfunction and progressing to fatty streaks in the arteries and potentially to the development of a complicated plaque that may rupture leading to an acute cardiac event. Thus, it may be useful to consider cardiovascular health and disease as a continuum rather than as dichotomous states. In research, it is common to use clinical descriptors of disease free, subclinical disease, and cardiovascular disease, which may be mild to severe. However, in reality, it is often difficult to know the precise health status of coronary arteries, and the descriptors that are used to describe cardiovascular disease are often dependent upon the measures used to investigate health. Realizing the distinctions between cardiovascular health and disease, and how they are operationalized will make the interpretation of the research findings presented in this chapter much easier to understand.

Cardiovascular Disease

CHD is often defined in research as having had a cardiac event (fatal or nonfatal), or symptoms related to a cardiac event (chest pain, shortness of breath) and reasonable suspicion of CVD (such as elevated blood cholesterol), ECG changes indicative of ischemia, or evidence from advanced testing (such as coronary artery calcium scans or CT imaging) or clinical procedures (angiogram) that verify the presence of coronary artery narrowing of 50% or more. There are also laboratory-based studies that assess endothelial function (using flow-mediated dilation, pulse wave velocity, etc.) that allow researchers to determine early stages of disease progression (subclinical disease). These tests are currently used more for research than for clinical practice.

Ideal Cardiovascular Health

Ideal cardiovascular health is a concept that is being espoused by the American Heart Association (AHA) to promote improved health outcomes and to lessen disease burden. **Ideal**

cardiovascular health is defined as the absence of manifest CVD together with the simultaneous presences of optimal levels of seven cardiovascular health metrics (health behaviors or factors) (Virani et al., 2021). The cardiovascular health metrics include four health behaviors (not smoking, getting sufficient physical activity, having a healthy diet pattern, and normal body weight) and three health factors (optimal total cholesterol, blood pressure, and fasting blood glucose). **Table 15.1** presents definitions for poor, intermediate, and ideal cardiovascular health for each of the cardiovascular health metrics for adults and children. **Figure 15.4** presents the prevalence (percentage) of adults who have values in the ideal category for each of the metrics over time. The prevalence of these ideal metrics varies from 0.5 (healthy diet patterns) to approximately 80% (never smoked or former smoker who has quit for at least 12 months).

Ideal Cardiovascular Health The absence of manifest CVD together with the simultaneous presences of optimal levels of seven cardiovascular health metrics: not smoking, getting sufficient physical activity, having a healthy diet pattern, normal body weight, optimal total cholesterol, optimal blood pressure, and optimal fasting blood glucose.

TABLE 15.1 Definitions of Poor, Intermediate, and Ideal Cardiovascular Health for Each American Heart Association Metric in the American Heart Association

| | Level of Cardiovascular Health for Each Metric | | |
|---|--|---|--|
| | Poor | Intermediate | Ideal |
| Current smoking | | | |
| Adults ≥20 y of age | Yes | Former ≤12 mo | Never or quit >12 mo |
| Children 12–19 y of age | Tried during the prior 30 d | | Never tried; never smoked whole cigarette |
| Body mass index* | | | |
| Adults ≥20 y of age | ≥30 kg·m ⁻² | 25–29.9 kg·m ⁻² | <25 kg·m ⁻² |
| Children 12–19 y of age | >95th percentile | 85th–95th percentile | <85th percentile |
| Physical activity (PA) | | | |
| Adults ≥20 y of age | None | 1–149 min·wk ⁻¹ moderate or 1–74 min·wk ⁻¹ vigorous or 1–149 min·wk ⁻¹ moderate + 2 × vigorous | ≥150 min·wk ⁻¹ moderate or ≥75 min·wk ⁻¹ vigorous or combination |
| Children 12–19 y of age | None | >0 and <60 min of moderate or vigorous every day | ≥60 min of moderate or vigorous every day |
| Healthy diet pattern, no. of components*,† | | | |
| Adults ≥20 y of age | 0–1 | 2–3 | 4–5 |
| Children 12–19 y of age | 0–1 | 2–3 | 4–5 |
| Total cholesterol, mg·dL ⁻¹ | | | |
| Adults ≥20 y of age | ≥240 | 200–239 or treated to goal | <200 |
| Children 12–19 y of age | ≥200 | 170–199 | <170 |
| Blood pressure | | | |
| Adults ≥20 y of age | SBP ≥ 140 mmHg or DBP ≥ 90 mmHg | SBP 120–139 mmHg or DBP 80–89 mmHg or treated to goal | <120 mmHg or <80 mmHg |
| Children 12–19 y of age | >95th percentile | 90th–95th percentile or SBP ≥120 mmHg or DBP ≥80 mmHg | <90th percentile |
| Fasting plasma glucose, mg·dL ⁻¹ | | | |
| Adults ≥20 y of age | ≥126 | 100–125 or treated to goal | <100 |
| Children 12–19 y of age | ≥126 | 100–125 | <100 |

*Represents appropriate energy balance, that is, appropriate dietary quantity and PA to maintain normal body weight.

†In the context of a healthy dietary pattern that is consistent with a Dietary Approaches to Stop Hypertension [DASH]–type eating pattern, to consume ≥4.5 cups·d⁻¹ of fruits and vegetables, ≥2 servings·wk⁻¹ of fish, and ≥3 servings·d⁻¹ of whole grains and no more than 36 oz·wk⁻¹ of sugar-sweetened beverages and 1,500 mg·d⁻¹ of sodium. SBP, systolic blood pressure; DBP, diastolic blood pressure. Reprinted with permission from Lloyd-Jones, D. M., H. Yuling, D. Labarthe, et al.: Defining and setting national goals for cardiovascular health promotion and disease reduction. *Circulation*. 121(4):586–613 (2010).

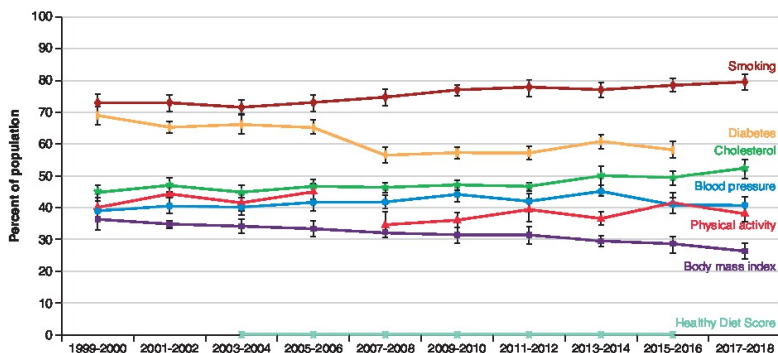


Figure 15.4 Trends in the Prevalence of Meeting Criteria for Ideal Cardiovascular Health for Each of the Seven Metrics of Cardiovascular Health among U.S. Adults ≥ 20 Years of Age.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. 143(8):e254-e743 (2021). Copyright © 2021 American Heart Association, Inc.

Cardiovascular Disease Risk Factors

While the concept of ideal cardiovascular health is an attractive option for the promotion of positive cardiovascular health, many researchers and practitioners continue to focus on identifiable risk factors for developing CVD. The emphasis on risk factors assumes that if an individual can be identified as “at risk,” then interventions can be designed to treat or lessen that risk. Obviously, focusing on risk factors or ideal cardiovascular health represents different approaches to achieving the same goal—improved cardiovascular health.

Given the high prevalence of CVD and its morbidity (disease) and mortality (death) consequences, the ability to predict who is at greatest risk for developing CVD is valuable. A **risk factor** is an aspect of personal behavior or lifestyle, an environmental exposure, or an inherited characteristic that has been shown by epidemiological evidence to predispose an individual to the development of a specific disease (Caspersen and Heath, 1993). **Table 15.2** lists various classifications of risk factors for coronary heart disease. Age, race, sex, and heredity are risk factors that cannot be changed. Age becomes a risk factor for males at 45 years and for females at 55 years. Heredity is interpreted as a family history of myocardial infarction, coronary revascularization (bypass surgery), or sudden death before 55 years of age in father or other male first-degree relative if male or before 65 years of age in mother or other female first-degree relative if female (ACSM, 2022).

Risk Factor An aspect of personal behavior or lifestyle, an environmental exposure, or an inherited characteristic that has been shown by epidemiological evidence to predispose an individual to develop a specific disease.

TABLE 15.2 Coronary Heart Disease Risk Factors

| Nonmodifiable Risk Factors | Major Modifiable Risk Factors |
|----------------------------|-------------------------------|
| Age | Cholesterol-lipid fractions |
| Heredity | Cigarette smoking |
| Race | Diabetes mellitus |
| Sex | Hypertension |
| | Obesity |
| | Physical inactivity |

The major modifiable risk factors are, at least to some degree, changeable through diet, exercise, medication, or other lifestyle alterations. These modifiable risk factors correspond closely to

the ideal cardiovascular health metrics listed in [Table 15.1](#). For example, [Table 15.1](#) indicates that a $\text{BMI} \geq 30 \text{ kg}\cdot\text{m}^{-2}$ (for adults) is defined as a “poor” cardiovascular health metric. This correlates with obesity being a risk factor. Similarly, [Table 15.1](#) indicates that a fasting plasma glucose $\geq 126 \text{ mg}\cdot\text{dL}^{-1}$ is defined as a “poor” cardiovascular health metric. This corresponds with diabetes mellitus, whereas blood glucose $100\text{--}125 \text{ mg}\cdot\text{dL}^{-1}$ is termed prediabetes and indicates an intermediate-risk factor for CVD.

Cardiorespiratory Fitness and Cardiovascular Mortality

There is strong epidemiological evidence that higher levels of physical fitness and physical activity are associated with decreased risk of cardiovascular mortality and morbidity. This information alone is enough to state definitely that the regular physical activity and higher levels of cardiorespiratory fitness play an important role in preventing cardiovascular disease.

FOCUS ON APPLICATION

The Impact of a Change in Physical Fitness on Cardiovascular Disease Mortality

This chapter describes the inverse relationship between physical activity and mortality from coronary heart disease and the positive impacts of exercise training on each cardiovascular disease risk factor. The cited studies relating physical activity or physical fitness to mortality, however, generally used only a single baseline evaluation of activity or fitness with subsequent follow-up to determine the incidence of death from cardiovascular causes. Although extremely

important, these results could have been influenced both by genetics and by changes in risk factor status between the baseline testing and time of death. Conversely, the studies of exercise training had both pretraining and posttraining evaluations but did not examine the overall impact on cardiovascular mortality. From a public health, exercise professional, and personal perspective, the overall goal of exercise training is to enhance both the quality and quantity of life. It is important to determine whether improving and maintaining physical fitness can attain these goals. Therefore, a linkage between the two types of studies is important.

Blair et al. (1995) studied the mortality and relative risk of death ratios from cardiovascular disease of 9,777 males who had at least two complete examinations at the Cooper Institute for Aerobics Research between 1970 and 1989. The average interval between the two testing sessions was 4.0 ± 4.1 years, and the average follow-up for mortality was 5.1 ± 4.2 years, but the range was 1–18 years for both. Treadmill time to volitional fatigue, converted to maximal oxygen uptake, was the measure of cardiovascular physical fitness. Individuals whose results fell into the bottom quintile (lowest 20%) based on age-adjusted results were labeled unfit. The unfit group had the following $\text{VO}_2 \text{ max}$ values for each age group:

Less than $35.0 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, 20–39 years

Less than $32.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, 40–49 years

Less than $29.4 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, 50–59 years

Less than $24.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, 60+ years

All individuals in quintiles (Q) 2, 3, 4, and 5 were labeled fit. Men who were unfit at both testing times had the highest death rate from CVD; men who were fit at both testing times had the lowest death rate; and men who changed fitness status between testing sessions had intermediate death rates, as shown in panel A of the accompanying graph. Although the values for the men who changed fitness status appear to be similar, these must be interpreted relative to the direction of the change. The unfit men who moved out of the lowest quintile died at a rate less than half that of those who

remained unfit, whereas those who moved from fit to unfit had an increased death rate of approximately 25%. When changes in status occurred among the four upper quartiles, a dose-response relationship was seen. A low mortality rate was evident in men who remained in quintiles 2 and 3; a lower mortality rate was seen in men who moved from quintiles 2 and 3 to 4 and 5, and the lowest mortality rate was seen in men in the upper two quintiles (4 and 5) who remained there.

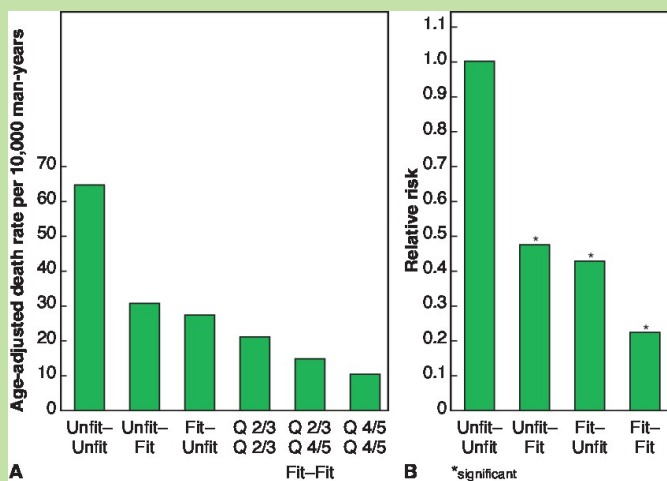
The comparable results in terms of relative risk are shown in panel B of the graph. The unfit men who moved into the fit category reduced their risk of death from cardiovascular disease by 52% (relative risk = 0.48). After adjustment for potential confounders, each minute of increase in treadmill

time (equivalent to $\text{VO}_2 \text{ max}$ increase of only $1.75 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in the protocol used) was associated with a reduced risk of 8.6% for CVD mortality. Those who changed from fit to unfit had a risk less than half that of the consistently unfit, but almost twice that of those who remained fit.

A follow-up analysis of these data ([Kampert, 2004](#)) confirmed that mortality risk was inversely related to change in fitness after correction for measurement error and adjustment for initial CVD, fitness level, age, health status, and exam year.

The message is clear. Individuals who are unfit must be encouraged and helped to attain at least a minimal level of fitness. This minimal level can be achieved by engaging in at least 30 minutes of moderate physical activity 5 days of the week or vigorous activity 3 d·wk⁻¹ (ACSM, 2022; [Haskell et al., 2007](#)). Individuals who are already fit must be equally encouraged and helped to maintain or improve their level of fitness. Although Blair and his colleagues' results were compiled only on males (because of an insufficient number of female subjects), [Manson et al. \(1999\)](#) have reported similar findings from the Nurses' Health Study, which followed 72,488 women 40–65 years of age.

It is never too late to try to become fit; it is never too soon to make a lifetime commitment to activity and fitness.



Sources: [Blair et al. \(1995\)](#); [Kampert \(2004\)](#); [Manson et al. \(1999\)](#).

Moderate to high levels of cardiorespiratory fitness are associated with decreased risk of all-cause mortality and a decreased risk of a sudden cardiac event. In a landmark study, Jimenez-Pavon et al. investigated the magnitude of risk reduction in a group of over 55,000 individuals. The authors found an inverse relationship between cardiorespiratory fitness and the risk of sudden cardiac events even after adjusting for potential confounders like cardiovascular disease risk factors. Those who had moderate to high cardiorespiratory fitness (based on age and sex-specific distribution) had a 44% and a 48% lower risk of sudden cardiac event than those with a low fitness level (a

$\dot{V}O_2$ max of approximately 8 METs or 28 mL.kg.min⁻¹). Further analysis showed a 14% reduction in risk of sudden cardiac event for every 1 metabolic equivalent (3.5 mL.kg.min⁻¹ of oxygen) increase in measured fitness (Jimenez-Pavon et al., 2016). Importantly, the results also demonstrated that individuals who were hypertensive, overweight, or had other risk factors but had moderate to high levels of cardiorespiratory fitness had a lower risk of sudden cardiac death than those who had similar risk factor profiles but were unfit.

Exercise Training and Modifiable Cardiovascular Disease Risk Factors

Much of the influence of exercise training (or consistent physical activity) on atherosclerosis and the risk of cardiovascular disease is likely mediated through changes in cardiovascular risk factors, and a great deal of research has been conducted in this area. Each major risk factor acts independently, but many are also interrelated and act jointly. In general, the greater the number of risk factors an individual has, the greater the level of risk of cardiovascular disease risk. The following sections discuss each of the major modifiable risk factors and how they are influenced by exercise training.

Cholesterol-Lipid Fractions

Lipids, or fats, are by definition water-insoluble substances. They may be classified as simple (or neutral) fats, compound fats, and derived fats. The primary simple fat is triglyceride. Compound fats are combinations of a neutral fat and another substance such as phosphate (phospholipid), glucose (glucolipid), or protein (lipoprotein). Derived fats originate from simple and compound fats. Chief among them is **cholesterol**, a derived fat that is essential for the body but may be detrimental in excessive amounts.

Cholesterol A derived fat that is essential for the body but may be detrimental in excessive amounts.

Combining fats with another substance typically makes the compound water soluble, and it is through such compounds—specifically **lipoproteins**—that fat is transported in the bloodstream. Lipoproteins are so named due to the presence of lipids (triglycerides or free fatty acids and cholesterol) and proteins (**apolipoproteins**) in their structures. Five types of lipoproteins circulate in blood: chylomicrons, very-low-density lipoproteins, intermediate-density lipoproteins, low-density

lipoproteins, and high-density lipoproteins (Table 15.3). As depicted in Figure 15.5, *chylomicrons* are microscopic fat particles that transport dietary sources of triglycerides and cholesterol to the lymphatic system and ultimately to the bloodstream. In the blood, chylomicrons and *very-low-density lipoproteins (VLDLs)* deliver triglycerides and cholesterol to adipose tissue for storage or to muscle for use as fuel. The removal of triglyceride from a VLDL results in an *intermediate-density lipoprotein (IDL)*, which in turn is converted in the liver to a **low-density lipoprotein (LDL-C)**. LDL-C is composed of protein, a small portion of triglyceride, and a large portion of cholesterol. LDL-C transports 60–70% of the total cholesterol in the body to all cells except liver cells. The major apolipoprotein of LDL-C is called Apo-B. As mentioned earlier, LDL-C is involved in the formation of atherosclerotic plaque.

Lipoprotein Water-soluble compound composed of apolipoprotein and lipid components that transport fat in the bloodstream.

Apolipoprotein The protein portion of lipoproteins.

Low-Density Lipoprotein (LDL-C) A lipoprotein in blood plasma composed of protein, a small portion of triglyceride, and a large portion of cholesterol whose purpose is to transport cholesterol to the cells.

TABLE 15.3 Types of Lipoproteins Circulating in the Blood

| Lipoprotein | Description/Composition | Function | Atherosclerotic Role |
|-------------|---|--|----------------------|
| Chylomicron | Triglyceride: 75% Protein: 5–10% Cholesterol: 5–10% Phospholipid: 5–10% | Transports dietary triglycerides from small intestines to liver and muscle cells | Neutral |
| HDL | Triglyceride: 10% Protein: 40–50% Cholesterol: 15–20% Phospholipid: 25–30% | Transports synthesized cholesterol from body cells to liver for excretion | Atheroprotective |
| LDL | Triglyceride: 10% Protein: 25% Cholesterol: 45% Phospholipid: 20% | Transports synthesized cholesterol from liver to body cells | Atherogenic |
| VLDL | Triglyceride: 55–65% Protein: 5–10% Cholesterol: 10–15% Phospholipid: 15–20% | Transports triglycerides and cholesterol from liver to body cells | Neutral |

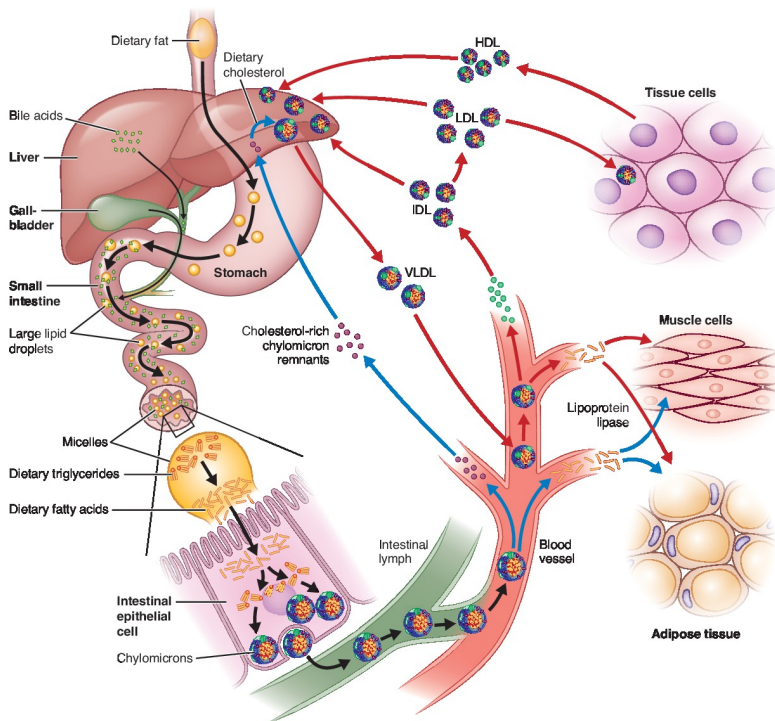


Figure 15.5 Fat and Cholesterol Transport in the Body.

High-density lipoprotein (HDL-C) is a lipoprotein in blood plasma composed primarily of protein and a minimum of

cholesterol or triglyceride. The purpose of HDL-C is to transport cholesterol from body tissues to the liver where the cholesterol can be broken down and eliminated in bile.

High-Density Lipoprotein (HDL-C) A lipoprotein in blood plasma composed primarily of protein and a minimum of cholesterol or triglyceride whose purpose is to transport cholesterol from the tissues to the liver.

Triglycerides (but not the associated VLDLs) represent an independent risk factor (Summary of the Third Report of the National Cholesterol Education Program [NCEP], 2001). Chylomicrons are not thought to be atherosclerotic. Abundant evidence shows, however, that an elevated total cholesterol (TC) level is independently and directly related to CHD incidence. A classification system for TC, and other blood lipids, is presented in Table 15.4 (ACSM, 2022; Summary of the Third Report of the National Cholesterol Education Program [NCEP], 2001). They apply to all adults regardless of age or sex. (The risks in children are considered later.) The term *hyperlipidemia* (excessive fat in the blood) is commonly used to describe this risk factor. As seen in Figure 15.4, just over 50% of American adults have ideal total cholesterol levels ($<200\text{ mg}\cdot\text{dL}^{-1}$).

TABLE 15.4 Classification of Total, LDL-C, HDL-C, Blood Levels, and Triglycerides

| Lipid and Category | Level for Adults (mg·dL ⁻¹) | Level for Children and Adolescents (mg·dL ⁻¹) |
|--------------------|---|---|
| Total cholesterol | | |
| Desirable | <200 | <170 |
| Borderline high | 200–239 | 170–199 |
| High | ≥240 | ≥200 |
| Non-HDL-C | | |
| Desirable | <130 | |
| Above desirable | 130–159 | |
| Borderline high | 160–189 | |
| High | 190–219 | |
| Very high | ≥220 | |
| LDL cholesterol | | |
| Desirable | <100 | <110 |
| Above desirable | 100–129 | 110–129 |
| Borderline high | 130–159 | 130 |
| High | 160–189 | |
| Very high | ≥190 | |
| HDL cholesterol | | |
| Low | <40 (males) <50 (females) | |
| High | ≥60 | |
| Triglyceride level | | |
| Normal | <150 | |
| Borderline high | 150–199 | |
| High | 200–499 | |
| Very high | ≥500 | |

Source: ACSM (2022); [Summary of the Third Report of the National Cholesterol Education Program \(NCEP\) \(2001\)](#).

Not only is the total amount of cholesterol important but also the fractions of LDL-C and/or HDL-C are important. High levels of LDL-C and/or Apo-B are positively related to CHD—hence the common term “bad cholesterol.” High levels of HDL-C and/or

Apo-A1 are inversely related to CHD—hence the term “good cholesterol.” The optimal level of LDL-C is less than 100 mg·dL⁻¹. A low level of HDL-C is less than 40 mg·dL⁻¹ for men and less than 50 mg·dL⁻¹ for women. An HDL-C level ≥ 60 mg·dL⁻¹ is actually so good that it is said to be a negative risk factor, which means it lowers one’s risk for disease (American College of Sports Medicine [ACSM], 2022). The beneficial effect of HDL are largely due to its role in transporting cholesterol from the peripheral tissues to the liver. However, evidence also indicates that HDL also has anti-inflammatory, antioxidative, and vasodilatory properties (Nagao et al., 2018). The major apolipoprotein of HDL-C is called Apo-A1. Because HDL-C transports cholesterol to the liver for elimination, high levels of HDL are good. In fact, high levels are cardioprotective, sometimes called *atheroprotective*.

Some researchers consider decreased Apo-A1 and increased Apo-B to be independent risk factors and believe they add considerably to risk prediction (Barter et al., 2006; Sacks, 2006; Tian et al., 2019). However, other researchers have found that these values add little to the predictive value of existing risk factors (van der Steeg et al., 2007), while still other authorities are cautious about the ability of any new risk factor to add substantially to the prediction of a chronic medical disease with many contributing causes (Berkwits and Guallar, 2007). The combination of hypertriglyceridemia (high triglycerides), low HDL-C, and small, dense LDL-C is known as the **dyslipidemia triad** and is associated with a much higher risk of CVD than any single risk factor (Jellinger et al., 2012). While the association between elevated lipid levels and cardiovascular disease has been well-established, the extent to which different blood lipids directly promote atherosclerosis or represent a biomarker of risk continues to be debated (Tian et al., 2019).

Dyslipidemia Triad The combination of hypertriglyceridemia (high triglycerides), low HDL-C, and small, dense LDL-C, which is associated with a much higher risk of CVD than any single factor.

The Influence Exercise Training on Blood Lipids

The impact of exercise on lipid levels may be both transient (a last-bout effect from a single bout of exercise) and chronic (a consistent adaptation resulting from exercise training). Studies routinely report beneficial effects of endurance training on serum lipid profile, including significant decreases in TC, LDL-C, and triglycerides and a significant increase in HDL-C, but the mechanisms for the protective effects of exercise on lipid metabolism are not yet fully understood (Muscella et al., 2020).

The majority of studies that last longer than 12 weeks and have a training volume equivalent to at least $15 \text{ km}\cdot\text{wk}^{-1}$ ($9.3 \text{ mi}\cdot\text{wk}^{-1}$) of running or $1,000\text{--}1,200 \text{ kcal}\cdot\text{wk}^{-1}$ ($4,180\text{--}5,016 \text{ kJ}\cdot\text{wk}^{-1}$) of aerobic activity have shown a significant increase in HDL-C levels. Postexercise increases in HDL-C range from 3 to 34% and last from 24 to 72 hours (Grandjean and Crouse, 2004). These positive changes in HDL-C with high-intensity training appear to be similar for men and women, independent of age (Tambalis et al., 2009).

A high level of energy expenditure is apparently needed to positively alter blood lipids. If the objective is to modify one's lipid profile, more than 30 minutes of light to moderate activity is needed. A dose-response relationship seems to occur such that more vigorous activity and higher total calorie expenditure result in greater HDL-C increases. Figure 15.6 presents data on HDL-C and triglycerides based on distance run per week in a group of over 8,000 male recreational runners (Williams, 1997). As the figure shows, increased running mileage was associated with a proportional increase in HDL-C levels over most of the range. The lack of a proportional increase in the mean HDL-C value for those running more than $80 \text{ km}\cdot\text{wk}^{-1}$ ($\sim 50 \text{ mi}\cdot\text{wk}^{-1}$) reflects more on the low number of individuals in that category than does any ceiling effect for HDL-C change. The percentage of individuals with HDL-C values greater than $60 \text{ mg}\cdot\text{dL}^{-1}$ for each 15 km (~ 10 miles) category of running per week was 17, 20, 26, 31, 39, and 42, respectively. Remember that at $60 \text{ mg}\cdot\text{dL}^{-1}$, HDL-C becomes a negative risk factor. A meta-analysis (Kelley and Kelley, 2006) has shown consistent increases in HDL-C with aerobic training of at least 8 weeks, independent of decreases in body weight, body

mass index (BMI), and percent body fat. Triglycerides are also consistently reduced for up to 44 hours after activity ([Grandjean and Crouse, 2004](#)). Also, as indicated in [Figure 15.6](#), TG show a dose-response relationship with greater reductions occurring as mileage run per week increases.

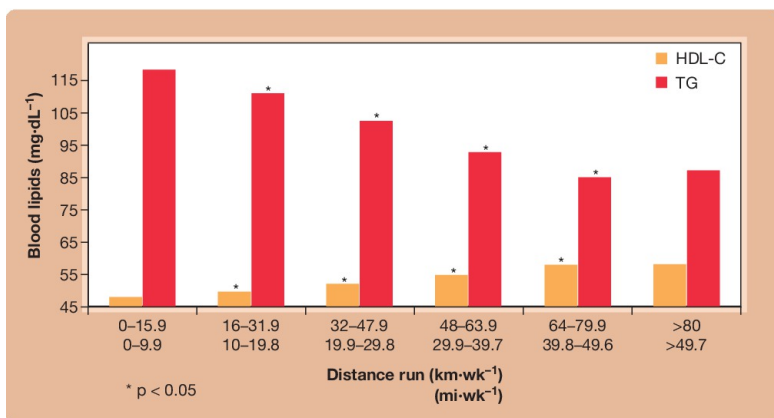


Figure 15.6 Dose-response Relationship between Distance Run per Week and Levels of High-Density Lipoprotein (HDL-C) and Triglycerides (TG) in Males.

*Significantly different from next lower distance category.

Source: Based on data from [Williams \(1997\)](#).

Favorable changes in blood lipids measured after eating are generally seen when aerobic exercise is completed in the period extending from 16 hours before to 1.5 hours after the meal, is of at least moderate intensity, and expends at least 500 kcal (2,090 kJ) ([Katsanos, 2006](#)). Highly trained individuals may need a higher caloric expenditure to elicit HDL-C increases. In addition, to maintain the beneficial effects in lipid variables, the individual must consistently engage in aerobic exercise of sufficient intensity and duration, that is, at least every 3rd day and preferably every 2nd day ([Grandjean and Crouse, 2004](#)). Note also that both the positive results in lipid profiles and the exercise recommendations are based on studies that investigated aerobic activities. Research investigating resistance training programs have reported some beneficial effects on lipid metabolism, but others have found that

resistance-trained individuals have lipid profiles similar to those of untrained individuals ([Grandjean and Crouse, 2004](#); Muscella et al., 2020).

Cigarette Smoking

Cigarette smoking in the United States is declining, but it still continues to be a significant cause of CVD and death. The AHA reports that globally approximately 8.7 million deaths were attributable to cigarette smoking in 2019 (Virani et al., 2021). Of course, smoking also increases the risk of cancer and other respiratory diseases. For males, smoking one pack a day doubles the risk compared to not smoking, and smoking more than one pack a day triples the risk. For females, smoking as few as 1–4 cigarettes per day doubles the risk. The longer an individual smokes, the higher the risk ([Gordon et al., 2006](#)). Individuals who currently smoke (or have exposure to environmental tobacco smoke) and those who have quit within the last 6 months are considered to have the same risk (ACSM, 2022). On average, male smokers die 13.2 years earlier than do nonsmokers and female smokers 14.5 years earlier ([Mozaffarian et al., 2015](#)). Breathing secondhand smoke also increases the risk of heart disease by at least 25% ([Gordon et al., 2006](#)). Cigar and pipe smokers have a higher risk for CVD than do nonsmokers but lower than do cigarette smokers. As seen in [Figure 15.4](#), the incidence of smoking has decreased over the last decade (i.e., percentage of Americans meeting ideal smoking metric increased). However, approximately 20% of adults are engaging in an activity that represents a significant risk for CVD as well as lung disease and cancer. Another concerning issue is the number of children who are using e-cigarettes or vaping. These devices also carry significant health risks.

The chemicals in cigarettes, particularly nicotine, stimulate the sympathetic nervous system, causing an acute increase in heart rate and blood pressure, thus making the heart work harder. Carbon monoxide in smoke binds with hemoglobin, thus reducing oxygen transport. The atherosclerotic process is accelerated because smoking injures the arterial wall lining (endothelium); increases the levels of circulating TC, LDL-C, and TG; and decreases the amount of HDL-C.

Smoking also causes blood platelets to adhere to each other, speeds up internal blood clotting, and makes clots that do form tougher to dissolve. Prostacyclin, which is partially responsible for blood vessel dilation, is decreased. Capillaries and small arteries constrict and may spasm shut. Thus, smoking increases the possibility of a thrombus (clot) or an embolism (moving clot) blocking an artery already narrowed by atherosclerosis.

Narrowing of blood vessels to the arms and legs also makes smokers vulnerable to peripheral vascular disease, which may lead to gangrene and amputation. Smokers are 10 times more likely to develop peripheral vascular disease than are nonsmokers ([American Heart Association \[AHA\], 2007](#)). Certain life-threatening dysrhythmias of the heartbeat are also more likely to occur. Thus, smoking both operates independently and contributes to other CHD risk factors (AHA, 2021).

The Influence of Exercise and Exercise Training on Cigarette Smoking

The relationship between exercise training and smoking is only indirect. One study of over 3,000 individuals showed a consistent and statistically significant inverse relationship between the number of cigarettes smoked and the level of physical activity in both males and females ([Dannenberg et al., 1989](#)). Another study (a random sampling of Peachtree Road Race runners) indicated that 85% of both male and female runners had never smoked. In addition, 81% of the men and 75% of the women who had smoked when they began running had since stopped. Only 1% of the men and 2% of the women who had been nonsmokers started smoking after beginning to run ([Koplan et al., 1982](#)).

Brief bouts of aerobic exercise have been shown to result in an acute reduction in tobacco withdrawal symptoms and cravings to smoke ([Elibero et al., 2011](#); [Haasova et al., 2013](#)). Similar results were found with static exercise (pushing one hand against another) performed for a short period of time ([Ussher et al., 2006](#)). Although these results are encouraging, there is no direct cause-and-effect relationship between exercise training and smoking or not. You may know someone who is active or a good athlete who also smokes. Individuals must make a conscious

decision to stop smoking. Research has found that among heavy smokers, smoking cessation was associated with a significantly lower risk of CVD within 5 years. However, relative to never smokers, former smokers' CVD risk remained significantly elevated beyond 5 years after smoking cessation ([Duncan et al., 2019](#)). This information certainly supports vigorous efforts to provide smoking cessation programs and also reinforces the importance of education and policies aimed at preventing young people from beginning to smoke.

Prediabetes and Diabetes Mellitus

Diabetes mellitus is a complex metabolic disease characterized by an inability to use carbohydrates effectively (glucose intolerance). There are four categories of diabetes based on etiology (cause): type 1, type 2, gestational (onset during pregnancy), and other (due to genetic abnormalities, medication, or other illnesses). Only type 1 and type 2 are considered here. Diabetes has become a widespread epidemic in this country, with type 2 diabetes accounting for 90–95% of all cases of diabetes in the United States. Approximately 9.8% of Americans were diagnosed with diabetes in 2016, and an additional 3.7% of the population had undiagnosed diabetes (Virani et al., 2021). The number of individuals diagnosed with diabetes in the United States has risen in the last few years and presents a serious health challenge. It is projected that the total prevalence of diabetes mellitus will more than double in the United States from 2005 to 2050 (from 5.6 to 12.0% of the population) ([Mozaffarian et al., 2015](#)). A substantial percentage of American adults (~38%) have prediabetes (Virani et al., 2021). People with prediabetes have an increased risk for developing type 2 diabetes, heart disease, stroke, blindness, and kidney disease. In one study of almost 250,000 individuals with diabetes, about 66% of them had two or more cardiovascular disease risk factors ([McGurnaghan et al., 2019](#)), highlighting the tremendous overlap between diabetes and cardiovascular disease.

Type 1 diabetes begins most commonly in childhood and adolescence but is occurring more frequently in older individuals. In type 1 diabetes, an environmentally triggered autoimmune process destroys the insulin-producing beta cells in the pancreas.

Thus, an external source of insulin must be supplied.

Type 2 diabetes is a progressive disease whose diagnosis is often delayed for years. The underlying causes of type 2 diabetes are insulin resistance (an inability to achieve normal rates of glucose uptake in response to insulin) and defective secretion of insulin by pancreatic beta cells. **Prediabetes**, also called *insulin resistance (IR)*, typically precedes the onset of type 2 diabetes. Prediabetes is characterized by elevations in blood sugar level that get progressively higher until reaching the level of actual diabetes.

Prediabetes Blood glucose levels between 100 and 125 mg·dL⁻¹ confirmed by measurements on at least two separate occasions, also called insulin resistance.

The normal fasting glucose level is less than 100 mg·dL⁻¹. Values between 100 and 125 mg·dL⁻¹ confirmed by measurements on at least two separate occasions are designated as prediabetes. Diabetes is diagnosed by a fasting blood glucose of greater than 126 mg·dL⁻¹, or hemoglobin A1c $\geq 6.5\%$ (ACSM, 2022). Genetic and environmental factors affect the development of type 2 diabetes, but the risk of developing prediabetes and type 2 diabetes increases with age, obesity (predominantly abdominal visceral obesity), and a lack of physical activity. Type 2 diabetes used to occur predominantly in adults over the age of 40 years, but in recent years, it has become increasingly prevalent in younger individuals, including children and adolescents.

Early in the progression of type 2 diabetes, insulin may be produced in sufficient or even excessive amounts. Thus, individuals with type 2 diabetes are initially not insulin dependent, but eventually, approximately 40% of these individuals will require insulin injections. Among the many pathological complications resulting from prediabetes and diabetes are an acceleration of atherosclerosis, impaired myocardial contraction, poor peripheral perfusion, and alterations in blood coagulation mechanisms, including increased fibrinogen levels (Aronson and Rayfield, 2005).

Figure 15.7 illustrates the complex interactions between prediabetes/diabetes and increased cardiovascular risk. In the prediabetic state, cells of the body are insulin resistant; thus, the pancreas secretes more insulin in order to facilitate the transfer of glucose into the cell. At this stage, blood glucose levels are slightly elevated, and the damage done to the cardiovascular system is mediated primarily through the negative consequences of hyperinsulinemia (elevated insulin) and insulin resistance. As seen in the large wedge at the bottom of **Figure 15.7**, insulin resistance is associated with several negative cardiovascular consequences, including dyslipidemia, hypertension, impaired fibrinolysis (clot breakdown), endothelial dysfunction, and inflammation. The pattern of dyslipidemia seen with insulin resistance often includes the disorders that make up the dyslipidemia triad (described earlier) (Jellinger et al., 2012). As the disease progresses, the beta cells of the pancreas become unable to continue secreting elevated insulin, and insulin levels fall. The falling levels of insulin, along with the increasing insulin resistance of cells, means that blood glucose levels rise (the defining characteristic of diabetes). At this stage in the disease, the cardiovascular system is negatively impacted by the high glucose levels (hyperglycemia). As depicted in the top wedge in the diagram, hyperglycemia is also associated with a number of negative cardiovascular consequences, including increased oxidative stress, endothelial dysfunction, and hypercoagulability (increased clotting potential). Thus, both hyperglycemia and insulin resistance contribute to increased cardiovascular complications. Importantly, **Figure 15.7** illustrates that the negative cardiovascular consequences associated with insulin resistance can precede the diagnosis of diabetes by many years. In other words, prediabetes is a very serious condition.

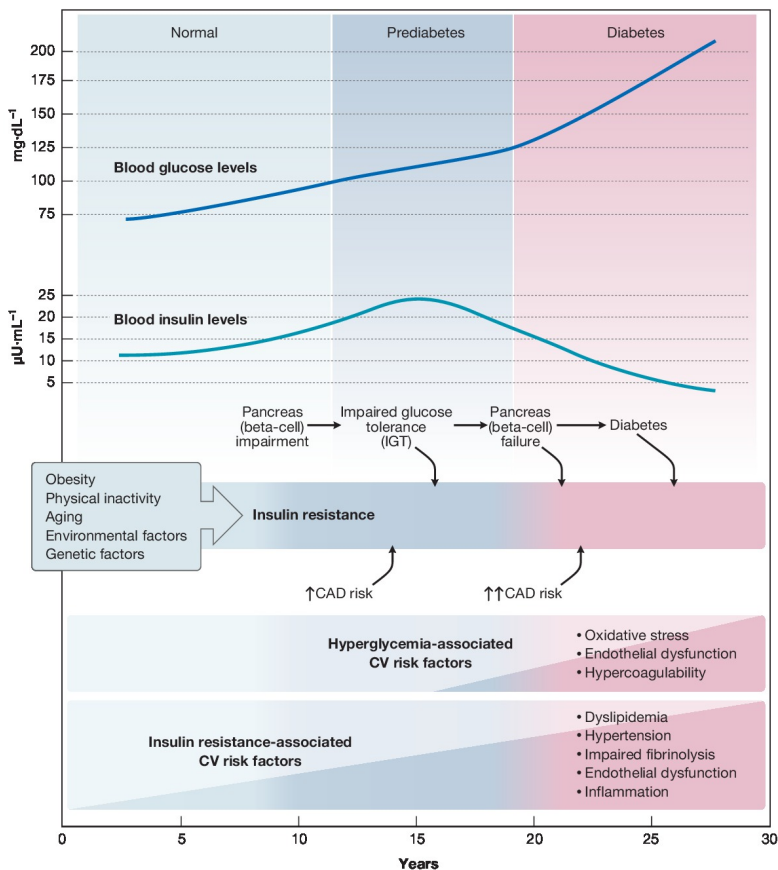


Figure 15.7 Relationship between Prediabetes/Diabetes and Increased Cardiovascular Risk.

CAD, coronary artery disease; CV, cardiovascular. **Source:** Modified with permission from Aronson, D., & E. J. Rayfield: Diabetes, obesity, and the metabolic syndrome. In Fuster, V., R. Ross, E. J. Topol (eds.): *Atherosclerosis and Coronary Artery Disease* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins, 263 (2005).

The Influence of Exercise Training on Prediabetes and Diabetes

The evidence is solid and consistent that exercise training is important in both the prevention and treatment of type 2 diabetes

and a reduction in the progression of prediabetes to type 2 diabetes. Evidence also consistently shows a reduction of CVD risk in individuals with prediabetes and diabetes as a result of exercise training (Alcazar et al., 2006; ACSM and American Diabetes Association, 2010; Pan et al., 2018; Sigal et al., 2006).

One of the acute effects of exercise is an increase in non-insulin-dependent uptake of glucose into the active skeletal muscle. This effect continues after exercise while the depleted stores of glucose (as glycogen) are restored. Studies have shown increased insulin sensitivity and glucose tolerance as a result of exercise training. These changes are transient last-bout or augmented last-bout effects persisting from 12 to 72 hours from a single bout of aerobic exercise. The effect of a dynamic resistance exercise session may last somewhat longer. This means, of course, that a constant pattern of exercise (defined as no more than 2 consecutive days without exercise) must be established to maintain the benefits.

Exercise is usually one of the first recommendations for patients newly diagnosed with type 2 diabetes. Exercise training leads to improved glucose regulation and improved cardiovascular health metrics. Both aerobic exercise training and resistance training exercise programs are effective in reducing blood glucose and HgA1c and in leading to improvements in cardiovascular disease risk factors in individuals with diabetes (Pan et al., 2018). High intensity has also been shown to be an effective way (and time efficient way) for individuals with diabetes to lower blood sugar levels (Kirwan et al., 2017). Exercise training exerts positive effects on blood glucose parameters, the lipid profile, and blood pressure through multiple overlapping mechanisms, including loss of body weight, decreased oxidative stress, and a reduction in systemic inflammation (Amanat et al., 2020).

Exercise is important to maintaining glycemic control in individuals with diabetes and can be an important way to manage comorbidities such as obesity and hypertension. The American College of Sports Medicine recommends moderate-to-vigorous aerobic exercise be performed 3–7 d·wk⁻¹ for a cumulative time of 150 min·wk⁻¹. Additionally, individuals with diabetes should undertake moderate-to-vigorous resistance exercise at least 2–3

d·wk⁻¹ (ACSM, 2022).

Exercise training for individuals with diabetes requires close monitoring and should be done in conjunction with medical personnel. Individuals with diabetes, especially type 1 individuals, may have abnormal blood pressure and other cardiovascular responses to exercise. Insulin injections (the amount used and choice of injection site to avoid working muscles) and carbohydrate ingestion must be carefully adjusted to prevent an exercising diabetic from developing hypoglycemia.

Complete the [Check Your Comprehension 1—Case Study 1](#) analysis to determine your understanding of prediabetes.

CHECK YOUR COMPREHENSION 1—CASE STUDY

Jansson is a 42-year-old sales manager who works 60 hours a week, eats poorly when he is on the road, and seldom exercises. He recently had an annual medical evaluation, and his physician informed him that his blood glucose was 120 mg·dL⁻¹ and that he was prediabetic. Is this a serious medical concern? What can he do to decrease his risk of developing diabetes?

Check your answer in Appendix C.

Hypertension

Hypertension increases the risk of cardiovascular events and stroke. Nearly half of American adults have **hypertension** or high blood pressure (Virani et al., 2021). Ninety to ninety-five percent of the cases of hypertension lack a known cause. Such hypertension is called *primary or essential hypertension*. The prevalence of hypertension increases with advancing age, reaching greater than 75% in those over 65 years. Furthermore, prevalence varies widely by race and sex. Black males and females have a higher prevalence of hypertension (58.3 and 57.7%, respectively) than whites (51.0 and 40.5%) based on data from 2015 to 2018 (Virani et al., 2021). [Figure 15.4](#) presents the population of individuals (just over 40%) who meet the ideal classification for blood pressure (<120/80 mmHg). In contrast,

approximately one third of adults are hypertensive.

Hypertension High blood pressure, defined as adult values equal to or greater than 130/80 mmHg.

The blood pressure values (threshold) that determines hypertension has varied over time and by organization as research has accumulated on the risk associated with elevated blood pressure. The 2017 Hypertension Clinical Practice Guidelines define hypertension as SBP \geq 130 mmHg or DBP \geq 80 mmHg confirmed by a minimum of two measurements on at least two occasions, or use of antihypertensive medicine, or having been told by a health care professional that you have hypertension. The stages of hypertension are presented in [Table 15.5](#). These values apply to both sexes and all races throughout the entire adult lifespan. All stages of hypertension are associated with an increased risk of CVD. The higher the stage of hypertension, the higher the risk of CHD and the more aggressive monitoring and treatment should be. Hypertension is a leading factor in endothelial injury and calcium deposition in the coronary arteries, as well as thickening and stiffening of smaller blood vessels. The elimination of hypertension would result in 30% reduction in cardiovascular disease mortality among men and a 38% reduction in cardiovascular disease death among women (Virani et al., 2021). Hypertension imposes an afterload on the heart, thus increasing ventricular muscle hypertrophy (thickness) and reducing early diastolic filling. Left ventricular hypertrophy independently and powerfully predicts morbidity and mortality and is associated with increased risk for heart failure, ventricular tachycardia, stroke, atrial fibrillation, and the risk of arrhythmia and sudden cardiac death ([Victor, 2012](#)).

TABLE 15.5 Classification of Blood Pressure for Adults

| Category | Systolic (mmHg) | | Diastolic (mmHg) |
|--------------------------|--------------------|-----|---------------------|
| Adults (>19 y) | | | |
| Normal | <120 | and | <80 |
| Elevated | 120–129 | or | <80 |
| Hypertension | 130–139 | or | 80–89 |
| Stage I | ≥140 | or | ≥90 |
| Stage II | | | |

Source: Reprinted with permission from Whelton, P. K., R. M. Carey, W. S. Aronow, D. E. Casey Jr, K. J. Collins, C. D. Himmelfarb, S. M. DePalma, S. Gidding, K. A. Jamerson, D. W. Jones, E. J. MacLaughlin, P. Muntner, B. Ovbiagele, S. C. Smith Jr, C. C. Spencer, R. S. Stafford, S. J. Taler, R. J. Thomas, K. A. Williams Sr, ... J. T. Wright Jr. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 71(6):e13–e115. Copyright © 2017 by the American College of Cardiology Foundation and the American Heart Association, Inc.

The Influence of Exercise Training on Hypertension

Research strongly demonstrates the role of exercise training in the primary prevention, treatment, and control of moderate hypertension. Individuals at high risk for developing hypertension—because of genetics, body composition, primary disease status, or an exaggerated blood pressure response to acute exercise—can reduce their risk through exercise training programs. Longitudinal studies show that high levels of both physical activity and physical fitness are associated with decreased risk of developing hypertension ([ACSM, 2004](#), 2022).

The benefits of exercise training on BP have been reviewed and summarized by the American College of Sports Medicine, which provides strong and convincing evidence of the importance

of exercise/physical activity in the prevention of hypertension and of its protective effects in the treatment of hypertension. The review of the research found a dose-response relationship with more exercise conferring a greater reduction in blood pressure. The analysis also found evidence that aerobic and dynamic resistance exercise training alone or combined were equally effective in lowering BP among adults with normal BP, elevated BP, and hypertension. They also reported that the magnitude of blood pressure reduction is dependent upon baseline blood pressure. Normotensive, those with elevated blood pressure, and hypertensive individuals who performed a 4-month training program had a decrease in SBP of approximately 1 mmHg, 4 mmHg, and 8 mmHg, respectively ([Pescatello et al., 2019](#)). Another meta-analysis that examined the efficacy of exercise training programs compared to medication also found that exercise programs result in a reduction of approximately 7 mmHg reduction in SBP and approximately a 5 mmHg reduction in DBP, which is a smaller reduction than is seen with many medications but still clinically important. The authors conclude that exercise may be a meaningful way to begin to treat elevated blood pressure if it is the preferred course of treatment of the individual (Noone et al., 2020).

Current recommendations are that those with hypertension engage in at least 30 minutes of moderate-intensity aerobic exercise such as walking, jogging, cycling, or swimming on 5–7 d·wk⁻¹. In addition, dynamic resistance exercises is recommended 2–3 d·wk⁻¹ (ACSM, 2022).

Isometric resistance training has also been found to be an effective way to lower both SBP and DBP. Thus, isometric exercise could be used as a training modality to produce clinically meaningful blood pressure reductions or it could be used in addition to aerobic and resistance exercise programs ([Carlson et al., 2014](#)).

Obesity and the Metabolic Syndrome

Obesity is now a common feature of American society. A BMI of greater than 30 kg·m⁻² and a waist girth greater than 102 cm (40 in.) for males and greater than 88 cm (35 in.) for females are

field measures to determine obesity as a risk factor for disease (ACSM, 2022). Body composition measures are fully discussed in the text in [Chapter 7](#). [Figure 15.4](#) reveals that in 2018, less than 30% of U.S. adults were considered normal weight (BMI 18.5–24.9 kg·m⁻²), which equates to having an ideal cardiovascular health metric for BMI (Virani et al., 2021). In contrast, approximately 40% of U.S. adults were obese (BMI ≥ 30 kg·m⁻²), which is defined as a “poor” cardiovascular health metric. Although there have been several years when the prevalence of obesity remained unchanged, the prevalence of obesity is projected to increase and reach 51% by the year 2030.

The relationship between obesity and CVD is both independent of and interrelated to the other cardiovascular risk factors in a disease process known as the metabolic syndrome (Aronson and Rayfield, 2005; Welk and Blair, 2000). The **metabolic syndrome** is a cluster of interrelated risk factors of metabolic origin that directly promotes the development of atherosclerotic CVD and increases the individual’s risk of diabetes and stroke. The predominant underlying risk factors for the metabolic syndrome appear to be abdominal obesity (high amounts of upper body visceral fat) and insulin resistance (Grundy, 2007). Under the stimulation of the enzyme lipoprotein lipase, visceral abdominal adipocytes readily release free fatty acids (FFA) into the circulation. Free fatty acids have two possible fates: (1) they may be transported to the liver, where they are converted to VLDL and ultimately LDL cholesterol or (2) they may be taken up by other cells, including skeletal muscle cells, and oxidized to provide ATP energy by cellular respiration. This enhancement of lipid oxidation may lead to a reduced use of glucose as fuel. At the same time, the increased FFA levels act directly in the liver to inhibit insulin clearance, resulting in hyperinsulinemia. Hyperinsulinemia combined with high levels of blood glucose (hyperglycemia) leads to a reduction in insulin sensitivity. The combination of high glucose and hyperinsulinemia can hasten the development of CVD (see [Figure 15.7](#)). Hyperinsulinemia also increases sodium retention and in susceptible salt-sensitive individuals may precipitate hypertension. Thus, high visceral abdominal obesity is directly related to dyslipidemia (low HDL-C and high triglycerides), reduced glucose tolerance, insulin resistance, hypertension, and

inflammation, which together form the cluster of risk factors for CVD known as the metabolic syndrome (Balagopal et al., 2011; Kannel and Wilson, 1999; Welk and Blair, 2000). Although all major organizations recognize the importance of abdominal (central) obesity, insulin resistance, dyslipidemia, and high blood pressure in the definition of the metabolic syndrome, there remains some differences in levels used to define the metabolic syndrome. The National Cholesterol Education Program Expert Panel's definition includes the presence of any three of the following: (1) abdominal obesity defined as a waist circumference of greater than 102 cm for males and greater than 88 cm for females, (2) plasma TG ≥ 150 mg·dL⁻¹, (3) HDL-C less than 40 mg·dL⁻¹ in males and less than 50 mg·dL⁻¹ for females, (4) BP $\geq 135/85$ mmHg, and (5) fasting glucose ≥ 110 mg·dL⁻¹ (AHA, 2002). Treatment of the "syndrome" is no different from treatment for each of its individual components. However, at the very least, the concept of a syndrome may help the individual realize that all risk factors need to be identified and treated simultaneously (Marks, 2006; Venkat Narayan, 2006).

Metabolic Syndrome A cluster of interrelated risk factors of metabolic origin that directly promotes the development of atherosclerotic CVD and increases the individual's risk of diabetes and stroke.

Complete the [Check Your Comprehension 2](#) exercise to determine your understanding of the metabolic syndrome.

CHECK YOUR COMPREHENSION 2

How is the metabolic syndrome diagnosed?

Check your answer in Appendix C.

The Influence of Exercise Training on Obesity and the Metabolic Syndrome

Exercise training, or fitness level, has a large influence on obesity,

metabolic syndrome, and CVD. **Figure 15.8** presents data from a landmark study showing the relative risk of CVD mortality (adjusted for age, exam year, smoking, alcohol intake, and heredity) by percent body fat (lean, normal, obese) and cardiorespiratory fitness (fit, unfit) in males (Lee et al., 1999). Relative risk indicates how much more likely a group of individuals with the risk factor are to develop the outcome. A value of 1.0 is the baseline for the group without the risk factor; in this case, 1.0 represents lean fit males. Statistically significant numbers higher than 1.0 mean that individuals with that risk factor have a higher “relative risk” of dying from CVD. For example, a relative risk of 3.16 (as for the unfit, lean group) means that these individuals are 316% more likely to die of CVD than individuals in the fit, lean group! Importantly, unfit, lean males had a higher risk of CVD mortality than did fit males in all body fat categories. Unfit obese males had the highest relative risk (4.11). Fit obese males had a lower risk of CVD mortality than did unfit, lean men.

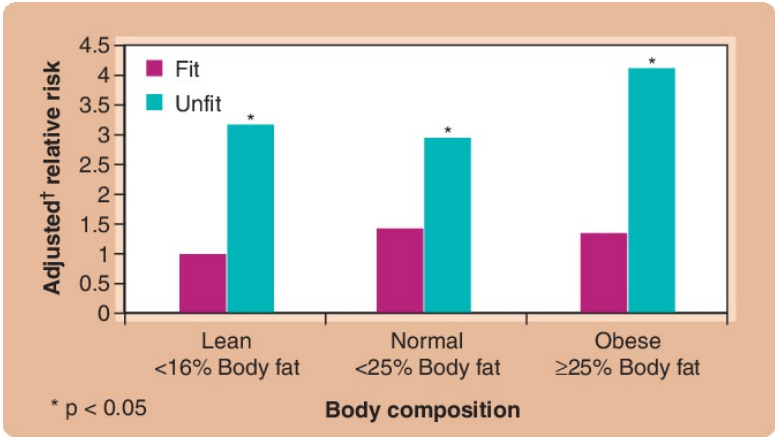


Figure 15.8 Cardiovascular Disease Mortality by Cardiorespiratory Fitness and Percent Body Fat in Males.

†Adjusted for age, exam year, smoking, alcohol, and heredity. **Source:** Based on data from Lee et al. (1999).

The effect of exercise training on visceral and total body

obesity is detailed in the metabolic unit of this text. The effect of exercise training on each of the other risk factors in the cluster of metabolic syndrome is described in the appropriate sections of this chapter. A sedentary lifestyle, poor cardiovascular fitness (Lakka et al., 2003; Mileski et al., 2015), and poor muscular strength (Jurca et al., 2005; Magyari and Churilla, 2012) have all been shown to be associated with the metabolic syndrome. Regular physical activity can improve the metabolic risk profile and reduce the risk of CVD.

Figure 15.9 presents the results of 20 weeks of aerobic training on men and women with the metabolic syndrome. As the figure shows, significant improvements occurred in all risk factors except HDL-C (which often requires more vigorous exercise to change). The overall result was that 30.5% of the participants classified as having metabolic syndrome at the beginning of the exercise training were no longer classified this way at the end of the training (Katzmarzyk et al., 2003). Research continues to identify the optimal training program to improve cardiovascular risk associated with the metabolic syndrome (Churilla et al., 2012; Ciolac et al., 2010; Pattyn et al., 2013; Stensvold et al., 2010; Strasser et al., 2010). Cardiorespiratory training reduces several metabolic syndrome risk factors. The magnitude of the effect of exercise on risk factors varies based on the training program employed and may not be consistent among risk factors (ACSM, 2011).

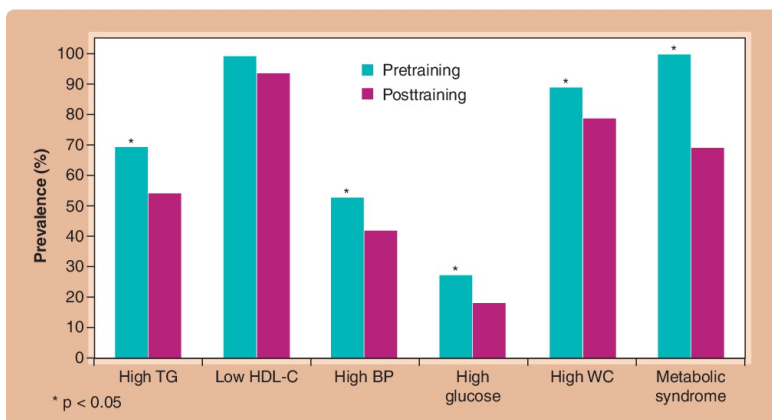


Figure 15.9 Effect of Aerobic Training on Individual

Risk Factors and the Metabolic Syndrome.

TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; BP, blood pressure; WC, waist circumference.

Source: Reprinted with permission from Katzmarzyk, P. T., A. S. Leon, J. H. Wilmore, J. S. Skinner, D. C. Rao, T. Rankinen, & C. Bouchard: Targeting the metabolic syndrome with exercise: Evidence from the Heritage Family Study. *Medicine & Science in Sports & Exercise*. 35(10):1703–1709 (2003). Copyright ©2003 The American College of Sports Medicine.

A significant dose-response relationship has been observed between cardiorespiratory fitness and mortality in males with metabolic syndrome. In one study of 3,757 men with metabolic syndrome, men in the middle and lower tertiles of cardiorespiratory fitness had 2.08 and 3.48 times the risk of CVD mortality than did the men in the upper tertile of CVD fitness (Katzmarzyk et al., 2004). Another study categorized men into low (lowest 20%), moderate (middle 40%), and high (highest 40%) cardiorespiratory fitness based on age and treadmill test results. For any given level of waist circumference, visceral fat, or subcutaneous fat, the high-fitness group had lower TG levels and higher HDL-C than did either the moderate- or low-fitness groups. The relative risks of having the metabolic syndrome were 1.8 and 1.6 times higher in the low- and moderate-fitness groups than in the high-fitness group after adjusting for age, visceral fat, and subcutaneous fat (Lee et al., 2005). Collectively these, and many other studies, indicate that regular exercise is important in the prevention of and treatment of metabolic syndrome.

In general, physical activity, exercise training, and cardiovascular-respiratory fitness bring about beneficial changes that limit the progression of metabolic syndrome, with or without changes in total body weight and composition (Carroll and Dudfield, 2004; Welk and Blair, 2000).

Physical Inactivity

In the last three decades, emphasis has shifted from the product

of physical fitness (typically defined as cardiorespiratory fitness as measured by $\dot{V}O_2 \text{ max}$) to the behavior of physical activity to achieve health benefits. There is, of course, evidence that both *physical fitness* and *physical activity* lead to health benefits and decreased negative health outcomes. The physical activity and public health recommendations (ACSM, 2011; U.S. Department of Health and Human Services, 2018) suggest that adults from 18 to 65 years participate in at least 30 minutes of moderate aerobic activity 5 days of the week (for a total of 150 minutes) or vigorous aerobic activity for 20–60 minutes 3 d·wk⁻¹ (for a total of 75 minutes). These guidelines also point out that additional health benefits accrue as activity levels increase. Such activities must be in addition to routine activities of daily living that are of light intensity such as casual walking or grocery shopping. However, moderate or vigorous activities performed as part of daily life such as brisk walking or heavy manual labor can be counted toward the activity recommendations. In addition, muscle strengthening activity consisting of 8–10 exercises involving the major muscle groups for 8–12 repetitions, 2–3 d·wk⁻¹ should be included. Individuals not participating in a regular exercise program or not meeting these minimal physical activity guidelines are considered to have the risk factor of a sedentary lifestyle (ACSM, 2022). It was thought that it would be easier to motivate the public to accumulate lifestyle moderate activity than to undergo the regimented vigorous exercise usually prescribed for physical fitness enhancement. Therefore, the risk factor for CVD is labeled *physical inactivity* rather than *lack of physical fitness*. As seen in **Figure 15.4**, only approximately 40% of individuals meet the criteria for an ideal cardiovascular health metric for physical activity (Virani et al., 2021). **Figure 15.10** documents the prevalence of individuals who meet physical activity guidelines among men and women of different racial and ethnic backgrounds. Furthermore, within each ethnic group, women are less likely to meet current guidelines than are men.

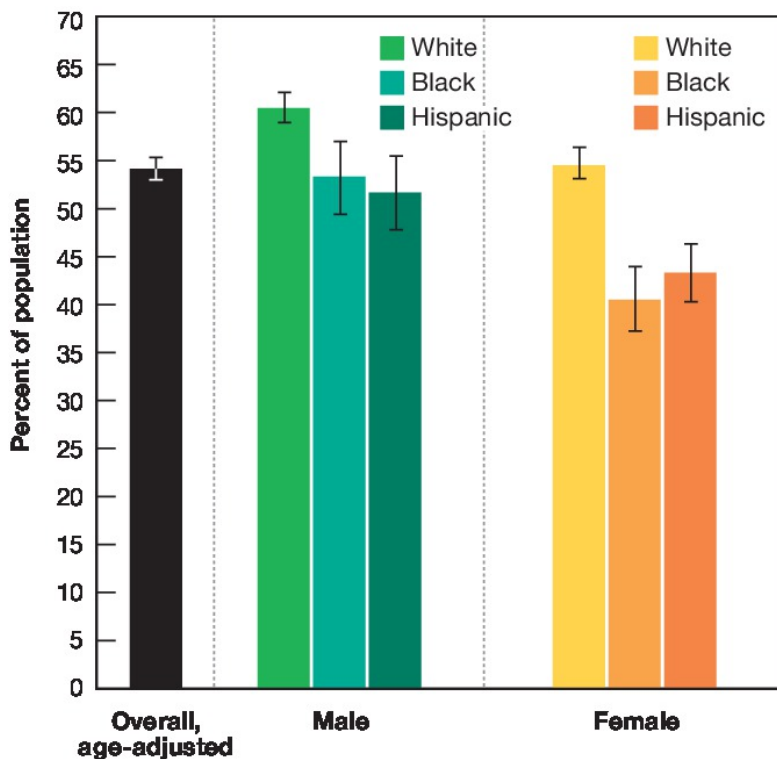


Figure 15.10 Prevalence of Adults Meeting Aerobic Activity Guidelines by Race/Ethnicity and Sex.

(“Meeting aerobic guidelines” was defined as engaged in moderate aerobic leisure-time physical activity for at least 150 min-wk⁻¹ or vigorous aerobic activity for at least 75 min-wk⁻¹ or an equivalent combination.) **Source:** Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021). Copyright © 2021

Figure 15.11 presents findings from a single study investigating cardiovascular risk reduction based on total physical activity. In this study, the third quintile (Q3) represents the level of activity then recommended in the Surgeon General’s Report ([U.S. Department of Health and Human Services, 1996](#)), that is, the equivalent of walking briskly for 30 min·d⁻¹, 5 d·wk⁻¹. Note that the baseline relative risk of 1.0 represents the relative risk of those with no physical activity (Q1) and that relative risk values less than 1.0 represent a lower risk with more activity. Participants in this study were postmenopausal females. As the figure shows, women with the recommended level of activity had a 19% lower risk for CVD than did those who engaged in no physical activity. Those who engaged in higher amounts of activity had even more reduction in risk, 22 and 28% in Q4 and Q5, respectively. Those with less than the recommended level of total activity had no significant benefit. Results were similar whether the women were normal weight, overweight, or obese and whether their race was black or white ([Manson et al., 2002](#)).

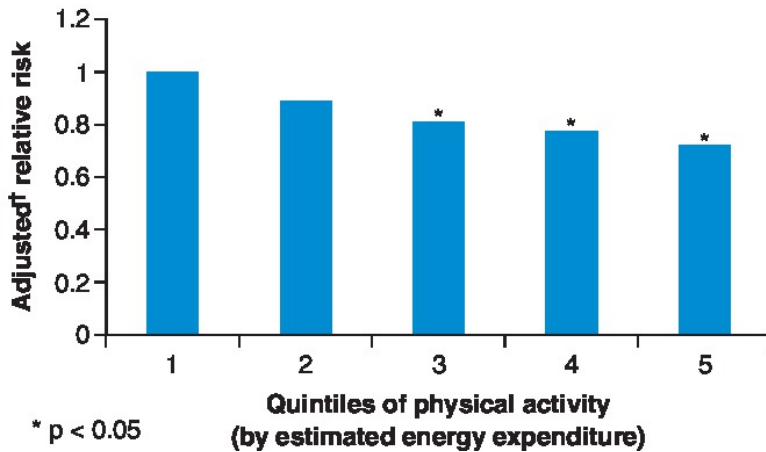


Figure 15.11 Relative Risk of Cardiovascular Disease by Quintile of Physical Activity.

†Adjusted for age, personal habits, socioeconomic factors, heredity, and body composition. **Source:** Based on data

from [Manson et al. \(2002\)](#).

Figure 15.12 is a composite graph of the effects of *physical activity* and *physical fitness* on relative risk of heart disease based on a meta-analysis of eight studies of physical fitness and 30 studies of physical activity. This graph reveals that (1) the relative risk of CHD/CVD decreases linearly with increasing amounts of physical activity; (2) the risks of CHD/CVD drop precipitously before the 25th percentile of physical fitness and then parallel the slope of the decline in physical activity; and (3) a significant difference in risk reduction is associated with being more physically fit than physically active ([Williams, 2001](#)). The author suggested that this might indicate that lack of physical fitness and physical inactivity are separate, distinct risk factors for CVD, and that both physical fitness and physical activity should be measured and interventions followed if warranted. One of the issues is that physical fitness can be measured more accurately than physical activity, and this could at least partially explain the results. Whether the difference in risk reduction between physical fitness and physical activity is true or a measurement artifact needs more study. It is clear, however, that at least moderate physical activity should be undertaken by all, but that the attainment of physical fitness is certainly not to be discouraged once individuals have achieved the minimal level of physical activity ([Plowman, 2005](#)). Current recommendations explicitly encourage vigorous physical activity for those who are willing and able to do it ([ACSM, 2011, 2022](#); [USHHS, 2018](#)).

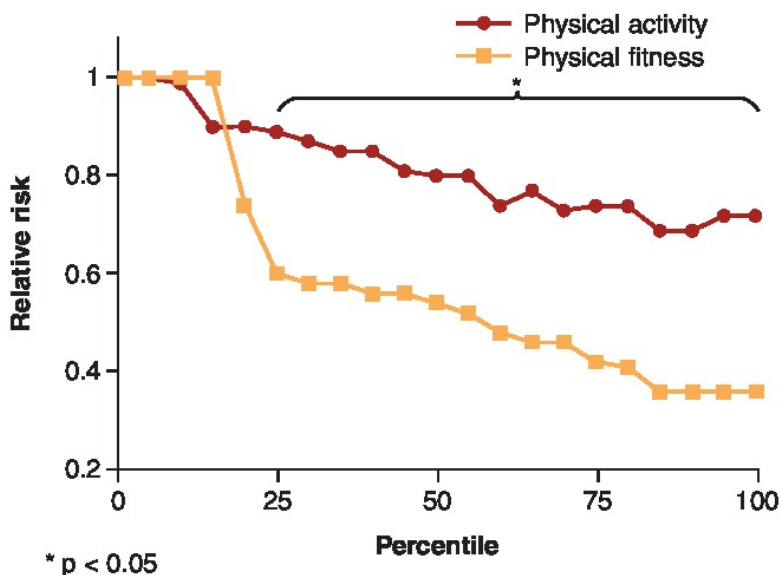


Figure 15.12 Effects of Physical Fitness and Physical Activity on Heart Disease Risk Factors.

Source: Reprinted with permission from Williams, P. T.: Physical fitness and activity as separate heart disease risk factors: A meta-analysis. *Medicine & Science in Sports & Exercise*. 33(5):754–761 (2001). Copyright ©2001 The American College of Sports Medicine.

The degree of risk for those who are physically inactive, about twice that of those without this risk factor, is approximately the same as the risk associated with systolic hypertension, cigarette smoking, and hyperlipidemia. As has been discussed in the sections for each of these other risk factors, physical activity positively impacts the risk from these as well. Given the high prevalence of physical inactivity and the benefits derived from becoming more physically active, potentially more benefit overall could be achieved by increasing the activity level of U.S. citizens than by changing any other single risk factor (AHA, 2007). One of the most important facts about activity or exercise training is that the greatest health benefits occur when very sedentary individuals increase their endurance activity levels even minimally (Blair and Minocha, 1989; USHHS, 2018).

The mechanisms by which physical activity achieves its protective effect are many and varied. Some are independent and a direct result of the adaptive changes that occur in the cardiovascular system and its neural control, as were documented earlier in this unit. For example, the increases in parasympathetic tone and decreases in sympathetic response reduce the resting and exercise heart rate in physically active individuals and possibly influence hypertension. These changes also enhance the electrical stability of the myocardial cells, reducing the risk of potentially fatal conduction system defects and coronary vessel spasms. Physical activity also decreases oxidative stress and improves endothelial function. Obviously, participating in physical activity directly eliminates physical inactivity as a risk factor. The impact of physical activity or exercise training on all of the major modifiable risk factors is summarized in [Table 15.6](#).

TABLE 15.6 Summary of Impact of Physical Activity or Exercise Training on Modifiable CHD Risk Factors

| Risk Factor | Impact of Exercise Training |
|---------------------------|--|
| Cholesterol-lipid factors | ↑ HDL-C fraction ↓ TC |
| Cigarette smoking | Indirect |
| Hypertension | ↓ SBP; ↓ DBP (incomplete normalization) |
| Physical inactivity | Directly eliminates |
| Diabetes mellitus | ↑ glucose tolerance ↑ insulin sensitivity |
| Obesity | ↓ body fat ↓ visceral abdominal fat |

Note: The impact is the same for children, adolescents, and adults.

↑, increased; ↓, decreased.

Another research approach to understanding the impact of

physical activity on health is to consider sedentary behavior or lack of physical activity on cardiovascular health (the other end of the physical activity spectrum). Research studies have provided evidence that excessive sedentary behavior has implications for health and that too much sedentary behavior is distinct from too little exercise or too little physical activity. **Sedentary behavior** is defined as any waking behavior that expends ≤ 1.5 METs of energy often, but not always, stipulated as being in a reclining or sitting posture (Gibbs et al., 2015). For example, prolonged sitting time in transit (driving/riding in a car), at work (computer/desk tasks), at home, and in leisure time (television watching/computer activities). Observational epidemiological studies strongly suggest that excessive daily sitting time, limited standing, infrequent getting up and down from a chair, and/or minimal incidental walking may have a significant direct relationship to mortality, CVD, type 2 diabetes, metabolic syndrome, obesity, depression, increased waist circumference, hypertension, depressed lipoprotein lipase activity, blood glucose, insulin, and lipoproteins. A 2013 meta-analysis (Chau et al., 2013) found a dose-response relationship between hours of sitting and the risk of all-cause mortality. There was a 52% higher mortality risk for adults sitting ≥ 10 hr·d⁻¹ when physical activity was not taken into account and a 34% higher mortality risk after taking moderate-to-vigorous physical activity into account. Some previous studies had found that meeting physical activity guidelines was not sufficient to counteract the effects of excessive sitting (Katzmarzyk et al., 2009; Owen et al., 2009, 2010). This meta-analysis, however, suggests that moderate-to-vigorous physical activity had a partially protective effect especially when sitting time was high. As always, for public health purposes, it is important to determine what level of exercise/activity is needed to counteract sitting inactivity. Note that standing, although at an MET level below 1.5, is not specified in the definition of sedentary behavior. Indeed, standing has been proposed as a feasible and promising strategy to reduce sitting time and hence risk. Recent studies on large numbers of both Canadian and Australian adults have found that greater time spent standing was associated with a lower risk of mortality, especially in those who were physically inactive (Katzmarzyk, 2014; van der Ploeg et al., 2014). A study of older

Americans found that in less active individuals (<2 hr·d⁻¹) replacing 1 hr·d⁻¹ of sitting with an equal amount of either purposeful exercise or nonexercise activities (e.g., household chores, lawn and garden work, shopping) was associated with lower all-cause mortality and cardiovascular mortality. For more active individuals (>2 hr·d⁻¹), replacement of sitting time with purposeful activity, but not nonexercise activity, was associated with lower mortality (Matthews et al., 2015). Thus, the baseline activity status of the individual appears to be critical in determining whether purposeful or nonexercise physical activity is needed to reduce the adverse biological effects of excessive sitting. Both common sense and research data suggest that it is prudent to try to minimize prolonged sitting with frequent breaks every hour to walk around, stretch, or do some activity of daily living (ACSM, 2011; Ekelund et al., 2016). If you have been diligently sitting and studying for at least an hour, it is time for you to take such a short break.

Sedentary Behavior Any waking behavior that expends ≤ 1.5 METs of energy often, but not always, stipulated as being in a reclining or sitting posture.

Complete the **Check Your Comprehension 3—Case Study 2** evaluation to determine your understanding of the major modifiable risk factors for CHD.

Check Your Comprehension 3—Case Study

Vivian is a 63-year-old female with the following characteristics:

WT = 76.4 kg

Waist circumference = 95 cm; hip circumference = 103 cm

RBP seated = 124/82 mmHg; RHR seated = 80 b·min⁻¹

Total cholesterol = 280 mg·dL⁻¹; HDL-C = 34 mg·dL⁻¹

Glucose (fasting) = 116 mg·dL⁻¹ and 118 mg·dL⁻¹

Her father died of a myocardial infarction at 52 years of age;

her mother is alive. Vivian cares for her mother night and day. She has felt chest pain when moving her mother, doing housework, or walking briskly. She smoked two packs of cigarettes per day until 1 month ago. Vivian feels under pressure and claims to have no time to exercise. Recently, she went to her physician and was diagnosed with “walking pneumonia.” She is taking an antibiotic and a daily multivitamin.

Complete the following CVD risk analysis for Vivian:

Name the six major modifiable risk factors.

Provide cutoff numerical values including units for each major modifiable risk factor.

Provide values for Vivian from her history, and determine whether she has each risk factor.

How many total risk factors for CVD does Vivian have?

Check your answer in Appendix C.

Contributing and Selected Nontraditional Risk Factors

The contributing and nontraditional risk factors are risk factors that may predict CVD but that are not yet considered major risk factors because of a lack of evidence from large-scale studies, because of the infeasibility of measuring these factors, in large numbers of people, or because they do not significantly increase the predictability of CVD disease beyond the traditional risk factors. There are many potential nontraditional risk factors, and some of these may play a role by being mediators of other risk factors or of the disease progression itself. Several common contributory nontraditional risk factors include CRP, apolipoproteins (discussed earlier), clotting and clot dissolving factors, and stress.

C-reactive protein (CRP) is a marker of systemic inflammation, tissue damage, and infection. Inflammation plays a central role in atherosclerotic plaque development and in plaque rupture. As described previously, the early phase of atherosclerosis is characterized by an inflammatory response in

the arterial wall. Research suggests that plaque rupture, leading to clot formation, is also associated with inflammation. A study using autopsy data found that individuals who suffered a plaque rupture had a higher CRP than did those who died of nonvascular causes (Burke et al., 2002). One of the major dangers of atherosclerotic plaque is that plaque rupture would lead to clot formation that could block an already narrowed blood vessel. Fibrinogen is a protein present in blood plasma that, under certain physiological circumstances, is converted into fibrin threads that form the basis of a blood clot. Fibrinolytic activity refers to the breakdown of fibrin clots. Enhanced fibrinolytic activity can reduce the risk of clots and thus the risk of CHD. Stress (see Chapters 1 and 21) is widely believed to play a role in CVD progression but identifying the extent of the relationship or the mechanism by which stress impacts CVD is complicated by difficulties in differentiating and quantifying different types of stress. Many factors affect how stress impacts an individual, and these factors are difficult to identify and measure. How much stress any given stressor causes depends not just on the stressor but also on characteristics of the individual. For example, what vulnerabilities or, conversely, what coping strategies does the individual have? The response to the stressor varies according to the individual's psychological and physiological level of reactivity (Childs et al., 2014). For example, among the acute physiological responses to fear or anger, mediated through the neural and hormonal systems, are increases in heart rate, blood pressure, respiratory rate, and blood viscosity and decreases in clotting time and the breakdown of fats used as a fuel.

Children and the Cardiovascular Risk Factors

The concern about CVD risk factors in children is not that children exhibit clinical CVD but that atherosclerotic CVD is a lifelong process that begins in childhood. Therefore, if risk factors can be prevented, modified, or counteracted in childhood, it may be possible to prevent or at least delay or reduce the severity of cardiovascular problems in adulthood.

In relation to physical activity, the intent is to establish patterns of participation in children that will continue throughout adulthood. To a large extent, the success of this strategy depends on a phenomenon called tracking. In this context, **tracking** means that a characteristic is maintained, in terms of relative rank, over a long time span or even a lifetime. An example of tracking a nonrisk factor is height. Children in the upper percentiles of height at a very young age tend to maintain that relative position and be taller than average adults. Thus, in relation to children, it is important to determine the presence or absence of modifiable risk factors, the tracking strength of risk factors, and the impact of physical activity and exercise training on both short-term risk factor reductions and long-term lifestyle modifications (Rowland, 1991; Rowland and Freedson, 1994).

Tracking A phenomenon in which a characteristic is maintained, in terms of relative rank, over a long time span or even a lifetime.

FOCUS ON APPLICATION | *Clinically Relevant*

Influence of Physical Activity on Mortality in Elderly with Coronary Artery Disease

This chapter describes the inverse relationship between physical activity and mortality from coronary heart disease and the positive impact of exercise training on each cardiovascular disease risk factor. The following study specifically investigated the dose-response relationship between physical activity and the relative risk of mortality in patients with coronary artery disease in an observational cohort study.

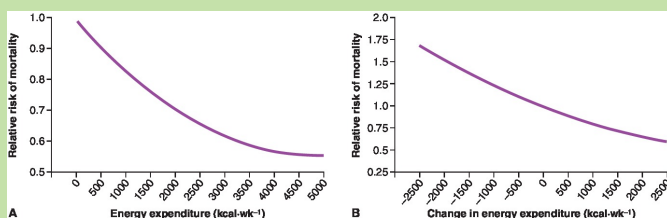
Janssen and Jolliffe (2006) studied the dose-response relationship between baseline physical activity level and all-

cause mortality risk over a 9-year period in 1,045 men and women with coronary artery disease. Participants were stratified based on sex, age, smoking, adiposity, self-perceived health, number of risk factors, and coronary artery disease subtype so that the relationship between physical activity and mortality could be examined within each strata. Participants returned for a 3-year follow-up examination. A separate analysis was performed on a subset of participants ($n = 785$) who had changed their physical activity level (from baseline to the 3-year follow-up) to determine the relationship between changes in physical activity over the initial 3-year follow-up and mortality risk over the subsequent 6-year period.

The dose-response curve shown in panel A depicts the relationship between energy expenditure and relative risk of mortality for the entire group. The data are adjusted for age, sex, race, smoking, alcohol consumption, socioeconomic status, adiposity, prevalent disease, and type of coronary artery disease. As seen in other studies, the relative risk of mortality decreased with increasing levels of activity (expressed as energy expenditure). The data were then examined by strata. Independent of age, sex, smoking, adiposity, self-perceived health status, number of risk factors, or type of coronary artery disease, the relative risks of mortality were significantly lower in the active participants ($>1,500 \text{ kcal}\cdot\text{wk}^{-1}$) compared to inactive participants ($<1,500 \text{ kcal}\cdot\text{wk}^{-1}$). However, even those individuals who accumulated only approximately $1,000 \text{ kcal}\cdot\text{wk}^{-1}$ had a 19% reduction in mortality risk, again showing that some exercise is always better than no exercise.

The dose-response curve shown in panel B depicts the relationship between change in energy expenditure (from baseline to the 3-year follow-up) and relative risk of mortality at the study completion (9 years). Again, the data are adjusted for age, sex, race, smoking, alcohol consumption, socioeconomic status, adiposity, prevalent disease, and type of coronary artery disease. Clearly, the risk of mortality was higher for individuals whose physical activity level decreased and lower for those whose physical activity level increased.

The practical implications of this study are immense. This study reinforces the common finding of an inverse relationship between physical activity and mortality risk and clearly demonstrates that in patients with coronary artery disease, regardless of age, sex, severity of disease, or other risk factors, there is a protective benefit to higher levels of physical activity. Finally, in a group of patients with coronary artery disease, an increase in physical activity level was associated with reduction in mortality risk. The bottom line is that physical inactivity increased mortality risk in study participants regardless of whether they were men or women, old or very old, smokers or nonsmokers, lean or overweight, or otherwise healthy or unhealthy. The take-home message is that physical activity should be encouraged for the vast majority of people, including coronary artery disease patients.



Source: Reprinted with permission from Janssen, I., & C. J. Jolliffe: Influence of physical activity on mortality in elderly with coronary artery disease. *Medicine & Science in Sports & Exercise*. 38(3):418–423 (2006). Copyright ©2006 The American College of Sports Medicine.

Cholesterol-Lipid Fractions

At birth, total cholesterol levels are approximately 70 mg·dL⁻¹, with 35 mg·dL⁻¹ of that total being HDL. During the first few weeks, the level of TC rises rapidly to between 100 and 150 mg·dL⁻¹. By 2 years and until adulthood, the average value for males is about 160 mg·dL⁻¹ and for females about 165 mg·dL⁻¹, with HDL-C levels between 50 and 55 mg·dL⁻¹. At puberty,

males show a decline of HDL-C and females a decline in LDL-C values. In general, cholesterol levels do track from childhood to adulthood, although not all children with high juvenile levels of cholesterol will have elevated adult levels. In one study, 43% of children with cholesterol values above the 90th percentile also had adult values above the 90th percentile, and 81% had values above the 50th percentile (Armstrong and Simons-Morton, 1994; Mahoney et al., 1991). Figure 15.13 depicts blood lipid profiles of youths using data collected through 2016.

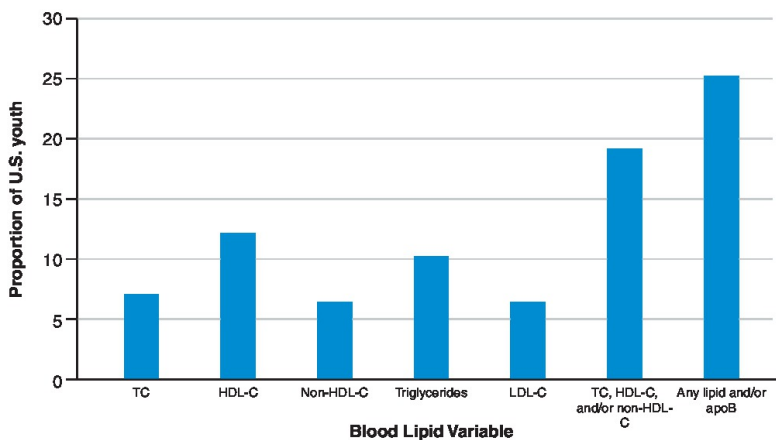


Figure 15.13 Prevalence of Abnormal Lipids among Adolescents.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021). Copyright © 2021 American Heart Association, Inc.

Values that represent acceptable, borderline, and high-risk

levels for children are included in [Table 15.4](#). In adults, high TC, high LDL-C, and low HDL-C levels have been found to relate to the magnitude of early atherosclerotic lesions. Autopsy reports have confirmed that early signs of atherosclerosis are present in children as young as 3 years and are frequently evident by age 10. Poor lipid profiles often run in families because of both genetics and shared lifestyle factors.

Cross-sectional studies of the relationship between physical activity and lipid levels have reported that active youngsters have higher HDL-C values and lower TG than do inactive youngsters, but TC levels and LDL-C that do not differ with activity status. However, the relationship between physical fitness and lipid risk factors is stronger than that of physical activity and these factors. Studies indicate that high levels of cardiorespiratory fitness for both boys and girls and/or muscular fitness in girls are associated with a more favorable lipid-metabolic profile ([Garcia-Artero et al., 2007](#); [Janssen and LeBlanc, 2010](#); [Ortega et al., 2008](#)). Low cardiorespiratory fitness in childhood and adolescence is a predictor of abnormal blood lipids later in life ([Ruiz et al., 2009](#)). Training studies, including clinical and school-based programs, have shown beneficial effects for HDL-C and TG if the intervention has been sufficient to also improve aerobic fitness but not for TC or LDL-C. However, a study that compared a 12-week training program for adolescent swimmers and soccer players to a control group of nonathletic but physically active contemporaries found that the soccer group decreased LDL-C levels compared to the other two groups. A minimum of 40 min·d⁻¹, 5 d·wk⁻¹ of at least moderate to vigorous activity over 4 months is apparently needed to achieve beneficial effects in lipid levels in children and adolescents ([Andersen et al., 2011](#); [Mountjoy et al., 2011](#); [Strong et al., 2005](#)).

Cigarette Smoking

Figure 15.14 reports the prevalence of tobacco use among high school students by product, race/ethnicity, and sex. The highest prevalence of student smoking occurs in males, regardless of race ([Virani et al., 2021](#)). Approximately 80% of adult smokers started smoking before age 18 ([AHA, 2007](#)); smoking definitely tracks from adolescence to adulthood. The physiological effects of

smoking and the indirect relationship between activity and smoking are the same regardless of the age of the smoker. There has been a sharp increase in the use of electronic cigarette among adolescents, increasing from 1.5 to 27.4% between 2011 and 2019 with electronic cigarettes now being the most commonly used tobacco product for adolescents (Virani et al., 2021).

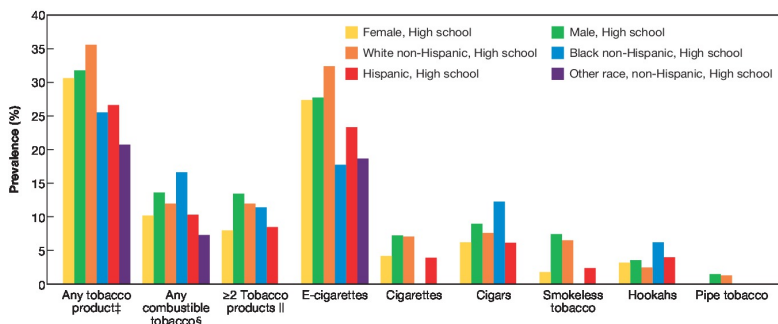


Figure 15.14 Prevalence of Different Tobacco Products by High School Students by Race/Ethnicity and Sex.

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One study of Finnish youth showed that over a 6-year span, with individuals who were 12, 15, and 18 years old at the beginning of the study, higher activity levels were associated with less smoking in both boys and girls. Almost half of those who were sedentary smoked, but only about 10% of those who were active smoked (Raitakari et al., 1994). A 2011 study (Horn et al., 2011) investigated the effects of physical activity on teen

smoking cessation and found that those in a smoking cessation plus physical activity group had significantly higher cessation rates than did those in the smoking cessation program alone. While physical activity can assist in smoking cessation, it remains a primary goal of physical education and exercise science professionals to encourage an active lifestyle and discourage the onset of smoking in children and adolescents.

Diabetes Mellitus

Children with type 1 and type 2 diabetes are at risk of prematurely developing atherosclerosis. Furthermore, diabetes tracks into adulthood. The incidence of type 2 diabetes mellitus has risen dramatically in children and adolescents in recent years—in large part paralleling the incidence in obesity ([Figure 15.15](#)). Furthermore, among U.S. adolescents (12–19 years), the prevalence of prediabetes is approaching 30% of the population (Menke et al., 2016).



Figure 15.15 Unhealthy Food Choices Contribute to the High Level of Childhood Obesity.

There is growing evidence of a positive relationship between physical activity (both aerobic and resistance) and insulin sensitivity independent of adiposity status or changes in children and adolescents. More physical activity results in improved insulin sensitivity (and hence reduced insulin resistance) in both type 1 and type 2 diabetes (Chimen et al., 2012; Kim and Park, 2013). However, even if positive changes in adiposity are not required to reap insulin sensitivity changes, the early establishment and maintenance of activity can benefit both glucose/insulin variables and body fatness (Bunt et al., 2003; Rowland, 2006). It may be that regular, high-intensity physical activity is needed for both cardiorespiratory fitness and physical activity to show associations with insulin sensitivity as the relationship between physical activity and insulin sensitivity is somewhat stronger and more consistent than that for cardiovascular fitness (Berman et al., 2012). Results are inconsistent for the influence of physical activity and cardiovascular fitness on glycemic control in type 1 and 2 diabetes (Chimen et al., 2012; Liese et al., 2013), although a 2014 meta-analysis of 10 studies reported an improvement in glycemic control from systematic physical activity in those with type 1 diabetes (MacMillan et al., 2014). Regular physical activity has beneficial effects on the presence of glucose in the blood in type 1 diabetes without increasing the risk of hypoglycemia (Chimen et al., 2012; Herbst et al., 2006). The importance of and cautions related to physical activity and exercise training for individuals with diabetes were discussed earlier in the section on adults but also apply here for younger individuals. The optimal exercise prescription or recommended activity level has yet to be determined for reducing insulin resistance and improving insulin sensitivity (Chimen et al., 2012; Kim and Park, 2013).

Hypertension

Hypertension is evident in children as young as 6 years of age. In children, blood pressure varies based on age and height. However, there are insufficient data to identify a specific level of blood pressure associated with adverse cardiovascular outcomes in adulthood. Thus, blood pressure categories are often based on age and height, and normative data are used to define normal,

elevated, and stage 1 or stage 2 hypertension (Flynn et al., 2017). For children over 13 years of age, adult blood pressure values are used to define hypertension. Recent data suggest that only 40% of children met the ideal cardiovascular health metric for blood pressure, a troubling statistic given the negative health consequences of hypertension (Virani et al., 2021).

Although childhood blood pressures do not predict adult blood pressures per se, there is a definite tendency for both systolic and diastolic blood pressures to track from childhood to adulthood. In one study, children with blood pressures at the 90th percentile had three times the risk of having high adult systolic blood pressure and twice the risk of having high adult diastolic blood pressure as those whose childhood values were at the 50th percentile (Mahoney et al., 1991). In another study, participants with self-reported adult hypertension (at age 47 years) were found more likely to have had high blood pressure and adiposity by age 10 years and abnormal blood lipids and glucose by age 16 years (Urbina et al., 2019). There is strong evidence indicating that cardiorespiratory fitness in childhood and adolescence is a predictor of high blood pressure later in life (Ruiz et al., 2009). In addition, there is a dose-response relationship between aerobic fitness and blood pressure in youth. That is, the least fit boys and girls have been shown to be more likely to have hypertension than their high-fit peers. This relationship is strongest in overweight children (Andersen et al., 2011). A similar inverse relationship has been found between at least moderate physical activity and avoidance of hypertension (Gopinath et al., 2011; Wellman et al., 2020).

Physical activity intervention of at least 30 minutes, three times per week with intensity sufficient to increase aerobic fitness, has been shown to reduce blood pressure in both boys and girls with essential hypertension (Andersen et al., 2011; Janssen and LeBlanc, 2010; Mountjoy et al., 2011). As with adults, the reductions that do occur rarely result in normal blood pressure levels, and regular physical activity or exercise is necessary to maintain the beneficial results. Dynamic resistance training used by itself, in one study, did not bring about a reduction in either systolic or diastolic pressure. However, when weight training was instituted after a period of aerobic endurance training that resulted in reduced blood pressures, the reduction in

blood pressure was maintained with just the weight training (Andersen et al., 2011). Therefore, as with adults, the best procedure is to begin young individuals with hypertension on a dynamic endurance activity regimen and add resistance training several months later, if desired (Alpert and Wilmore, 1994). There is neither a need for nor any evidence of a reduction in blood pressure in normotensive children and adolescents with exercise training (ACSM, 2004; Mountjoy et al., 2011; Rowland, 2006; Strong et al., 2005).

Overweight and Obesity

As seen in **Figure 15.16**, more than 13% of children who are 2–5 years are obese, and this percentage increases with age. There is a strong socioeconomic impact on the obesity prevalence in adolescents—obesity prevalence is decreasing in adolescents from high socioeconomic status but continues to rise in adolescents from low socioeconomic status. Unfortunately, overweight and obesity show evidence of tracking. An estimated 70% of overweight adolescents have a 70% chance of becoming overweight adults. This increases to an 80% chance if one or both parents are overweight or obese (Roger et al., 2011). Approximately 80% of obese adolescents become obese adults.

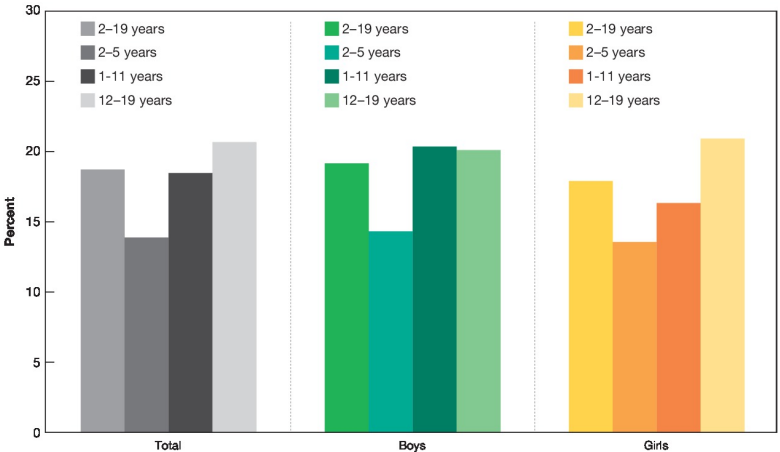


Figure 15.16 Prevalence of Obesity among U.S. Children and Adolescents by Age.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021).
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There is strong evidence that body composition in childhood and adolescence is a predictor of adult CVD risk factors. There is also strong evidence that high body mass index (BMI) in childhood and adolescence increases the risk of early adult death ([Eisenmann et al., 2005](#); [Ruiz et al., 2009](#)). Furthermore, fatness has been determined to be a mediator of the relationship between physical activity and several other risk factors ([Gutin and Owens, 2011](#)). A significant relationship between adolescent cardiorespiratory fitness and adult body fatness has been demonstrated. The deleterious consequences of high fatness can to a meaningful extent be counteracted by having high levels of cardiorespiratory fitness ([Ortega et al., 2008](#)).

As for adults, the relationship between exercise training and reduced body weight and body fat is detailed in the metabolic unit of this text for children and adolescents (see [Chapter 8](#)). A few brief comments are appropriate here, however. Research suggests that *prevention* of excess fatness in youths is primarily dependent on an adequate volume of vigorous-intensity exercise. Youths of both sexes who participate in moderate to vigorous aerobic physical activity have less body fat than do those who do not, but vigorous physical activity is associated more with lower levels of fatness than is moderate physical activity. That is, there is a dose-response relationship between physical activity and obesity. Resistance exercise may also elicit favorable changes in body composition ([Dietz et al., 2012](#); [Gutin and Owens, 2011](#); [Janssen and LeBlanc, 2010](#); [Ruiz et al., 2009](#)). Exercise training of

moderate intensity 30–60 min·d⁻¹, 3–7 d·wk⁻¹ reduces total body and visceral fat in overweight children and adolescents. Limited evidence suggests that more intensive and longer-duration (>80 min·d⁻¹) exercise training is needed to reduce the body fat percentage in normal weight youths of both sexes (Strong et al., 2005). A combination of aerobic and resistance training may be more effective than either aerobic or resistance training alone in reducing total body fat and waist circumference (Sigal et al., 2014). Although beneficial changes often occur in body composition in obese youngsters, decreases in body weight and body mass index are not consistently found (Watts et al., 2005).

Obese children have abnormalities in vascular function and structure and exhibit biochemical markers of inflammation (Kapiotis et al., 2006). Research shows that 60% of overweight children between the ages of 5 and 10 years have at least one other major CV risk factor (Centers for Disease Control and Prevention, 1999). The clustering of risk factors known as the metabolic syndrome is evident in children and adolescents with prevalence varying by region of the country: Children in the northeast and western parts of the United States have lower rates of metabolic syndrome (about 6%) compared to those in the Midwest (about 11%) (Virani et al., 2021). Both physical fitness and physical activity are inversely related to metabolic syndrome risk factors. Furthermore, an interaction between physical activity and physical fitness suggests a stronger relationship between activity and metabolic risk factors in those with low cardiorespiratory fitness. Thus, children and adolescents with the lowest levels of fitness should benefit most from increasing their physical activity (Andersen et al., 2011; Brage et al., 2004; Steele et al., 2008; Wedderkopp et al., 2003). Several exercise studies have shown improvements in specific elements of metabolic syndrome in both obese and nonobese youth (Kamal and Ragy, 2012; Strong et al., 2005).

Physical Inactivity

Neither lifestyle physical activity nor exercise training is likely to alter cardiovascular risk factor variables in children or adolescents whose values for these variables are normal.

Maintenance of normal values, however, can be assisted by activity. Abnormal levels are consistently and positively impacted by physical activity or exercise interventions—thus deterring the continued development of that risk factor and establishing a lifestyle pattern for adulthood (Rowland, 2006). For example, significant tracking of physical activity was observed in a Finnish study of both boys and girls, with 44% remaining active from age 12 to 18 years, from 15 to 21 years, and from 18 to 24 years. Physical inactivity tracked even more closely, with 57% remaining inactive over the 6 years of the study. Those who remained active had better risk factor profiles than did their inactive counterparts. They smoked less, were less fat, had higher HDL-C and lower TC values, and had favorable differences in insulin levels. They also had a healthier diet, which would, of course, have affected these results (Raitakari et al., 1994). Another study showed that physically active young male adults had had better physical fitness test results as children or adolescents (Dennison et al., 1988).

Twisk et al. (2002) summarized the results from five longitudinal observational studies investigating relationships between physical activity and cardiorespiratory physical fitness in youth and CVD risk factors later in life. Childhood physical fitness was found to be predictive of a healthy CVD risk profile later in life, while physical activity was not. As with adults, the difficulty of accurately measuring physical activity may be partially responsible for these results. It may also be that physical fitness more directly indicates the quality of the cardiorespiratory system or that the predictive value of physical fitness may be caused by the relationship between physical fitness and body fat percentage. Or, as with adults, it may be that physical fitness and physical activity need to be considered separate risk factors in youth. This means that both physical fitness and physical activity need to be measured, evaluated, and acted on when an individual's results warrant improvement.

Data regarding physical inactivity among children and adolescents are alarming. As seen in **Figure 15.17**, only slightly more than 50% of male students in grade 9–12 met physical activity guidelines, and only approximately 30–40% of females did so (Virani et al., 2021). Even more disconcerting, there is a marked discrepancy between those who self-reported being active

(as shown in the figure) and those who actually were that active when measured objectively with portable motion sensors. Part of the problem is that only 33.3% of students in K-12 attend physical education classes daily (Roger et al., 2011).

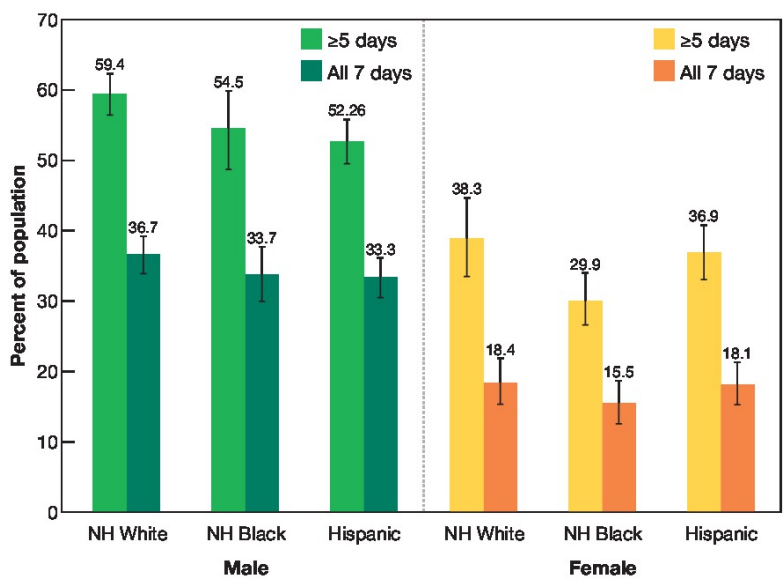


Figure 15.17 Prevalence of High School Students Who Meet Recommended Levels of Physical Activity by Sex and Race/Ethnicity. NH, non-Hispanic.

Source: Reprinted with permission from Virani, S. S., A. Alonso, H. J. Aparicio, E. J. Benjamin, M. S. Bittencourt, C. W. Callaway, A. P. Carson, A. M. Chamberlain, S. Cheng, F. N. Delling, M. S. V. Elkind, K. R. Evenson, J. F. Ferguson, D. K. Gupta, S. S. Khan, B. M. Kissela, K. L. Knutson, C. D. Lee, T. T. Lewis, ... ; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. *Circulation*. 143(8):e254–e743 (2021). Copyright © 2021 American Heart Association, Inc.

Note that the recommended amount of activity for children

and youths is 60 min·d⁻¹ (SHAPE America, 2013; U.S. Department of Health and Human Services, 2018). Research not only has reinforced the 60-minute daily minimum but has indicated that physical activity levels might need to be at least 90 min·d⁻¹ to prevent insulin resistance, which appears to be a central component in the clustering of cardiovascular risk factors (Andersen et al., 2006). This does not mean that children and adolescents should be expected to participate in 90-minute exercise sessions. It does mean that a variety of options for accumulating physical activity should be available each day, including daily school physical education classes, free play, recess, before and after school programs, intramurals, recreational and educational athletic programs, and lifestyle activities such as biking or walking for transportation (Pate et al., 2006; Weiss and Raz, 2006).

As with adults, there is now a large body of evidence showing that, independent of physical activity levels, sedentary behaviors exhibit a dose-response relationship with unfavorable health outcomes in children and adolescents. Similarly, there is evidence that decreasing any type of sedentary time (but especially limiting television time to under 2 hr·d⁻¹) is associated with lower health risks in youth aged 5–17 years. Thus, there is the need to advocate for both increases in physical activity and decreases in sedentary behavior in children and adolescents (Tremblay et al., 2011).

FOCUS ON RESEARCH | *Clinically Relevant*

The Cardiovascular Risk Associated with Acute Exercise

As has been discussed throughout this chapter, regular physical activity is widely advocated because substantial research evidence suggests that physical activity and exercise training delay the development of CVD risk factors, atherosclerosis, and CHD events. Despite all of the positive benefits, however, vigorous physical exercise can also acutely

and transiently increase the risk of heart attacks (acute myocardial infarctions) and sudden cardiac death in susceptible individuals. For example, studies have reported 1 death per 133,000 males and 1 death per 769,000 females for high school and college athletes, including all nontraumatic deaths. In older adults, 1 death per 82,000 members for a mortality rate of 1 per 2.57 million workouts over 2 years has been reported by a large commercial fitness center chain. This is based on 71 deaths in that time with a mean age of 52 years. Nearly half of these deaths occurred in members who exercised infrequently or less than once a week.

Exercise-associated acute cardiac events generally occur in individuals with known or unknown cardiac disease. In young individuals, this most commonly means hereditary or congenital cardiovascular abnormalities. In this population, the health risks of vigorous physical activity exceed the benefits, although moderate physical activity may be beneficial in terms of preventing other health risks or problems. In older individuals, coronary artery disease is the most common pathological finding. Habitual vigorous physical activity appears to reduce the risk of CHD death in patients with diagnosed disease such that the benefits of physical activity appear to outweigh the risks.

Several strategies are suggested for reducing the likelihood of an exercise-induced cardiac event:

1. Maintain physical fitness through regular physical activity because a disproportionate number of events occur in the least physically active subjects performing unaccustomed vigorous physical activity.
2. Both young athletes and adults should undergo preparticipation screening before engaging in vigorous physical activity.
3. High-risk individuals may need to be excluded from or restricted in vigorous activity.
4. Physically inactive individuals and patients with known cardiovascular disease should avoid strenuous, unaccustomed exercise in both excessively cold and hot conditions and at altitude.

5. Individuals should be encouraged to monitor their bodies for early symptoms of cardiovascular events and seek medical attention. Such symptoms include but are not limited to chest pain, increasing fatigue, feelings of indigestion, and excessive breathlessness.
6. Health-fitness facilities should:
 - a. Perform pre-entry screening by appropriately trained personnel
 - b. Have written emergency policies
 - c. Regularly conduct emergency drills and cardiopulmonary resuscitation practices
 - d. Have an automatic external defibrillator available for use by trained personnel
 - e. Establish a “hotline” to summon emergency medical care



Source: ACSM and AHA: Joint Position Statement: Exercise and acute cardiovascular events: Placing the risks into perspective. *Medicine & Science in Sports & Exercise*. 39(5):886–897 (2007).

A 2009 study ([Roemmich et al., 2009](#)) was the first to study stress reactivity and exercise in children. It determined that DBP,

SBP, and HR reactivity to a speech stressor was dampened after exercise compared to TV watching in a group of 8- to 12-year-old children. Surprisingly, physical fitness was positively associated with HR reactivity. Further research is needed to fully determine the impact of physical activity and physical fitness on the stress responses of children and adolescents.

Summary

1. Cardiovascular disease is a major cause of death in the United States, accounting for over 30% of all deaths. Coronary heart disease is the most prevalent type of CVD. Coronary heart disease is characterized by the formation of atherosclerotic plaque in the coronary arteries.
2. Cardiovascular risk factors are classified as nonmodifiable (age, heredity, race, and sex), major modifiable (cholesterol-lipid fractions, cigarette smoking, diabetes mellitus, hypertension, obesity, physical inactivity), and contributing and nontraditional (apolipoproteins, CRP, fibrinogen, fibrinolytic activity, and stress).
3. Triglycerides and a small amount of cholesterol are transported from the small intestines or liver to adipose tissue or muscles by chylomicrons and very-low-density lipoproteins (VLDL).
4. VLDLs are degraded into intermediate-density lipoproteins (IDL), which, in turn, are converted to low-density lipoproteins (LDL) in the liver. LDL transports 60–70% of the total cholesterol (TC) in the body to all cells except liver cells.
5. High-density lipoproteins (HDL) may block cholesterol uptake at the cellular or tissue level. HDL definitely carries cholesterol away from the sites of deposit to the liver, where the cholesterol can be broken down and eliminated in the bile.
6. Arteriosclerosis is characterized by a thickening of the arterial wall, inflammation, loss of elastic connective tissue, and hardening of the vessel wall.

7. Atherosclerosis is a pathological process that results in the buildup of plaque (composed of connective tissue, smooth muscle cells, cellular debris, and cholesterol) inside the vessel. The buildup of plaque obstructs blood flow and increases the risk of thrombosis. Depending on the amount of obstruction, the result can be pain, a heart attack, or a stroke.
8. Active individuals generally have lipid profiles that indicate a reduced risk for coronary heart disease (CHD).
9. The atherosclerotic process is accelerated in individuals who smoke cigarettes. Smoking injures the arterial wall lining, increases the levels of circulating TC, and decreases the amount of HDL-C. Smoking also causes blood platelets to adhere to each other, speeds up the rate of internal blood clotting, and makes the clots tougher to dissolve.
10. Individuals who are at high risk for developing hypertension can reduce the risk by participating in an endurance training program. Most hypertensive individuals experience decreased blood pressure as a result of a consistent aerobic endurance exercise program.
11. Only about 30% of Americans report regular leisure-time activity. Therefore, potentially more benefit overall could be achieved by increasing the activity level of U.S. citizens than by changing any other single CHD risk factor.
12. Metabolic syndrome is a progressive disease process in which high visceral abdominal obesity is directly related to dyslipidemia, inflammation, impaired glucose tolerance, insulin resistance, and hypertension, which together form a cluster of risk factors for CVD. Regular exercise results in favorable changes in each of these risk factors.
13. The beginnings of metabolic syndrome and CVD occur during childhood and adolescence. In general, risk factors track into adulthood and can predict future CVD. Physical activity and physical fitness are important moderators of risk factor development and progression in childhood and adolescence.

Review Questions

1. Describe the progression of atherosclerosis, indicating the role of lipids (specifically LDL-C) and inflammation.
2. Identify risk factors that cannot be changed, and discuss their relationship with CVD.
3. Identify the major modifiable risk factors, and discuss their relationship with CVD.
4. Discuss metabolic syndrome.
5. What is the impact of exercise training on each CHD risk factor?
6. What is the importance of identifying CVD risk factors in children?

Literature Search

Physical activity and physical fitness are important factors that influence the risk of cardiovascular disease. To better understand the research that has been done on this important issue, do a literature search using a search engine such as PubMed, Google scholar, or Web of Science.

- a. Search Exercise and Cardiovascular Disease. This search will yield many articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Walking programs and cardiovascular disease risk
 - ii. Resistance training and cardiovascular disease risk
 - iii. Exercise training recommendations following cardiac surgery
 - iv. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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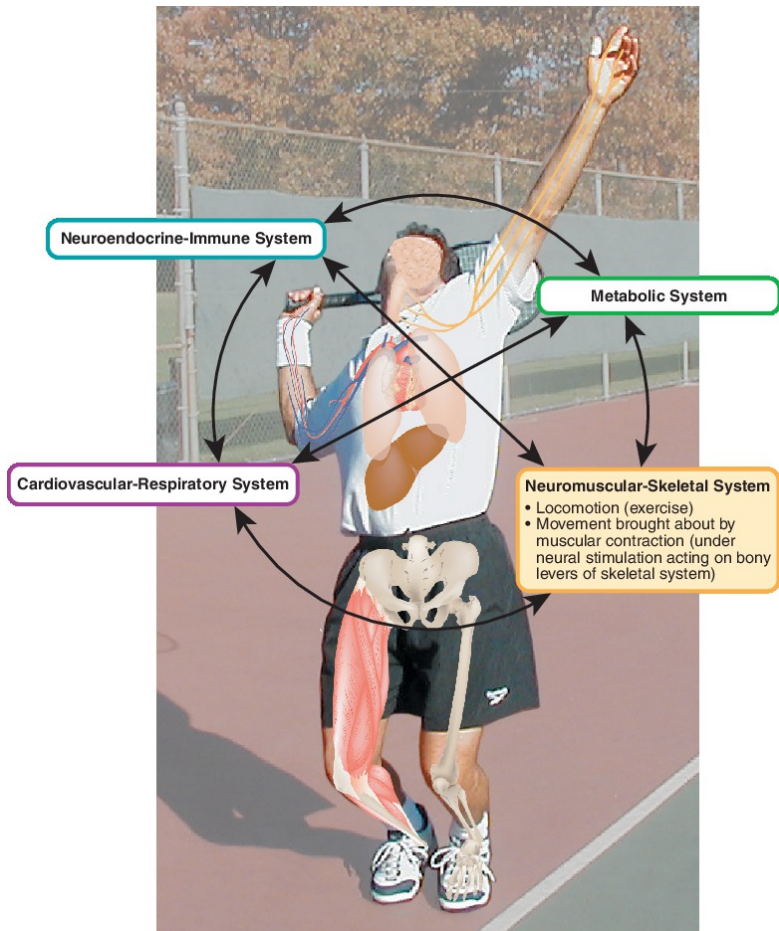
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Neuromuscular- Skeletal System Unit



The contraction of skeletal muscles, pulling on the bony levers of the body through connective tissue, causes movement. The nervous system provides the electrical signal that causes the contraction of skeletal muscle. Hence, exercise is, by definition, the direct result of neuromuscular activity. This unit will address how the skeletal, muscular, and neural systems function together to bring about movement of any type, including exercise. These systems are not the only body systems that play a role in exercise and other movement, however. In order for muscles to contract, they must continually produce energy (ATP), which means that the metabolic system is also responsible for muscle contraction and, therefore, exercise. Furthermore, the cardiorespiratory

system is responsible for supplying the muscle cells with oxygen, which supports the production of ATP necessary for continued contractions.

16 Skeletal System



CHAPTER OUTLINE

Introduction

Skeletal Tissue

- Functions

- Regulation of Blood Calcium

- Levels of Organization

- Bone Development

Measurement of Bone Health

- Dual-Energy X-Ray Absorptiometry

- Quantitative Computed Tomography

- Field Tests

- Bone Loading Questionnaire to Assess Bone Health

Factors Influencing Bone Health

Age-Related Changes in Bone
Male-Female Differences in Bone Mineral Density
Development of Peak Bone Mass

Exercise Response

Application of the Training Principles

Specificity
Overload
Rest/Recovery/Adaptation
Individualization
Retrogression/Plateau/Reversibility
Maintenance
Warm-Up and Cooldown

Skeletal Adaptations to Exercise Training

Special Applications to Health and Fitness

Osteoporosis
The Female Athlete Triad: A Portion of the Relative Energy
Deficiency in Sport Syndrome
Skeletal Injuries

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Identify the functions of the skeletal system.
- Differentiate between cortical and trabecular bone.
- Define bone remodeling, and explain how bone mineral density is affected by the balance of bone resorption and deposition.
- Describe the hormonal control of bone remodeling and growth.
- Explain the criterion method of measuring bone mineral density.
- Identify age-related changes in bone mineral density.
- Identify male-female differences in bone mineral density.

- Discuss the factors involved in attaining peak bone mineral density.
- Discuss the factors involved in the rate of bone loss in adults.
- Describe the exercise response in skeletal tissue.
- Apply the training principles to the development of an exercise program to enhance bone health.
- Describe the skeletal adaptations that occur as a result of an exercise training program.
- Describe that portion of the relative energy deficiency in sport syndrome known as the female athlete triad in relation to bone health.
- Describe micro- and macrotrauma skeletal injuries, and suggest ways of preventing them.

Introduction

The skeletal system includes the bones and connective tissue (cartilage, tendons, and ligaments) that provide the framework for the muscles and organs of the body. Like other systems of the body, the skeletal system adapts to exercise training. A healthy skeletal system is important for preventing sports-related injuries and major health problems, including osteoporosis.

Connective tissue plays a vital role in holding the skeleton together, creating articular surfaces, connecting muscle and bone to permit movement, and ensheathing organs. Like all systems of the body, connective tissues respond to stresses placed upon them. However, relatively little research exists to systematically describe the effects of exercise on connective tissues despite the fact that it provides an important component of the structure of the body, is essential to health and performance, and is often injured during fitness activities and athletic endeavors (e.g., torn cartilage or sprains that pull tendons). In fact, it is relatively recently that research has focused on how skeletal tissue, itself a type of connective tissue, responds to exercise. Exercise physiologists have focused on issues such as the maximization of peak bone mass, the prevention and treatment of osteoporosis, and the impact of heavy exercise training on the skeleton of growing prepubescent athletes. This chapter addresses these

important issues after reviewing basic concepts of skeletal physiology and the influence of physical activity on bone tissue.

Skeletal Tissue

Bone tissue, also called osseous tissue, is a dynamic, living tissue that is constantly undergoing change. In fact, adults recycle 5–7% of their bone mass every week (Marieb and Hoehn, 2019). **Bone remodeling** (bone turnover) refers to the continual process of bone breakdown (resorption) and formation (deposition of new bone). Bone remodeling has important roles in regulating blood calcium levels and replacing old bone with new bone to ensure the integrity of the skeletal system. The mass and shape of bones depend largely on the stress placed on them. The more bones are stressed (by mechanical loading during physical activity), the more they increase in volume and mass, specifically at the site of mechanical loading. The concept that bone adapts to changes in mechanical loading is described by **Wolff's law** (Beck and Marcus, 1999); that is, bone forms in areas of stress and is resorbed at areas of nonstress.

Bone Remodeling The continual process of bone breakdown (resorption) and formation (deposition of new bone).

Wolff's Law Bone forms in areas of stress and is resorbed in areas of nonstress.

Bones have a variety of shapes, sizes, and chemical makeup. This diversity of form allows bone to perform many varied functions (Hart et al., 2017). There are bones that conduct sound waves (in the ear), bone that provide structure (height), and bones that facilitate movement (spine and long bones).

Functions

The skeletal system provides a number of important structural

and physiological functions. Structurally, the skeletal system provides rigid support and protection for vital organs and allows for locomotion. Physiologically, skeletal tissue provides a site for blood cell formation (hematopoiesis in bone marrow), plays a role in the immune function (providing the site for white blood cell formation), and serves as a dynamic storehouse for calcium and phosphate, which are essential for nerve conduction, heart and muscle contraction, blood clotting, and energy formation (Bailey and McColloch, 1990; Marieb and Hoehn, 2019).

The ability of bone to perform its structural functions relates directly to its role in storing calcium. Because calcium is essential for many processes in the body, bone is broken down (resorbed) as needed to maintain blood calcium levels. The body sacrifices bone mineral (calcium) when it is needed to maintain blood calcium levels.

Regulation of Blood Calcium

As shown in **Figure 16.1**, the skeletal system (bone), the digestive system (stomach and intestines), and the urinary system (kidneys) operate together to regulate and maintain blood calcium levels. Adequate ingestion and absorption of calcium are required through the digestive system to provide the necessary calcium to be deposited in bone. In turn, because of the importance of calcium in so many vital processes of the body, bone mass is broken down to maintain blood calcium within normal limits (9–11 mg·dL⁻¹). The kidneys regulate blood calcium by filtering and reabsorbing it. The primary hormones involved in regulating blood calcium levels and bone remodeling are parathyroid hormone (PTH), calcitonin, and vitamin D (calcitriol).

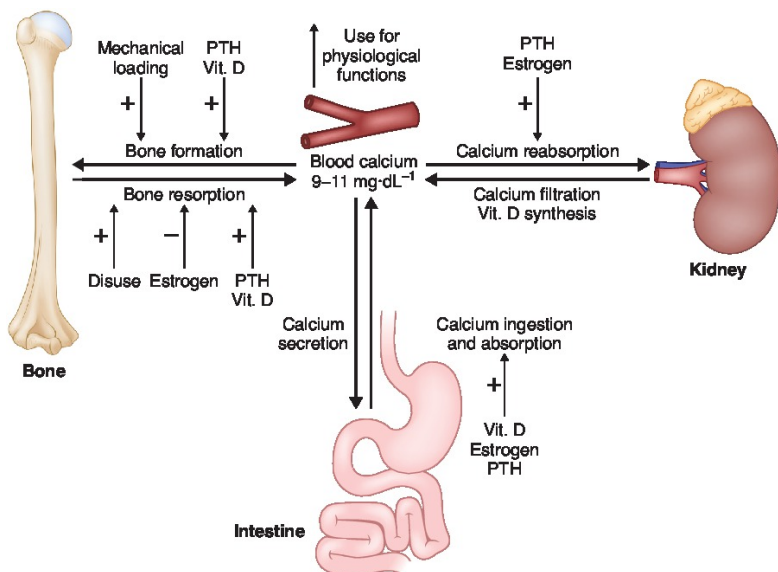


Figure 16.1 Regulation of Blood Calcium.

Blood calcium is a carefully controlled variable. Calcium is essential for the structure of bone (and teeth) and for numerous physiological functions (muscle contraction, nerve conduction, blood clotting). Blood calcium levels are maintained by the coordination of several systems, notably the digestive, urinary, skeletal, and hormonal systems. Bone loading affects bone formation and resorption. PTH, parathyroid hormone. **Source:** Adapted by permission from Springer: Borer, K. T.: Physical activity in the prevention and amelioration of osteoporosis in women: Interaction of mechanical, hormonal, and dietary factors. *Sports Medicine*. 35(9):779–830 (2005). Copyright © 2012 Springer Nature.

Levels of Organization

Understanding the structure and physiology of the skeletal system helps one understand how the skeletal system responds to exercise and training.

Bones as Organs

The human body contains over 200 different bones joined together at articulations known as joints. Joints enable movement when muscles exert force on the bones. The skeleton is typically divided into two categories: the axial (or central) skeleton that includes the bones of the skull, vertebral column, and rib cage, and the appendicular (or peripheral) skeleton that includes the bones of the hips, shoulders, and extremities. Bones have several different shapes—long, short, flat, and irregular—and each shape is specific to its function. Furthermore, according to Wolff’s law, a bone’s shape reflects its response to the stress placed on it.

Bone Tissue

The two types of bone tissue are cortical and trabecular bone. Cortical bone, also called compact, dense, or lamellar bone, is densely packed and makes up around 80% of the skeleton. Trabecular bone, also called spongy or cancellous bone, is more porous and is surrounded by cortical bone. Individual bones are composed of both types of bone tissue in varying relative proportions. **Table 16.1** presents relative percentages of trabecular and cortical bone tissue in various bones of the body. In general, bones of the axial skeleton have a much greater percentage of trabecular bone, whereas bones of the appendicular skeleton have a greater percentage of cortical bone. **Figure 16.2** shows the humerus, a typical long bone. The shaft is composed primarily of cortical bone, and the epiphyses have a greater percentage of trabecular bone.

TABLE 16.1 Composition of Various Bones

| Measurement Site | Cortical Percentage | Trabecular Percentage |
|-------------------------|---------------------|-----------------------|
| Calcaneus | 5 | 95 |
| Lumbar spine | | |
| Anterior-posterior view | 50 | 50 |
| Lateral view | 10 | 90 |
| Proximal femur | 60 | 40 |
| Total body | 80 | 20 |

Source: Highet (1989).

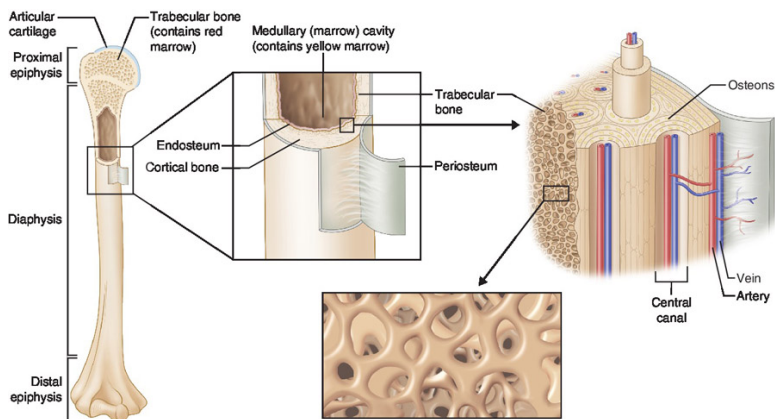


Figure 16.2 Typical Long Bone.

Structure and cross-sectional view of bone.

Cortical bone is composed of osteons, which are the functional units of bone (haversian system). Osteons are organized into concentric layers of matrix called lamellae, which are surrounded by widely dispersed cells. The matrix is made up of organic and inorganic substances.

Trabecular bone is composed of branching projections or struts, called trabeculae, which form a lattice-like network of interconnecting struts. Its appearance gives rise to another of its

names, spongy bone. Trabecular bone has the same cells and matrix elements as does cortical bone but with more porosity. About 80–90% of the volume of cortical bone is calcified, while only 15–25% of trabecular bone is calcified ([Baron, 1993](#)). The remaining volume is occupied by bone marrow, blood vessels, and connective tissue. Cortical bone is best suited for structural support and protection, and trabecular bone is best suited for shock absorption and physiological functions.

Because of its large surface area, trabecular bone can remodel more rapidly than can cortical bone. The greatest age-related loss of bone mineral density (BMD) also occurs in trabecular bone. Therefore, most osteoporotic fractures occur in areas composed predominantly of trabecular bone (wrist, hip, and spine).

Bone Development

Bone development involves three processes: bone growth, bone modeling, and bone remodeling. Each process predominates at different times throughout an individual's life.

Growth

Bone growth refers to size increase caused by an increasing number of bone cells ([Frost, 1991a](#)). There are two types of bone growth. Appositional growth is an increase in thickness or mass. Longitudinal growth occurs at the epiphyseal plate until a person reaches adult stature.

The longitudinal growth of bones results from the growth of cartilage, which is later replaced by bone. While growing, bone is also remodeling itself—that is, changing its shape and thickness. Bone growth and remodeling are distinct but closely related processes. In general, growth refers to the longitudinal growth of bone, and remodeling involves the balance between bone resorption and bone formation that affects shape of bone. If bone formation exceeds resorption, this process would also represent growth.

Modeling

Bone modeling is the process of altering the shape of bone and

adjusting bone strength through bone resorption and bone formation (Frost, 1991a; Khan et al., 2001c). Micromodeling involves the microscopic level of cell organization that occurs during formation; it determines what kind of tissue will be formed (Frost, 1991a). Macromodeling controls if, when, and where new tissue will form or old tissue will be removed. This process ensures that the bone's shape matches its role (Frost, 1988, 1991a,b; Khan et al., 2001c; Lanyon, 1989; Marcus, 1987). Modeling is largely responsible for bone growth during the years in which the skeleton is growing.

Bone Modeling The process of altering the shape of bone and adjusting bone strength through bone resorption and bone formation.

Remodeling

Bone remodeling involves a continual process of bone turnover, maintenance, replacement, and repair (Frost, 1991a). It reflects the balance between the coupled processes of bone resorption and bone formation. This ongoing process occurs because of the coupled actions of bone cells, with osteoclasts responsible for bone resorption and osteoblasts responsible for bone formation. Remodeling occurs in response to stress on the skeleton throughout the adult years. Physical activity influences bone strength and mass through remodeling, which is accomplished largely because of the activity of bone cells.

BONE CELLS The three types of bone cells are osteoclasts, osteoblasts, and osteocytes. These cells are the living part of bone. Although the cells represent a small fraction—less than 2% (Teitelbaum, 1993)—of the total composition of bone, they are responsible for its remodeling.

Osteoclasts are large, multinucleated bone cells that cause the resorption of bone tissue. Osteoclasts secrete enzymes that disintegrate bone matrix. As the bone is degraded, the mineral salts (primarily calcium and phosphate) dissolve and move into the bloodstream. **Osteoblasts** are bone cells that cause the deposition of bone tissue. Also called bone-forming cells,

osteoblasts produce an organic bone matrix that becomes calcified and hardens as minerals are deposited in it. Hardening of the bone matrix is known as ossification. **Osteocytes**, which are mature osteoblasts surrounded by calcified bone, help regulate the process of bone remodeling. Osteocytes appear to initiate the process of calcification.

Osteoclasts Bone cells that cause the resorption of bone tissue (bone-destroying cells).

Osteoblasts Bone cells that cause the deposition of bone tissue (bone-forming cells).

Osteocytes Mature osteoblasts surrounded by calcified bone that help regulate the process of bone remodeling.

The actions of osteoclasts and osteoblasts are coupled; they function as a coordinated bone multicellular unit (BMU) to remodel bone (Goldring, 2015). Osteoclasts must first cause bone resorption before the osteoblasts can form new bone. **Figure 16.3** outlines the major events of a bone-remodeling cycle (Marcus, 1987; Parfitt, 1987; Teitelbaum, 1993). From the resting phase (**Figure 16.3A**), the osteoclasts are stimulated and cause the resorption of bone, forming a cavity (**Figure 16.3B and C**). Osteoblasts then appear and deposit bone matrix where the cavity exists (**Figure 16.3D**). The matrix is called osteoid until it is calcified (**Figure 16.3E**). Calcification of the new bone occurs as calcium and phosphate minerals are deposited in the osteoid (**Figure 16.3F**). The bone then returns to the resting or quiescent phase.

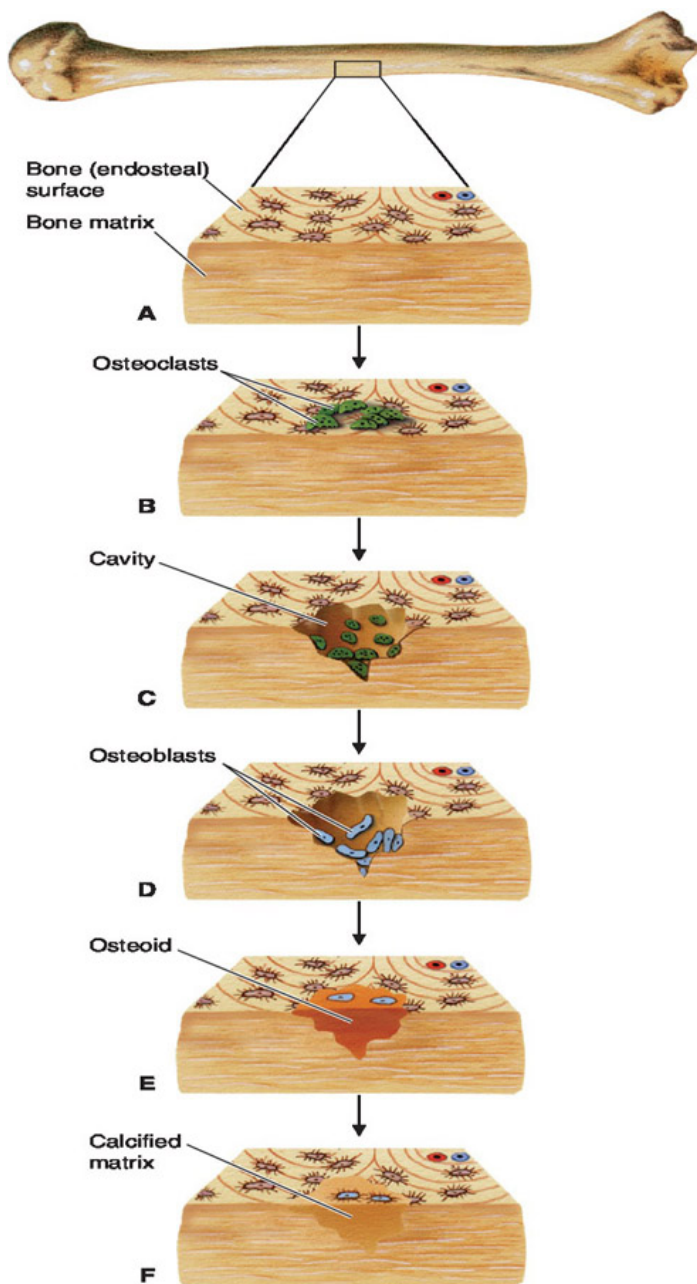


Figure 16.3 Stages of Bone Remodeling.

A. Resting cell surface. **B.** Osteoclasts (multinucleated) are activated and begin dissolving bone (minerals released to

the blood). **C.** Osteoclasts produce a cavity in the blood matrix. **D.** Osteoblasts appear. **E.** Osteoblasts secrete the osteoid (uncalcified matrix). **F.** The matrix is calcified.

Bone remodeling may result in greater bone mass, the same bone mass, or a reduction in bone mass. Through young adulthood, typically, more bone is formed than is resorbed, increasing bone mass. This increased mass strengthens the bone and accounts for the increase in bone mineral density (BMD) that commonly occurs during this period of life. When bone remodeling is in equilibrium, the amount of bone resorbed equals the amount of bone formed; thus, bone mineral density remains relatively constant. In older adults and those with certain diseases, the amount of bone resorbed is greater than the amount of bone formed, decreasing bone mineral density.

The remodeling of bone provides for skeletal growth and involves a constant turnover of bone throughout life. Bone remodeling is a complex process regulated by hormonal and local factors (Canalis, 1990).

HORMONAL CONTROL Bone remodeling reflects the interrelationship between the structural and physiological functions of bone. Calcium is not only necessary to provide structural integrity of bone but also essential for the proper functioning of the heart, skeletal muscles, and nervous tissue. Only about 1 g of calcium is present in the extracellular fluid of the body, compared to approximately 1,150 g of calcium present in bone tissue (Bailey and McColloch, 1990; Khan et al., 2001c).

Excess calcium in the blood leads to the release of calcitonin (from the thyroid gland), which causes deposition of calcium in the bone. This deposition decreases the blood calcium level and increases bone mineral density. Conversely, when the blood calcium level drops below normal, parathyroid hormone stimulates osteoclast activity, causing calcium to be released from its storage site, bone. This release of calcium from the bone causes the blood calcium level to increase and bone mineral density to decrease. **Table 16.2** summarizes the effect of calcitonin and PTH on bone and blood calcium levels. Vitamin D (calcitriol) is important for the absorption of calcium from the

intestines. Thus, it leads to an increased blood level of calcium.

TABLE 16.2 Effect of Calcitonin and PTH on Bone and Blood Calcium Levels

| Hormone | Stimulus for Release | Effect on Bone | Effect on Blood Calcium Levels |
|---------------------------|--------------------------------|-------------------------------------|--------------------------------|
| Calcitonin | Increased blood calcium levels | Bone deposition (increased calcium) | Decreased blood calcium levels |
| Parathyroid hormone (PTH) | Decreased blood calcium levels | Bone resorption (decreased calcium) | Increased blood calcium levels |

Other hormones that play an important role in skeletal health are the sex steroids (estrogen and testosterone) and growth hormone. These hormones stimulate the protein formation necessary for bone growth and are responsible for the eventual closure of the epiphyseal plate, which determines bone length and thus a person’s height (Bailey and McColloch, 1990). Estrogen promotes calcium retention and acts as an inhibiting agent of parathyroid hormone. The loss of the protective role of estrogen on the skeletal system after menopause or during secondary amenorrhea has important consequences for females. Decreased estrogen causes increased bone resorption. Growth hormone and insulin-like growth factor (IGF-1) also play an important role in bone formation and remodeling in children. Hormones are themselves stimulated by other factors, including physical activity and nutritional status.

Measurement of Bone Health

Bone strength is determined by bone mass, external geometry, and internal microstructure (Beck and Marcus, 1999; Frost, 1997; Hart et al., 2017; Heaney et al., 2000). Because it is difficult to quantify external geometry and microstructure, measures of bone

mass and bone mineral density are most often used to describe bone strength. Bone strength refers to bone's ability to withstand forces that may cause fracture. Bone strength is largely influenced by bone mass. A less dense bone will break with less force (Heaney et al., 2000). *Bone mineral content (BMC)* refers to the absolute amount of calcium and phosphate salts and is measured in grams. The calcium and phosphate salts are responsible for the hardness of the bone matrix. *Bone mineral density (BMD)* is defined as the relative value of bone mineral per measured bone area, expressed as grams per centimeter squared ($\text{g}\cdot\text{cm}^{-2}$) or milligrams per centimeter cubed ($\text{mg}\cdot\text{cm}^{-3}$), depending on the technology used to measure area.

Accurate measures of bone mineral content and bone mineral density can be obtained only in laboratory or clinical settings, primarily because of the cost of equipment and safety considerations. Nonetheless, these measurements have dramatically increased the information available to researchers, clinicians, and those in fitness professions.

Dual-Energy X-Ray Absorptiometry

Dual-energy x-ray absorptiometry (DXA) is the standard method of measuring BMD for research and clinical purposes (American College of Sports Medicine [ACSM], 2004). DXA uses an x-ray beam to measure regional and whole-body mineral content (Figure 16.4) and provides an areal bone mineral density (aBMD) value. Areal BMD provides a two-dimensional measure of density. Figure 16.5 shows a computer-generated printout of whole-body BMD and various regions of the body for a 37-year-old active female. Figure 16.5A represents regions of the body individually analyzed for BMD. The total body BMD is compared with standard references in Figure 16.5B. The blue area represents an average range across the age span of 20–100 years. Notice that the individual in the example is above average for total body BMD as represented by the asterisk (located at the intersection of age 37 years and BMD of $1.197 \text{ g}\cdot\text{cm}^{-2}$).

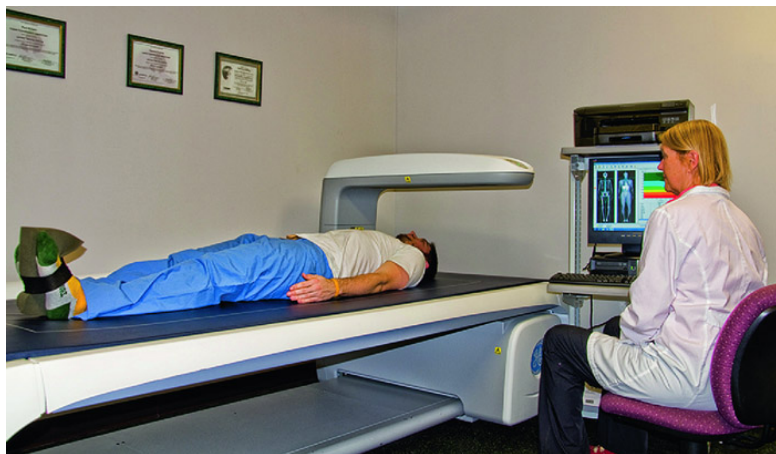
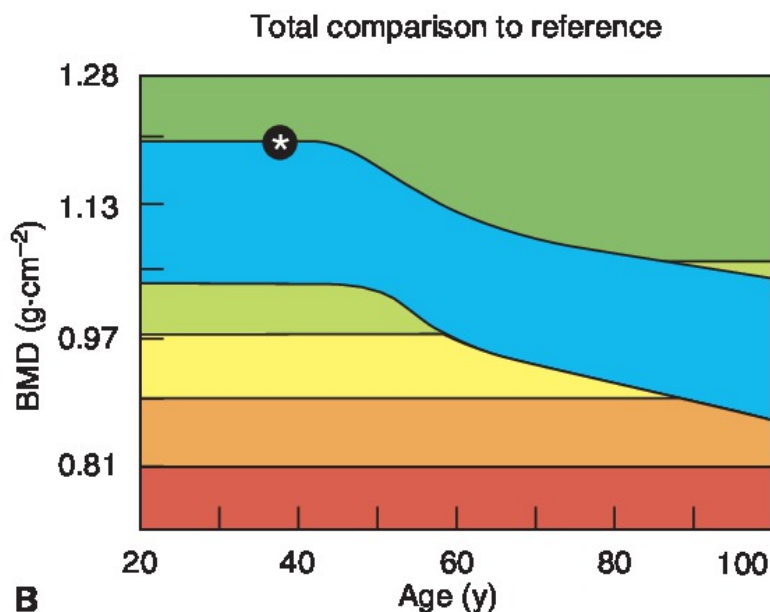


Figure 16.4 Dual-Energy X-Ray Absorptiometer.
Subject is positioned for a total body scan.



A



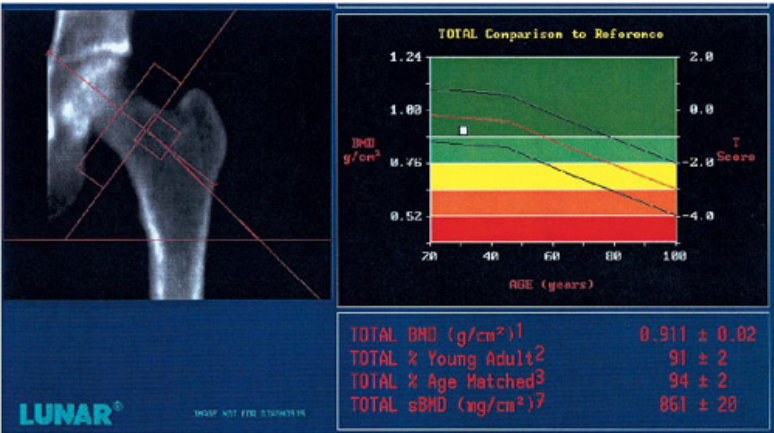
| Bone results | | | |
|--------------------------------------|---------------------------|------------------|------------------|
| Standard analysis – DEXA calibration | | | |
| Region | BMD g·cm ⁻² | % young adult | % age matched |
| Head | 2.323 | — | — |
| Arms | 0.977 | 116 | 117 |
| Legs | 1.229 | 106 | 108 |
| Trunk | 0.971 | 106 | 108 |
| Ribs | 0.711 | — | — |
| Pelvis | 1.217 | 110 | 112 |
| Spine | 1.289 | 113 | 116 |
| Thoracic | 1.185 | — | — |
| Lumbar | 1.507 | — | — |
| Total | 1.197 | 106 | 108 |

C

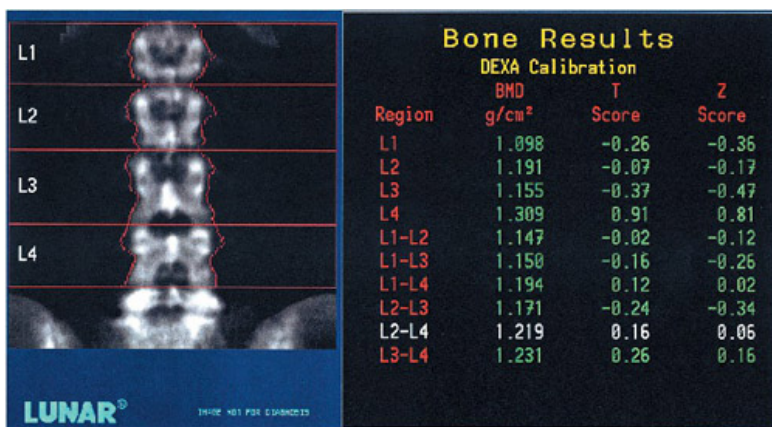
Figure 16.5 Computer-Generated Printout of Whole-Body BMD for Healthy Young Female.

A. Whole-body BMD scan. **B.** Graphical presentation of total body BMD compared to reference values and age. **C.** Printout of regional BMD values and compared to reference values. **Source:** Data from the University of Connecticut Health Center, Osteoporosis Research Center.

Regional BMDs are shown in **Figure 16.5C** along with comparisons with young adult normative data. Notice that each region of the body has a unique BMD value because of the varying composition of bones. For example, the legs have a BMD of $1.229 \text{ g}\cdot\text{cm}^{-2}$, whereas the pelvis has a BMD of $1.217 \text{ g}\cdot\text{cm}^{-2}$. These values correspond to 106% and 110% of young adult values, respectively. In addition to these total body scans, clinicians and researchers often measure BMD at specific, clinically relevant sites, such as the hip or spine, where osteoporotic fractures are more likely. When scanning just a small area (e.g., a hip or spine), a better-quality scan results. **Figure 16.6A and B** presents a hip and spine scan of an active, older woman. These site-specific scans enable researchers to investigate differences in bone mineral density at various sites, among various individuals, and as a result of adaptation to long-term exercise training.



A



B

Figure 16.6 DXA Scan.

A. Hip (head of femur). B. Spine.

Bone mineral density derived from DXA is often used to define osteopenia and osteoporosis. BMD is normally distributed and is often expressed in standard deviation (SD) units related to its T or Z distribution. The T distribution has a mean score of zero (0). **Osteopenia** is a condition of decreased bone mineral density defined as a T-score of -1 to -2.5 . This means a BMD value greater than 1 standard deviation below (but not more than 2.5 SD below) values for young normal adults (25–35 years, sex-matched). **Osteoporosis** is a condition of porosity and decreased BMD defined as a T-score greater than -2.5 , indicating a BMD more than 2.5 SD below values for young normal adults (WHO, 1994). *Established osteoporosis* is the term for the condition of osteoporosis, as defined above, plus one or more fractures (Kanis et al., 1993). **Figure 16.7** indicates the relative risk of fracture at the spine, hip, and forearm based on T-scores derived from DXA scans.

Osteopenia A condition of decreased bone mineral density (BMD) defined as a T-score of -1 to -2.5 , which means a BMD value greater than 1 standard deviation (SD) below (but not more than 2.5 SD below) values for normal young adults.

Osteoporosis A condition of porosity and decreased bone mineral density defined as a T-score below -2.5 , which indicates a BMD greater than 2.5 SD below values for young normal adults.

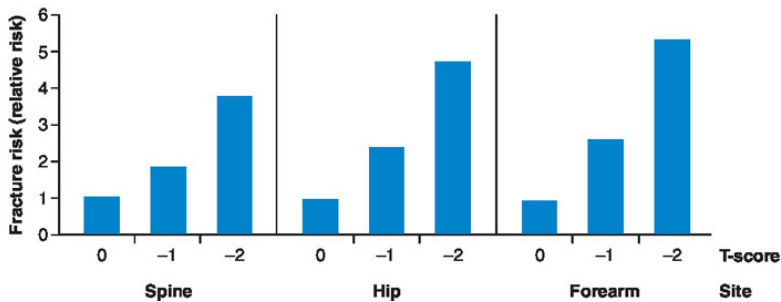


Figure 16.7 Relative Risk of Fracture at Various Sites Based on T-score.

Source: Data from [Khan et al. \(2001b\)](#).

The National Bone Alliance Network is a public-private collaboration that seeks to reduce the burden of osteoporosis. They have proposed expanding the definition for osteoporosis to include individuals with osteopenia (based on DXA measures) who have also had a low-trauma fracture and who have an elevated risk of fracture based on a Fracture Risk Algorithm (FRAX) (ACSM, 2022; [Lee et al., 2013](#)).

While the DXA scan is the most common technology used to quantify BMD and make predictions about relative fracture risk, there are limitations in the information that the DXA can supply since it is a two-dimensional measure of a three-dimensional bone. Research and development are continuing on several promising DXA-derived measures (using finite element analysis) of bone strength, including bone strain index (BSI) ([Ulivieri et al., 2020](#); [Ulivieri and Rinaudo, 2021](#)). This technique uses information from the DXA scan to derive indices of bone microarchitecture within the spine and hip. This is a promising area that will allow greater information to detect the ability of bones to resist fractures during physical activity.

Quantitative Computed Tomography

Quantitative computed tomography (QCT) is a newer technique that provides researchers with measures of bone health in addition to BMD (**Figure 16.8**). QCT can determine the volumetric bone mineral density (vBMD) of trabecular and cortical bone. Volumetric BMD is a measure of three-dimensional volume. Peripheral QCT (pQCT) also provides a measure of bone strength during compression (BSI) and of torsional strength (SSI_p). Since QCT appears more sensitive to bone changes than DXA, QCT measures will likely be used increasingly to assess bone adaptations to exercise ([Khan et al., 2001c](#); [Polidoulis et al., 2012](#)). High-resolution peripheral quantitative tomography (HR-pQCT) is a newer technique that provides measures of the volume of BMD (a three-dimensional measurement) and the microarchitecture of trabecular and cortical bone, thereby, allowing direct measures of bone strength and potentially providing a useful tool to predict fracture risk ([Mikolajewicz et al., 2019](#)).



Figure 16.8 A Peripheral QCT (pQCT) Is Used to Determine the Volumetric Bone Mineral Density (vBMD) of Trabecular and Cortical Bone.

Field Tests

The 2012 report “Fitness Measures and Health Outcomes in Youth” ([Institute of Medicine, 2012](#)) reviewed the literature relative to musculoskeletal fitness and bone health and concluded that there is a positive association between measures of muscle strength and power and bone health in adults but that there was a dearth of high quality of studies establishing this link in youth. Nonetheless, they recommended the inclusion of a muscle power test, the standing long jump, for health-related physical fitness testing of youth. Since that time, a growing body of research evidence has pointed to a jump field test as a valid predictor of bone health in children and adolescents. [Janz et al. \(2015\)](#) provided direct evidence that muscle power predicts bone strength. In this study, muscle power was assessed in male and female adolescents by the vertical jump, and bone strength was assessed at two locations on the tibia by pQCT. Strong and consistent associations were determined between the muscle power and bone strength measures of the cortical area (CoA), strength during compression (BSI), and strength during torsion (SSI_p). Similarly, a study of children 8–10 year demonstrated that vertical jump power was able to identify individuals with whole-body BMD below average as measured by DXA scans adjusted for height ([Baptista et al., 2016](#)). A third study assessed the association between standing broad jump and vertical jump with bone strength of the radius and tibia using quantitative ultrasonography (QUS) in adolescent athletes and nonathletes. QUS measures the speed of sound, is highly correlated with bone characteristics assessed by DXA, and reflects bone factors such as elasticity, structure, microstructure, and cortical thickness. Both jumps were found to be good indicators of bone health independent of sex and sport status ([Henriques-Neto et al., 2020](#)). Other studies have found similar results for college-aged athletes and nonathletes ([Yingling et al., 2020](#)) and young adults ([Higgins et al., 2018](#); [King et al., 2017](#); [Yingling et al., 2019](#)). All authors recommended that muscle power should be included as a component of health-related fitness ([Chapter 1](#)) and bone-enhancing health programs and as such that the vertical jump become part of the battery of health-related physical fitness tests.

Bone Loading Questionnaire to Assess Bone Health

The Bone-Specific Physical Activity Questionnaire (BPAQ) quantifies the types of physical activity that load the skeleton in ways that are known to enhance bone health (Weeks and Beck, 2008). The BPAQ was developed with attention to the ground reaction forces that impact the osteogenic (bone producing) effect of certain types of physical activity (Weeks and Beck, 2008). In a research study, in young and middle-age women, the scores from the BPAQ were a strong predictor of bone strength (measured from pQCT) and aBMD (measured from DXA) (Kim et al., 2018). This tool shows promise in assessing some measures of bone health. However, as has been discussed, there are several factors that impact bone health, and additional research is warranted to understand its full utility.

Factors Influencing Bone Health

Bone health is related to the mass, strength, and stiffness of bone and is influenced by many factors, including nutrition, hormonal status, body composition, and physical activity (Burr, 2011; Hart et al., 2017; Weaver et al., 2016). Bone health is largely related to peak bone mass attainment and bone loss rate. Both processes are influenced by age and sex.

Age-Related Changes in Bone

Bone changes in density throughout life. **Figure 16.9** shows the characteristic pattern between bone mass and age for males and females (Ott, 1990; Weaver et al., 2016). The first 20 years of life are characterized by active growth in bone mass. About 25% of the final adult bone is accumulated from approximately 11.5–13.5 years (around the age of menarche) for girls and 13.0–15.0 (peri- to postpuberty) for boys. This approximates the amount of bone lost in females in postmenopausal years (MacKelvie et al., 2003). The skeletal consolidation phase occurs in early adulthood, and peak bone mass is generally attained by 30 years.

Shortly after attainment of peak bone mass, bone mass loss begins. After a rapid-loss phase, the rate of bone loss decreases (Teitelbaum, 1993). The theoretical curves shown in **Figure 16.9** assume the attainment of full genetic potential. If environmental factors such as exercise, nutrition, and hormonal status are inadequate, full genetic potential for bone mass may not be realized, increasing fracture risk (Heaney et al., 2000). The specific effect of aging is difficult to know precisely because bone health is affected by many factors, especially physical activity level and nutritional patterns (Tucker et al., 2002; Weaver et al., 2016; Wohl et al., 2000).

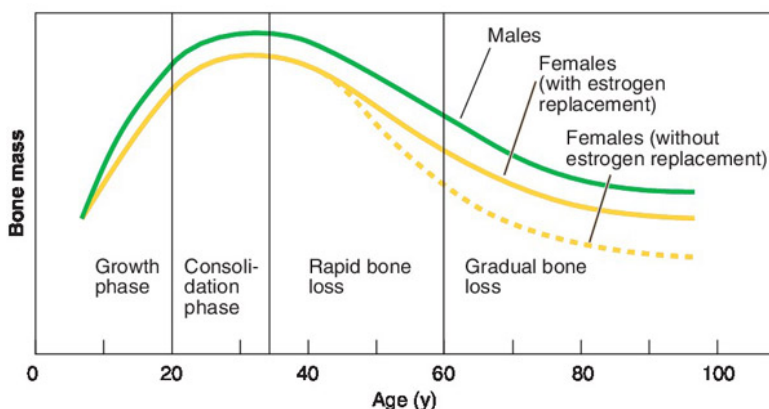


Figure 16.9 Comparison of BMD for Females and Males.

Bone mineral density values change throughout the life span. Female values are influenced by hormonal status.

Male-Female Differences in Bone Mineral Density

BMD varies between males and females. As shown in **Figure 16.9**, total BMD changes throughout the life span for both sexes. Bone mineral density increases throughout childhood and early adult life for both sexes, but the peak bone mineral density attained is less in females than in males (Rizzoli, 2014a).

In addition to differences in total BMD, BMD also varies between males and females according to measurement site. **Table**

16.3 compares adult BMD at various sites for men and women (Beck and Marcus, 1999).

TABLE 16.3 Comparison of Adult Male and Female Bone Mineral Density at Various Sites

| | Males | Females |
|-------------------------------------|-------|---------|
| Hip (g·cm ⁻²) | 1.033 | 0.942 |
| Spine (L2–L4) (g·cm ⁻²) | 1.115 | 1.079 |
| Radius (g·cm ⁻²) | 0.687 | 0.579 |

Source: Beck and Marcus (1999).

At menopause, females lose the protective influence of estrogen, and bone loss accelerates if estrogen is not pharmacologically replaced. On average, annual bone loss is 2–3% during the first few years following menopause and 0.5–1% thereafter (Rizzoli et al., 2014). The loss of the protective influence of estrogen and accompanying bone loss explains the prevalence of osteoporotic fractures in older postmenopausal women.

Development of Peak Bone Mass

Peak bone mass is attained during the mid-30s in both sexes, although 95% of peak bone mass is achieved by age 20 (Beck and Marcus, 1999). The individual’s peak bone mass developed in young adulthood is influenced by mechanical factors, nutrition, hormonal levels, and genetics (Heaney et al., 2000; Karlsson and Rosengren, 2020; Rizzoli, 2014a). Mechanical factors include physical activity and gravity. These forces are generally considered necessary stimuli for bone formation and growth (Frost, 1997). Studies done with astronauts and with individuals confined to bed rest clearly show a loss of bone mineral density when bone is not subjected to the force of gravity.

Although additional research is needed to specify exercise prescriptions for optimal skeletal development, children and

adolescents should be encouraged to engage in physical activity to promote bone health along with other positive changes and development within the body. Specifically, for bone development, children and adolescents should be encouraged to participate in high-impact activities (Greene and Naughton, 2006; Karlsson and Rosengren, 2020; Khan et al., 2001b; Macdonald et al., 2007; Tenforde et al., 2015; Vicente-Rodriguez, 2006). Puberty is a particularly important time period for the development of bone mass; bone size, mass, and BMD increase approximately 4% per year from childhood to late adolescence and early adulthood (Ackerman and Misra, 2011; Matkovic and Visy, 2015; Weaver et al., 2016). For girls, peak BMC velocity occurs on the average at about age 12.7 years (the average age of menarche); for boys, it occurs about 1.5 years later. Of course, maturational age varies widely among individuals. Specific exercises for bone development are generally important from approximately 9–16 years.

Adequate nutrition is necessary for developing a strong skeletal system. Dietary protein, calcium, and adequate vitamin D are essential for bone health but are often deficient in young athletes. **Table 16.4** shows the recommended dietary reference intake (DRI) of calcium and vitamin D. Note that these are adequate intake (AI) values, that is, recommended amounts based on observed or experimentally determined approximations from healthy individuals when a more specific recommended dietary allowance (RDA) has not been determined. Unfortunately, many individuals fall well below the recommendations. In particular, young women who are concerned about weight control often restrict dietary intake, leading to low calcium and vitamin D intake. For instance, young women may eliminate dairy products from their diet because they are high in fat. But dairy products also are an excellent source of calcium, vitamin D, and protein, all of which are essential to the development of good bone health. Thus, while trying to maintain weight, these athletes may be negatively affecting the attainment of peak bone mass. Exercise professionals must consider the need for dietary calcium, vitamin D, and protein when counseling young athletes (particularly females) about weight management (ACSM, 2022; Rizzoli, 2014a). The availability of the many low-fat dairy products makes including calcium in the diet easier. Importantly, fortified

dietary products result in more favorable changes in biochemical markers of bone metabolism than does supplementation alone, suggesting that dietary sources of these nutrients are more effective than simply taking a pill (Rizzoli, 2014b). Recommendations for play and exercise outdoors may also help increase the synthesis of vitamin D.

TABLE 16.4 Recommendations for Dietary Intake

| Age Group | Calcium (mg·day ⁻¹) | Vitamin D (IU·day ⁻¹) |
|-------------------------------------|------------------------------------|--------------------------------------|
| Infant | | |
| Birth–6 mo | 200 | 400 |
| 6 mo–1 y | 260 | 400 |
| Children | | |
| 1–3 y | 700 | 600 |
| 4–8 y | 1,000 | 600 |
| Adolescents and young adults | | |
| 9–18 y | 1,300 | 600 |
| Men | | |
| 19–70 y | 1,000 | 600 |
| >70 y | 1,200 | 800 |
| Women | | |
| 19–50 y | 1,000 | 600 |
| 51–70 y | 1,200 | 600 |
| >70 y | 1,200 | 800 |
| 14–18 y, pregnant or nursing | 1,300 | 600 |
| 19–50 y, pregnant or nursing | 1,000 | 600 |

IU, international units.

Source: Institute of Medicine: Dietary Reference Intakes for Calcium and Vitamin D. (2011). <https://>

As mentioned previously, adequate estrogen levels are also needed to attain peak bone mass. This is discussed further in relation to older adults and female athletes later in this chapter.

Finally, there are genetically determined limits to the amount of BMD that an individual can attain. The only way to achieve genetic potential, however, is to pay careful attention to modifiable factors: nutritional status, hormonal status, and activity level.

Exercise Response

Physical activity increases mechanical forces on bones. This leads to physiological changes in bone cells that allow bone to be modeled and remodeled. **Mechanotransduction** is the process by which a bone responds to a mechanical force on it. Mechanical forces are converted into biological signals (primarily by osteocytes) that signal bone remodeling by osteoblast activity and osteoclast resorption (Bonnet and Ferrari, 2010; Klein-Nulend et al., 2015). As illustrated in **Figure 16.10**, physical activity applies a mechanical force (e.g., bending or deformation) to bone. Bending causes both *compressive* and *tensile* stress that alters the hydrostatic pressure in different regions of the bone tissue, causing movement of fluid in this tissue. Fluid flows through the small canals and spaces within the bone matrix (lacunocanalicular system) and around osteocytes; this flow aids in the transport of nutrients and waste. This fluid movement also exerts a shear stress that may stimulate an osteogenic response, resulting in the formation of new bone.

Mechanotransduction The process by which a bone responds to a mechanical force on it.

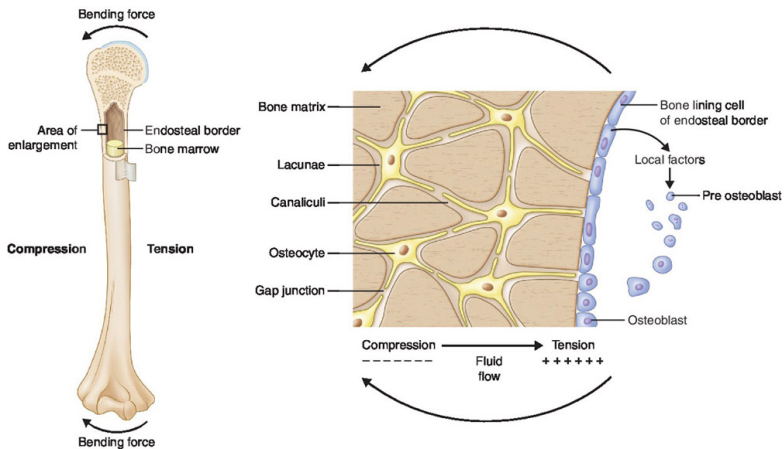


Figure 16.10 Effect of Bending Force on Bone Physiology.

Bending creates both compressive and tensile forces. Compression creates high hydrostatic pressure that facilitates movement of bone fluid to the area of tension, which has low hydrostatic pressure. The movement of fluid through the lacunocanalicular system aids in nutrient delivery and waste removal and plays an important role in the osteogenic response to loading. **Source:** Modified with permission from Zernicke, R. F., G. R. Wohl, & J. M. LaMothe: The skeletal-articular system. In Tipton, C. M., M. N. Sawka, C. A. Tate, & R. L. Terjung (eds.): *ACSM's Advanced Exercise Physiology*. Baltimore, MD: Lippincott Williams & Wilkins, 106 (2006).

Physical activity causes specific changes in bone physiology within minutes. Soon after a mechanical load is placed on bone cells, they release prostacyclin; this is followed within minutes by an increase in enzymes related to metabolism. Six to twenty-four hours after activity, RNA synthesis increases. There is evidence of increased collagen and mineral deposition on the bone surface within 3–5 days after a bout of loading (Khan et al., 2001c). The relationship between levels of blood biomarkers of bone turnover and structural and mineral adaptations of bone continues to be an area of research (Lester et al., 2009; Maïmoun and Sultan, 2011).

Biomarkers have the potential to provide insight into the dynamic changes to bone with exercise training programs; however, there are still challenges with interpreting the results of various studies based on issues with standardization (Marini et al., 2020).

Application of the Training Principles

Bone's adaptation to physical activity depends on the type of loading. In other words, the response is specific to the type of activity performed. Stress (or load) refers to the external force applied to a bone, whereas strain (deformation) refers to changes in the bone tissue. Adaptations in any given physiological system are dependent on the extent to which exercise stresses that system. For instance, adaptations in the cardiovascular system depend on the intensity, duration, and frequency of predominantly aerobic exercise training. Similarly, adaptations in skeletal muscle depend on the load, number of reps, rest period, number of sets, and frequency that load-bearing exercise is performed. The adaptation to a mechanical load (physical activity) in bone depends on the strain magnitude, strain rate, distribution of load on the bone, and number of cycles (Khan et al., 2001a). *Strain magnitude* is the amount of relative change in bone length under mechanical loading. *Strain rate* is the speed at which strain develops and releases. *Distribution of load* refers to how strain occurs across a section of bone. *Strain cycles* are the number of load repetitions.

The *mechanostat theory*, shown in **Figure 16.11**, suggests that bone adapts to set points of *minimal effective strain (MES)*. This theory suggests that a control system operates in which an MES is necessary to maintain bone and that a higher MES must be surpassed to overload bone appropriately for positive adaptations (increased bone mineral density and strength). Above the repair MES, bone enters a state of overuse. In the pathological overuse zone, bone suffers from microdamage, and woven (unorganized) bone is added as part of the repair process, leading to increased bone mass but not bone strength (Khan et al., 2001a).

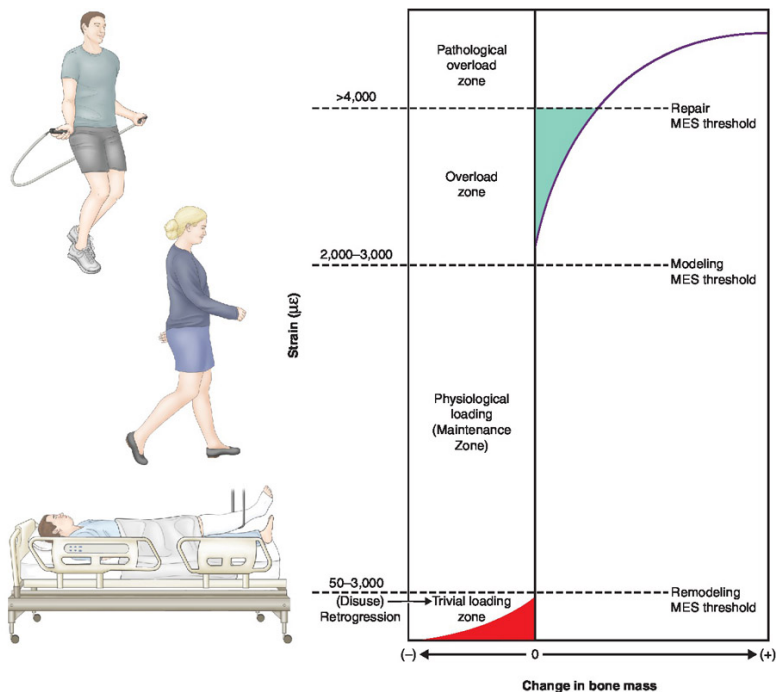


Figure 16.11 The Mechanostat Theory Relating Strain to Bone Mass.

MES, minimal effective strain. **Source:** Modified from Forwood, M. R., & C. H. Turner: Skeletal adaptations to mechanical usage: Results from tibial loading studies in rats. *Bone*. 17(4 Suppl):197S–205S (1995). Copyright © 1995 Elsevier. With permission.

The precise type and amount of activity for enhancing and maintaining bone health is not fully known at present. However, several recommendations can be made based on the mechanostat theory and research. The overall goals of physical activity relative to skeletal health are to (1) increase peak bone mass in adolescents, (2) minimize age-related bone loss, and (3) prevent falls and fractures (ACSM, 2004).

Specificity

The specificity principle applies to the particular bones being stressed, the composition of the bone being stressed (cortical vs. trabecular), and the type of activity being performed. Research data suggest that the type of exercise or activity performed greatly influences skeletal adaptations. **Weight-bearing exercise** refers to movement in which the body weight is supported by muscles and bones, thereby working against gravity. **Non-weight-bearing exercise**, in contrast, refers to movement in which the body is supported or suspended and thereby not working against the pull of gravity. Weight-bearing or impact-loading activities, such as running, gymnastics, stair climbing, volleyball, and resistance training, are more likely to stimulate increased bone mass than non-weight-bearing activities, such as swimming and cycling (ACSM, 2004; Duncan et al., 2002; Proctor et al., 2002; Tenforde et al., 2015). The best activity is chosen based on the individual's health and preference. When considering exercise for individuals with low bone mineral content, the risk of falling and causing a fracture is a major concern. Activities with a high risk of falls or collisions should not be recommended for certain populations, such as older adults. **Table 16.5** provides general guidelines for types of activities appropriate for various groups (ACSM, 2004).

Weight-Bearing Exercise A movement performed in which the body weight is supported by muscles and bones.

Non-Weight-Bearing Exercise A movement performed in which the body weight is supported or suspended and thereby not working against the pull of gravity.

TABLE 16.5 Recommended Activities for Skeletal Health

| | Children and Young Adults | Adults, Premenopausal | Adults, Below-Normal BMD | Adults, Very Low BMD |
|------------------|---|---|---|---|
| Goal | To attain peak bone mass | To slow the rate of bone loss and prevent musculoskeletal injury | To decrease risk of injury and/or slow rate of bone loss | To avoid injury |
| Type of activity | High-impact-loading movements Dynamic resistance exercise | Moderate-impact-loading movements Dynamic resistance exercise | Low- to moderate-impact-loading movements | Low-impact-loading movements |
| Examples | Sprinting, jumping, track and field, volleyball, basketball, gymnastics, soccer, weight training, rope skipping | Walking, jogging, running, hiking, stair climbing, stepping (machines), dancing, weight training, rope skipping, skiing | Stair climbing (machine), hiking, cross-country skiing, weight training | Walking, water aerobics, swimming, stationary cycling |

Because dynamic resistance training is associated with positive adaptations in skeletal tissue as well as muscular fitness, exercise physiologists generally recommend this type of exercise for maintaining both muscular and skeletal health (ACSM, 2004; Layne and Nelson, 1999). Loading seems to have a localized effect (*Wolff's law*); thus, specific sites can be isolated for impact. Conversely, a general dynamic resistance program that works all the major muscles of the body should benefit the total skeleton. Note that bone tissue does not appear to respond to static resistance exercise (Turner and Robling, 2003).

The **Check Your Comprehension box** provides you the opportunity to apply the information presented in this section.

CHECK YOUR COMPREHENSION 1

Use **Table 16.5** to answer the following questions.

1. What type of activities would you recommend for your 45-year-old aunt who has just learned that she has low BMD? How is this program consistent with the goals for this population?
2. Because of your aunt's diagnosis of low BMD, what physical activities and dietary recommendations would you give to your 15-year-old cousin (your aunt's daughter)? Why?

Check your answer in **Appendix C**.

Overload

As mentioned earlier, weight-bearing exercises result in positive skeletal adaptations. As shown in **Figure 16.11**, the threshold for a stimulus that initiates new bone formation is termed the minimal effective strain (MES) for remodeling (Frost, 1997). A load or force that exceeds this threshold and is repeated a sufficient number of times is thought to cause osteoblasts to secrete osteoid and lead to the formation of new bone. The MES for bone modeling, and thus the impact load necessary to induce positive skeletal adaptations in humans, is not precisely known, but the stimulus must include forces considerably greater than those of habitual activity. There is strong evidence that weight-bearing, impact-loading exercises can lead to an increase in bone mineral density in children and adolescents and also decrease the age-related loss of bone mineral density in adulthood (ACSM, 2022; Borer, 2005; NIH, 2001).

Impact loads, and thus the strain applied to bones, can be manipulated by increasing repetitions or by increasing the strain magnitude as measured by ground reaction force or joint force. For example, running loads bones by high repetition, whereas rope jumping overloads the bones primarily by intensity (strain magnitude). For adaptations in skeletal tissue, intensity is apparently more important than repetition (Beck and Marcus, 1999; Kato et al., 2006). Just 10 maximal vertical jumps per day, 3 days per week for 6 months has been shown to significantly increase bone mineral density in college-aged women (Kato et al., 2006).

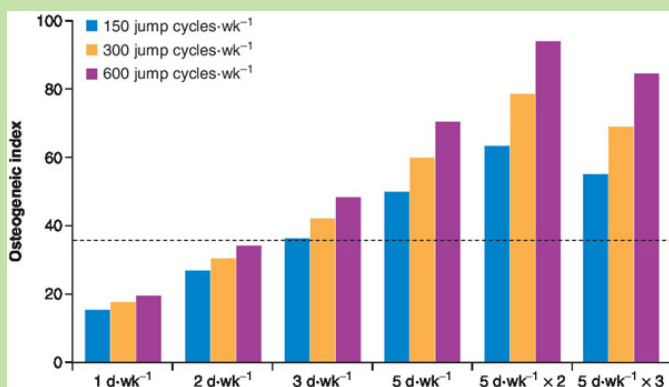
FOCUS ON APPLICATION | Clinically Relevant

Proposed “Osteogenic Index” to Measure the Effectiveness of Exercise on Bone Formation

Some researchers think that bone cells exhibit a desensitization when mechanical loading sessions are extended. Turner and Robling (2003) developed an

“osteogenic index” (OI) to determine the effectiveness of the stimulation for bone formation based on the number of repetitions of high-impact exercise (such as jumping) per session and per week. This is presented in the figure below.

In this figure, the cycles-wk⁻¹ refers to the number of jumps completed. Such mild-impact loading represents three times the body weight. The dashed horizontal line represents the OI generated by 20 minutes of walking 5 d-wk⁻¹ (800 load cycles per leg at 1.1 times body weight). According to this instrument, the OI increases threefold if the exercise is administered 5 d-wk⁻¹ as opposed to 1 d-wk⁻¹. Clearly, based on this tool, adding more sessions per week improved the OI more than did lengthening the exercise session by the number of jump cycles. Furthermore, the OI is increased as much as an additional 50% if the daily exercise is divided into two shorter sessions separated by 8 hours. However, dividing the exercises into three sessions separated by 4 hours does not achieve a better OI. If the theory proposed by these researchers proves correct and the OI is a valuable tool for measuring bone stimulation, then load-induced bone formation would be enhanced by regimens that incorporate rest periods between short vigorous bouts. For example, running short sprints should build more bone than an endurance run.



Source: Reprinted with permission from Turner, C. H., & A. G. Robling: Designing exercise regimens to increase bone

strength. *Exercise and Sport Sciences Reviews*. 31(1):45–50 (2003). Copyright ©2003 The American College of Sports Medicine..

Until the amount of exercise needed to impose an overload is known, the rate of adaptation or the ideal progression necessary to induce additional gains in bone density cannot be determined. However, any type of exercise overload (intensity, duration, or frequency) must begin at a level the individual can safely tolerate and progress gradually. Skeletal adaptations are unique in terms of the slow turnover rate of bone. Because it takes about 3–4 months for one remodeling cycle to complete the sequence of bone resorption, formation, and mineralization, a minimum of 6–8 months of exercise training are typically required to detect a measurable change in bone mass in humans, using current technology (ACSM, 2022).

Rest/Recovery/Adaptation

It has been difficult to quantify the osteogenic stimuli needed to result in positive bone adaptations in humans and to determine the optimal amount of rest and recovery for positive bone adaptations. Some researchers have used information from animal studies to develop an osteogenic index (see Focus on Application box) to guide exercise prescription for bone adaptations. This osteogenic index has proven useful in quantifying training load in intervention studies ([Kishimoto et al., 2012](#); [Lester et al., 2009](#)) and previous sporting activity in retrospective studies ([Kato et al., 2015](#)). Although research to determine the precise amount of overload required to lead to positive bone adaptation is a continuing area of research, a review of literature has shown that high-impact loading (e.g., gymnastics, hurdling, karate, volleyball, and other jumping sports) and odd-impact loading (e.g., soccer, basketball, racket games, and speed skating) are associated with higher bone mineral density and enhanced bone geometry ([Tenforde and Fredericson, 2011](#)). It is known that inadequate rest and recovery along with excessive repetitive loads can lead to stress fractures

(discussed later in this chapter).

Individualization

The individual response principle applies to the skeletal system as well as other body systems; that is, different people respond to the same exercise stress differently depending on their genetic makeup, hormonal and nutritional status, and so on. Individuals with low BMD have the greatest potential for benefit.

Additionally, exercise interventions goals vary for individuals across the life span. During childhood, the primary goal is to improve bone acquisition and attain the highest peak bone mass genetically possible. High-impact activities, such as jumping, hopping, and ball sports that incorporate high-impact, multidirectional loading, should be incorporated into activity starting in the prepubertal years (Tenforde et al., 2015). It has been shown that as little as 10 minutes of high-impact circuit training during physical education class performed 3 days a week over 2 years resulted in significantly greater improvement in bone mineral content in young girls (mean age = 9.9 years) than in age-matched controls (MacKelvie et al., 2003). The goal through early adulthood is to build bone and through middle age to maintain bone. This requires weight-bearing activity with a force of impact greater than 2.5 times body weight. For older adults, the goal is to reduce bone loss and prevent falls. This means emphasizing activities that challenge the postural system and use resistance for loading muscles and bone. Dynamic resistance programs should promote balance and upper and lower body muscle strength to reduce the risk of falling and possible resulting bone fractures. Osteoporotic individuals should not engage in jumping activities. Although walking in itself is not a strong bone stimulus (see Focus on Application box), a lifetime of walking may beneficially reduce bone loss (Beck and Snow, 2003).

Retrogression/Plateau/Reversibility

The reversibility principle suggests that if you cease exercising for a time, you lose the benefits of exercising. Studies of immobilized

patients (Donaldson et al., 1970; Vogel and Whittle, 1976) and discontinued training (Dalsky et al., 1988; Iwamoto et al., 2001; Nordström et al., 2005; Winters and Snow, 2000) indicate that this principle also applies to bone.

Maintenance

The increased BMD resulting from exercise training appears to be reduced with the cessation of training. The rate of bone loss is not known, however, nor is the level of activity needed to maintain bone mineral density or the threshold at which bone loss occurs. Intense exercise training during the pubertal years and early adulthood may lead to greater attainment of peak bone mass, which may protect against fractures later in life because more bone mass can be lost then before the bone is weakened to the point of fracture (Heaney et al., 2000; Karlsson, 2004). There is evidence that increases in BMC in the femoral neck gained during 7 months of high-impact training in prepubertal children were maintained during a 7-month detraining period (Fuchs and Snow, 2002). Clearly, activity is needed to maintain BMD, but additional research is necessary to determine the level of activity in various age groups for maintaining improvements in bone mineral density that resulted from exercise training.

Warm-Up and Cooldown

The effect of warming up and cooling down on bone density is not known. However, warming up and stretching are important for ligaments and tendons, which are part of the skeletal system.

In summary, the optimal exercise prescription for skeletal health varies over the life span, and research continues to further elucidate what types of activity are most effective for enhancing bone health. Clearly, weight-bearing or impact-loading exercise is beneficial, and most healthy individuals should undertake an exercise program using weight-bearing activity and dynamic resistance exercise.

Skeletal Adaptations to Exercise

Training

The adaptation of the skeletal system to exercise training is depicted in **Table 16.6**. As the table indicates, the adaptation of bone to exercise depends largely on the amount of activity and may be represented as a continuum. Measurable skeletal adaptation also depends on the type of bone being measured (trabecular or cortical) as well as the type of activity employed.

TABLE 16.6 Effects of Physical Activity and Exercise Training on Bone Health

| No or Too Little Activity | Acceptable Amounts of Activity | Too Much, High-Intensity, Long-Duration, Rapid Increment Activity |
|---|--|--|
| Low mineralization and density | Adequate mineralization or enhanced mineralization and density | Osteopenia or osteoporosis in amenorrheic athletes |
| Aging-associated osteoporosis | Normal rate of growth, stature, and proportion | Fragile bones, increased risk of fracture |
| Fragile bones, increased fracture potential | Delayed aging-associated osteoporosis | Overuse injuries include those to the elbow (bony spurs or bone disruption at joint surface), the vertebrae (microfractures leading to slippage), the knees (Osgood-Schlatter diseases: inflammation of the bones or cartilage), and the legs and feet (stress reactions and/or fractures) |

One approach to studying the effects of increased physical activity on bone density is to compare the dominant limb to the nondominant limb in sports such as tennis and baseball. These studies report that the dominant arm has greater bone mineral density or mass than does the nondominant arm ([Huddleston et al., 1980](#); [Jones et al., 1977](#); [Kontulainen et al., 2002](#)). This seems true for both females and males and across a wide age range. Furthermore, the difference in BMD between the dominant and nondominant arm appears related to the age at which participants started playing the sport. [Kontulainen et al. \(2002\)](#) have reported that BMD measures of the humerus of the dominant arm are approximately 17% greater than those in the nondominant arm in racquet sport players who began playing before menarche, compared to a 9% difference between dominant and nondominant limbs in players who began playing after menarche. The control group of nonathletic individuals evidenced a 3% difference in BMD between the dominant and nondominant arm ([Kontulainen et al., 2002](#)).

Another approach to studying skeletal adaptations to exercise training has been to compare different athletic groups with one

another and with control groups (see accompanying Focus on Research). These studies collectively suggest that individuals involved in athletics or participating in vigorous fitness training have greater bone mineral density than do sedentary controls. Furthermore, individuals involved in weight-bearing or impact-loading sports have higher BMD than do those involved in non-weight-bearing activities (ACSM, 2004; Duncan et al., 2002; Prelack et al., 2012; Proctor et al., 2002; Riser et al., 1990).

FOCUS ON APPLICATION

Plyometrics

Again, high-impact activities are thought to be the key for bone development. *Plyometrics* is an exercise training method originally designed to develop explosive power. It consists of exercises involving powerful contractions following dynamic loading or prestretching of the contracting muscles. Plyometrics depth jumps can produce ground reaction forces of up to seven times body weight. In terms of skeletal loading, ground reaction forces less than two times body weight are considered to be low intensity, ground reaction forces two to four times body weight are moderate intensity, and ground reaction forces greater than four times body weight constitute a high-intensity load (Witzke and Snow, 2000). Thus, plyometrics provides a potentially excellent training modality for building bone mineral density.

Basic plyometric exercises include all the following:

- Bounds for horizontal distance
- Jumps for vertical height
- Hops for vertical height as rapidly as possible
- Leaps for maximal vertical plus horizontal distance
- Skips for vertical height plus horizontal distance
- Ricochets for rapid leg and foot movement while minimizing vertical and horizontal distance
- Swings for trunk movements with involvement of

shoulders and arms

- Twists for lateral movement without shoulder or arm involvement

A program of plyometrics can be used with individuals as young as 12 years of age if designed carefully and applied progressively (Radcliffe and Farentinos, 1985). Witzke and Snow (2000) implemented a plyometrics training program in a freshman physical education class (3 d·wk⁻¹, 30–45 min·d⁻¹, for 9 months). All participants volunteered for this class. Despite the fact that the control subjects in a “traditional” physical education class were active more hours per week outside class than the plyometrics exercisers (5.6 vs. 2.6 hr·wk⁻¹), the plyometrics exercisers had greater increases in bone mass measured at all sites. However, the difference between groups was statistically significant at only one site, the greater trochanter. The researchers reported that although some students worked harder in class than others, most enjoyed the plyometrics class and participated in it safely. Only one injury occurred during the entire program. Thus, plyometrics training may be a viable alternative activity for junior and senior high school physical education classes or after-school programs in fitness facilities. The implementation of such programs requires a knowledgeable instructor and a conscientiously constructed training program.



Sources: [Radcliffe and Farentinos \(1985\)](#); [Witzke and Snow \(2000\)](#).

Training studies have also been conducted of sedentary individuals beginning an exercise program. BMD measurements were compared before and after the exercise training. A review of 21 longitudinal studies in which participants were randomly assigned to exercise treatment or control groups strongly suggests that regular physical exercise can delay the physiological decrease in bone mineral density that occurs with aging and reduce the risk of osteoporosis. Weight-bearing exercises, including weight lifting, jumping, and running, were associated with the greatest improvements in bone mass ([Ernst, 1998](#)).

Skeletal adaptation to exercise depends on the age of the participant. Vigorous exercise helps increase bone mass and strength in children and is thus important for the attainment of peak bone mass. Furthermore, bone mass tracks from childhood to adulthood, as shown by the following studies. One study

(Barnekow-Bergkvist et al., 2006) tested female students at age 16.1 and 20 years later. Active girls had higher BMD than inactive girls as adolescents. Those who continued to be active in weight-bearing activity had significantly higher BMD (5–19%) in adulthood than did those who ceased participation or who had never been active. Membership in a sports club and site-specific physical performance in adolescence were significantly associated with higher adult BMD. Another study (Delvaux et al., 2001) tested males at ages 13 and 40 years. Static arm strength, running speed, and upper body muscular endurance as an adolescent contributed significantly to the prediction of adult bone mass. Additionally, no consistent evidence suggests that exercise training negatively affects either skeletal maturation (measured by ossification) or bone length in growing children. Although isolated studies have shown both retarded and accelerated growth in stature in young athletes, the consensus is that youngsters involved in exercise training grow at the same rate and to the same extent as do their sedentary counterparts (Baxter-Jones and Maffulli, 2002; Caine, 1990; Malina, 1988; Plowman et al., 1991; Sprynarova, 1987).

A study by Welsh and Rutherford (1996) suggests that elderly men and women respond to exercise in a similar manner. High-impact aerobics, performed 2–3 d·wk⁻¹, resulted in an increase in total body BMD and in hip and spine BMD. The increase in BMD was similar for the males and females who participated in the 12-month study.

Overall, studies suggest that weight-bearing and resistance exercise training play an important role in maximizing bone mass during childhood and adolescence, increasing or maintaining bone mass through adulthood, attenuating bone loss with aging, and reducing falls and fractures in the elderly (ACSM, 2004; Polidoulis et al., 2012). However, if some is good, more is not necessarily better when it comes to exercise training and bone health. Excessive physical activity, noted as the pathological overload zone in **Figure 16.11**, can exceed the adaptive ability of bone, resulting in overuse injuries.

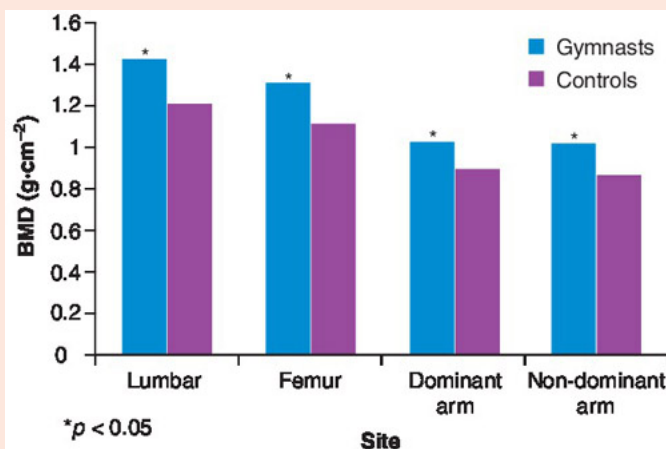
Gymnastics Are Associated with Higher Upper Limb BMD

To examine the effect of high-impact-loading forces on BMD, Proctor and colleagues examined female college gymnasts (NCAA Division I) and age- and weight-matched controls. Gymnastics is a unique sport because the limbs of the upper body are weight bearing and subject to high-impact loading.

Following are the BMD figures of the gymnasts and controls.

As found in earlier studies focusing on the lower body of weight-bearing athletes, this study found that gymnasts had higher BMD values at the lumbar spine and femur than did controls. The major new finding was that female collegiate gymnasts had 17% higher arm BMD than did controls. Furthermore, while the control group had the typical pattern of slightly greater BMD in the dominant arm, this difference was not evident in the gymnasts.

These results are consistent with the mechanostat theory and suggest that gymnastic training provides a sufficient training stimulus (overload) to elicit positive bone adaptations.



Source: Proctor, K. L., W. C. Adams, J. D. Shaffrath, & M. D.

Van Loan: Upper-limb bone mineral density of female college gymnasts versus controls. *Medicine & Science in Sports & Exercise*. 34(11):1830–1835 (2002).

Special Applications to Health and Fitness

The following sections discuss three practical implications of skeletal health of special interest to those involved in health and fitness: osteoporosis, the female athlete triad (a portion of the relative energy deficiency in sport Syndrome), and skeletal injuries.

Osteoporosis

Osteoporosis is the most common bone disease, and it represents a serious health problem affecting millions of Americans. The prevalence of osteoporosis (at the femoral neck, lumbar spine, or both) was 12.6% for adults over age 50 years and 17.7% among adults over age 65 years based on data collected in 2017–2018 ([Sarafrazi et al., 2021](#)). These data also reveal that women have a higher prevalence of osteoporosis than men. Osteoporosis is a dangerous condition that increases the risk of fracture, which can be fatal and which can lead to long periods of immobilization that lead to loss of muscle mass and increased cardiovascular risk. Osteoporosis is also associated with significant economic burden. A report commissioned by the [National Osteoporosis Foundation](#) reported that approximately 1.8 million Medicare beneficiaries suffered osteoporotic fractures in 2016 and that over 5.7 billion dollars were paid for subsequent fractures following the initial bone fracture ([Millman Report, 2021](#)). The U.S. National Bone Health Alliance (NBHA) is a public-private partnership that recognizes the burden of osteoporosis and seeks promote bone health and prevent disease; improve bone disease diagnosis and treatment; and enhance bone research and surveillance, through collaboration between the government, nonprofit, and for-profit sectors ([Lee et al., 2013](#)).

Osteoporosis, meaning “porous bones,” is a condition characterized by compromised bone strength that increases the risk of fracture. Bone strength is determined by both bone density and bone quality (including external geometry and internal microstructure) (Seeman and Delmas, 2006). Osteoporosis results from an imbalance between bone resorption and bone formation and is characterized by low bone mass and increased bone fragility and susceptibility to fracture (Klein-Nulend et al., 2015). Low bone mass, or bone mineral density, occurs because resorption occurs faster than formation. **Figure 16.12** shows normal and osteoporotic trabecular bone. Note the more porous trabeculae in the osteoporotic bone, a condition that weakens the bone.





B

Figure 16.12 Trabecular Bone.

A. Normal. B. Osteoporotic.

The most common fracture sites related to osteoporosis are the hip, spine, and wrist. Additionally, spinal vertebral fractures occur when an osteoporotic bone is literally crushed by the weight of the body, resulting in a loss of height, curvature of the spine, and considerable pain. The number, cost, and consequences of fracture warrant concern.

The cause of osteoporosis is not known, although several risk factors have been identified (**Table 16.7**) (Carmona, 2004; NIH, 2014; Sarafrazi et al., 2021; Weaver et al., 2016). Genetic risk factors include sex, race, heredity, and body build. Women are more likely to develop osteoporosis; those with a parent with osteoporosis are more likely to develop osteoporosis; and a small body frame (low body mass index [BMI]) is associated with increased risk of osteoporotic fracture in the elderly. Low BMI may result in several factors, including low BMD, less soft tissue to protect bone from impact forces, and an increased risk of falling due to muscle weakness (Nielson et al., 2012). The relationship between race/ethnicity and osteoporosis is challenging to quantify as researchers have noted different rates of fracture and BMD among men and women of different

geographical regions (cross-country comparisons) and among racial/ethnic groups within the United States, and many of these differences may be a result of other mitigating socioeconomic or cultural influences (Cauley, 2011). Nutritional risk factors include low calcium intake, excessive alcohol consumption, and consistently high protein intake. Lifestyle factors associated with osteoporosis include a lack of physical activity and smoking. Physiological factors include inadequate levels of estrogen (related to a delayed menarche, amenorrhea, or an early menopause). Several research studies have indicated that the rapid loss of BMD following menopause is related to the decrease in estrogen levels. The low activity levels of females in the United States, particularly older women, also contribute to the risk of developing osteoporosis. Bone mineral content is positively related to long-term physical activity.

TABLE 16.7 Risk Factors Associated with Osteoporosis

| Risk Factors | Relationship to Disease | Possible Explanations |
|--|---|---|
| Genetic | | |
| Race | Whites are more likely to develop disease than African Americans | Unknown |
| Sex | Women are four times more likely to develop osteoporosis than men | Lighter bones of females; rapid bone loss following menopause; longer life span |
| Heredity | There appears to be a genetic predisposition to the disease | Unknown |
| Body build | Petite individuals are at greater risk | Less peak bone mass to lose |
| Lifestyle | | |
| Lack of physical activity | Lack of weight-bearing activity increases risk of disease | No weight-bearing activities to stimulate bone formation |
| Smoking | Smoking increases risk of disease | May lower serum estrogen or cause early menopause |
| Sex hormones (late menarche, amenorrhea, or menopause) | Decrease in sex hormones is associated with increased risk | Loss of protective effect of estrogen on bone |
| Nutritional | | |
| Calcium intake | Low calcium level interferes with bone formation | Insufficient calcium to adequately ossify bone |
| Alcohol use | Alcohol use may be damaging to bone | Resulting in poor diet from excessive alcohol consumption |
| Protein intake | High protein intake is associated with low BMD | Protein increases calcium loss in urine |

The American College of Sports Medicine recommends that older adults acquire 150 min·wk⁻¹ of physical activity. If older adults are not able to achieve 150 min·wk⁻¹ of moderate-intensity activity because of chronic health conditions, they should be as physically active as their abilities and conditions allow (ACSM, 2009). These recommendations are made based on the understanding of multiple benefits of exercise, including

enhancing bone health. In addition to its positive effect on BMD, exercise also appears to help reduce the risk of fractures by increasing muscular strength and coordination, thereby decreasing the risk of falling (ACSM, 2022; [Kemmler et al., 2010](#)). The risk of falling is affected by the individual's coordination, sight, and muscular strength. Environmental risk factors that increase the risk of falling include poor lighting, uneven or slippery floor surfaces, and inappropriate footwear. Exercise leaders must provide an exercise area that reduces the risk of falling for older adults. While there is still insufficient evidence about the optimal exercise regime for individuals who have or who are at risk for osteoporosis, the ACSM recommends aerobic activity that lead to bone loading, including aerobic and resistance exercise (ACSM, 2022).

While it is clear that physical activity is important for maintaining bone health in older adults, nutritional factors also play a role. Calcium and vitamin D have a permissive effect on bone that acts synergistically with exercise to influence bone remodeling. There is no evidence that excessive amounts of calcium or vitamin D will result in greater skeletal gains, and in the absence of exercise, nutritional factors cannot adequately maintain bone mass or strength. However, adequate amounts of calcium, vitamin D, and protein are independently important for bone health ([Daly and Kukuljan, 2010](#); [Rizzoli et al., 2014](#); [Weaver et al., 2016](#)).

The Female Athlete Triad: A Portion of the Relative Energy Deficiency in Sport Syndrome

Postmenopausal females are not the only people at risk for developing osteoporosis and subsequent bone fractures. So too are young female athletes who exhibit components of a medical syndrome known as the female athlete triad ([Ackerman and Misra, 2011](#); [ACSM, 2007](#); [Beals and Meyer, 2007](#); [DeSouza and Williams, 2004](#); [International Olympic Committee, 2006](#)).

The **female athlete triad** was first recognized as a syndrome in 1997. In 2005, the International Olympic Committee (IOC) published a position stand on it. In 2007, the American College of

Sports Medicine refined the syndrome as the condition presented in **Figure 16.13**. That is, the syndrome was seen clinically as a collection of interrelated conditions including disordered eating (DE), menstrual dysfunction, and skeletal demineralization. The syndrome was originally narrowly defined in terms of the extremes of each of these conditions, but by 2007, it was recognized that each condition ranged along a continuum from health to disease with interaction between the factors (Beals and Meyer, 2007; DeSouza et al., 2014; Loucks, 2003b; Zach et al., 2011). Thus, disordered eating was seen as being directly related to both menstrual dysfunction and skeletal demineralization, while menstrual dysfunction in turn impacted skeletal demineralization. Now, however, there is compelling evidence that low energy availability resulting from disordered eating (DE) or a diagnosed eating disorder (ED) is the underlying factor in the female athlete triad. Moreover, as detailed in Chapter 6 (Figure 6.11), the resultant psychological/physiological, fitness/performance, and medical issues resulting from DE/ED are probably not confined to just menstrual dysfunction and bone demineralization. Many more systems and functions are impacted by low energy availability, including the cardiovascular system where endothelial function is impaired in those with DE/ED. These findings have led the International Olympic Committee expert panel to introduce a broader, more comprehensive term for the condition previously known as the female athlete triad. That term is the *relative energy deficiency in sport syndrome (RED-S)* (Mountjoy et al., 2014) and, again, this is discussed fully in Chapter 6. However, this does not negate the interaction between low energy deficiency and menstrual and bone health. For purposes of this chapter, the discussion will center around the impact of low energy availability and menstrual disruption on bone health for females. Male athletes can also be affected by energy deficiencies/low energy availability leading to functional hypogonadism and osteoporosis or low bone mineral density; however, the clinical outcomes associated with the Male Athlete Triad are less clearly defined (Bratland-Sanda & Sundgot-Borgen, 2013; Loucks et al., 2006; Nattiv et al., 2021).

Female Athlete Triad A portion of the relative energy

deficiency in sport syndrome that includes the interrelated conditions of disordered eating, menstrual dysfunction, and skeletal demineralization.

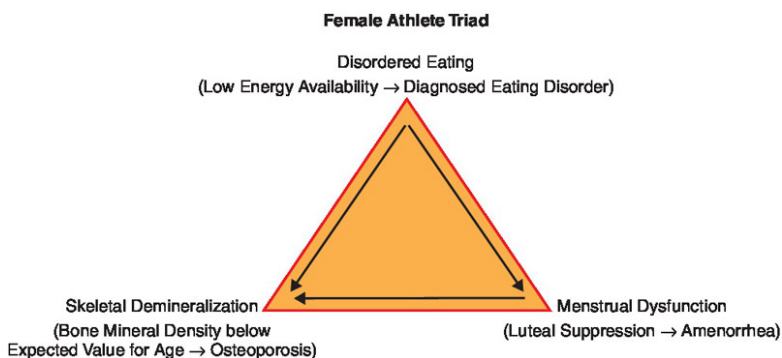


Figure 16.13 The Female Athlete Triad.

The female athlete triad is a portion of the relative energy deficiency in sport syndrome whose underlying causal factor is some degree of disordered eating that brings about menstrual dysfunction and skeletal demineralization. In the extreme, these conditions can result in a diagnosed eating disorder, amenorrhea, and osteoporosis. The *arrows* indicate that disordered eating can directly impact both menstrual dysfunction and skeletal demineralization, whereas menstrual dysfunction directly impacts only skeletal demineralization. **Sources:** Based on information from [Beals and Meyer \(2007\)](#); [Loucks \(2003b\)](#); [Mountjoy et al. \(2014\)](#).

Low Energy Availability

Energy availability (EA) is defined as the amount of dietary energy remaining for all other metabolic processes after the energy cost of training is subtracted from the daily energy intake. This is the amount of dietary energy remaining for all other physiological functions. Humans need dietary energy for five functions: cellular maintenance, thermoregulation, movement, growth, and reproduction. If a large proportion of the available energy is used for movement (exercise training), there may be

insufficient energy available for the other functions. Reproductive functions seem particularly vulnerable to the effects of insufficient energy, although more readily perceived in the female than the male (Bratland-Sanda & Sundgot-Borgen, 2013).

Energy Availability (EA) The amount of dietary energy remaining for all other metabolic processes after the energy cost of training is subtracted from the daily energy intake.

In exercising females, reproductive function and bone turnover are impaired when energy availability is decreased more than 33%. Many amenorrheic athletes decrease their energy availability by 67%, well over the threshold 33%. The threshold for sufficient energy availability appears to be approximately 30 kcal·kg LBM⁻¹·d⁻¹. Thus, female runners may be able to sustain normal reproductive hormonal function while running up to 8 mi·d⁻¹ as long as they maintain a dietary energy intake of at least 45 kcal·kg LBM⁻¹·d⁻¹ (Ihle and Loucks, 2004; Loucks et al., 2006; Loucks and Thuma, 2003; Sungot-Borgen et al., 2013).

EXAMPLE

For example, if the runner were 125 lb (56.8 kg) with a body fat of 20%, she would have 25 lb (11.4 kg) of fat and 100 lb (45.4 kg) of lean body mass (LBM). Multiplying 45.4 kg by 45 kcal·kg LBM⁻¹·d⁻¹ indicates she requires a caloric intake of 2,043 kcal·d⁻¹.

If this athlete is only ingesting 1,200 kcal·d⁻¹, she has a deficit of 843 kcal·d⁻¹. Among the results of such insufficient energy availability are impaired bone health and the possibility of increased stress fractures.

Menstrual Dysfunction

Menstrual dysfunction is one mechanism through which low

energy availability is thought to impact bone health. It is thought that insufficient fuel (especially carbohydrate) to meet energy requirements causes a change in brain function. The female reproductive system is controlled by the hypothalamus-pituitary-ovarian axis. A key role is the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus. Low energy availability somehow decreases GnRH release. Inadequate GnRH results in the suppression of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which causes, in turn, inadequate secretion of estrogen (estradiol) and progesterone. The resultant disruptions in menstrual function can include (1) luteal suppression (phase defects in which ovulation occurs but implantation cannot), (2) anovulation, (3) oligomenorrhea (irregular and inconsistent menstrual cycles), and (4) amenorrhea. Amenorrhea may be either primary (the failure to achieve menarche by age 15) or secondary (no menses for a minimum of 3 consecutive months in a female who has attained menarche) (DeSouza and Williams, 2004; Loucks et al., 2006).

While the athlete often feels the absence of a regular menstrual cycle is a good thing, physiologically it is not. A major concern with amenorrhea is that the low levels of circulating estrogen (hypoestrogenemia) can negatively impact bone (Giannopoulou and Kanaly, 2004). This is true for both primary amenorrhea (almost half of bone mass accrual occurs during adolescence and young adulthood) and secondary amenorrhea. An athlete who misses more than six consecutive menstrual periods has increased risk of failure to reach potential peak bone mass or premature bone loss. Indeed, athletes should have a BMD similar to or higher than (depending on the site and sport) sedentary individuals and nonmenstruating athletes, not lower, and regularly do (Ackerman and Misra, 2011; Beals and Meyer, 2007; DeSouza and Williams, 2004; Giannopoulou and Kanaly, 2004; International Olympic Committee, 2006). **Figure 16.14** presents data on bone mineral density at several sites in amenorrheic and eumenorrheic (regular menstrual cycling) athletes. This study demonstrates the important point that amenorrhea results in lower BMD at multiple sites.

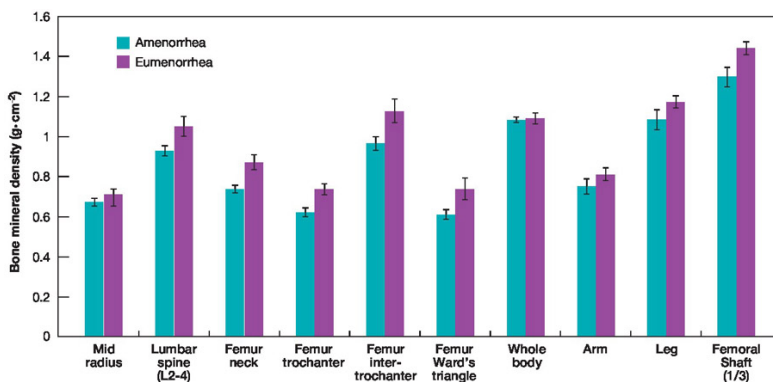


Figure 16.14 The Effects of Exercise-Induced Amenorrhea on Bone Mineral Density.

Source: Data from [Myburgh et al. \(1993\)](#).

Skeletal Demineralization

Given the relationship between menstrual function and bone health, the International Olympic Committee (IOC, 2006; [Sungot-Borgen et al., 2013](#)) suggests that any athlete identified with a current or past history of oligomenorrhea, amenorrhea, or fractures be further evaluated. Their osteoporosis decision tree is presented in **Figure 16.15**. Note that the IOC recommends that individuals with a Z-score of -1 or lower be referred for counseling or treatment. A Z-score of -2.0 or lower in an amenorrheic athlete 20 years of age or older is considered to be osteoporotic, placing the athlete at risk for fractures ([Beals and Meyer, 2007](#); [International Olympic Committee, 2006](#)).

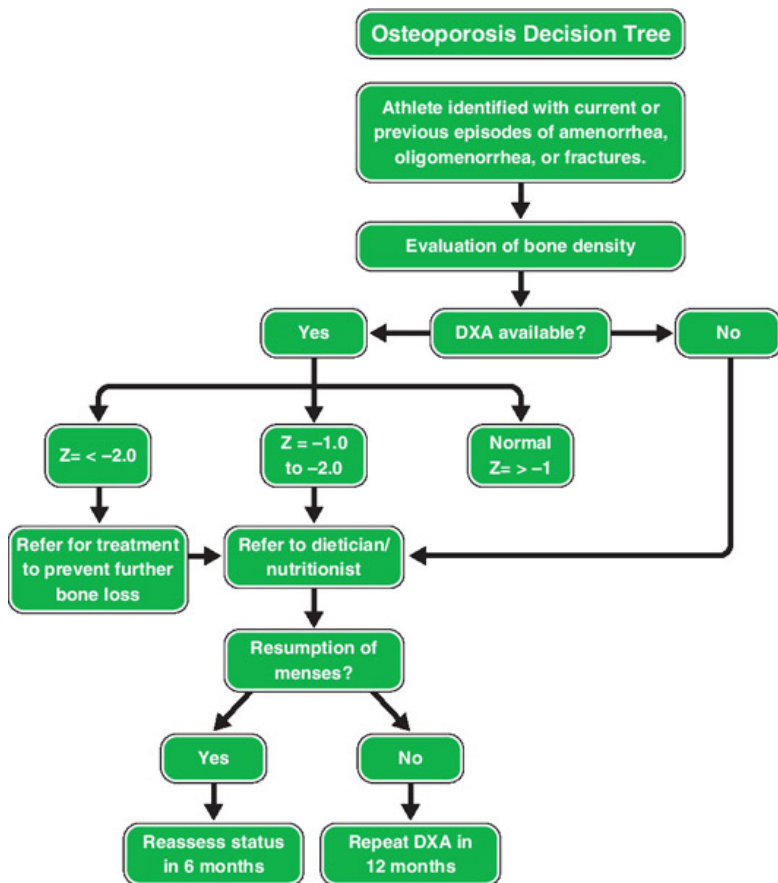


Figure 16.15 Osteoporosis Decision Tree.

Algorithm to decide whether an athlete needs evaluation and possibly treatment for low bone density.

The link between skeletal demineralization and low energy availability may be a dose-response relationship. A study by [Ihle and Loucks \(2004\)](#) showed that markers of *bone formation* were suppressed at moderate levels of energy restriction, whereas markers of *bone resorption* increased only when the energy restriction was severe enough to suppress estrogen. The primary role of estrogen in bone metabolism is to reduce the rate of bone resorption ([Loucks, 2003b](#)). Low energy availability likely initially affects metabolic substrates and hormones other than estrogen (insulin, growth hormone, insulin-like growth factor,

cortisol, leptin, and thyroid) that are important in bone metabolism and, then, with more severe energy restriction, impacts estrogen as well (DeSouza and Williams, 2004).

Note that although exercise is a stressor and thus elicits neurohormonal responses in the body, exercise itself does not appear to cause the female athlete triad beyond the impact of its energy cost on energy availability. No particular body weight or body composition appears to be a critical threshold level beyond which a disruption of the hypothalamus-pituitary-ovarian axis occurs. Eumenorrheic and amenorrheic athletes both span a common range of body weight and composition (Beals and Meyer, 2007; Loucks, 2003a, 2003b). However, athletes with a body mass index (BMI) lower than 18.5 kg.m^{-2} (M & F) or body fat of less than 12% (F) or less than 5% (M) should be closely monitored (Sungot-Borgen et al., 2013). Finally, while estrogen is important for both menstrual and bone health, estrogen supplementation alone has not been shown to restore BMD to normal in amenorrheic athletes. The Clinically Relevant Focus on Research box presents a case study demonstrating the roles of calcium, estrogen, and energy availability in treatment and recovery of low bone mineral density resulting from amenorrhea.

Skeletal Injuries

Skeletal injuries can be categorized as resulting from macrotrauma or microtrauma (Micheli, 1989). Macrotrauma injuries are sudden acute incidents, such as a broken leg or clavicle from impact or a fracture at the epiphyseal growth plate. Growth plate fractures have the greatest potential for harm, but fortunately, they are relatively rare. The probability of a growth plate injury is greatest in automobile accidents, falls, contact sports, and dynamic resistance training. Fractures of the growth plate can result in progressive bone shortening, deformity, or joint incongruity. Fortunately, acute traumatic growth plate injuries are less frequent than other injury types, and most such injuries appear not to result in growth disturbances (Caine, 1990). The risk of growth rate injuries among adolescent athletes appear to be related to periods of rapid growth (Wik et al., 2020).

Microtrauma injuries are overuse injuries from chronic

repetitive overtraining (Micheli, 1989). The anatomical site of microtrauma depends on the sport. Microtrauma injuries to bone generally involve an uncoupling or imbalance between bone resorption and bone deposition, called a stress reaction. **Stress reactions** refer to maladaptive areas of bone hyperactivity where resorption progressively exceeds deposition.

Stress Reactions Maladaptive areas of bone hyperactivity where the balance between resorption and deposition is progressively lost such that resorption exceeds deposition.

Early minor stress reactions may have no clinical manifestations. The individual feels no pain and has no swelling or tenderness. As the overuse continues and the imbalance becomes more extreme, several clinical symptoms may occur, including degeneration and loosening of portions of bone from the joint capsules, the formation of bone spurs, inflammation of bone and cartilage, and/or stress fractures. A **stress fracture** is a hairline break in bone that occurs without acute trauma, is clinically symptomatic, and is detectable by x-rays or bone scans. It is often difficult to determine exactly when a bone's stress reaction becomes a stress fracture. The typical fine hairline fracture may be undetectable by x-rays or bone scans for 3–4 weeks after pain occurs.

Stress Fracture A fine hairline break in bone that occurs without acute trauma, is clinically symptomatic, and is detectable by x-rays or bone scans.

Although exercise training can and does have a beneficial impact on bone growth and health, too much exercise training, usually in the form of repetitive overuse or rapid increments of intensity or duration, can be detrimental. Stress fractures are relatively common, accounting for 10% of all sports injuries requiring an appointment with a health care provider and responsible for approximately 30% of all running-related injuries. Stress fractures are most common in the tibial diaphysis,

accounting for nearly 75% of all stress fractures ([Robertson and Wood, 2015](#)). Harm is more likely if the bone already has a low bone mineral density. The concern about overuse injuries therefore focuses on females exhibiting the female athlete triad, males and females with disordered eating or low energy availability, and young growing athletes of both sexes ([Sterling et al., 1992](#)).

The concern for amenorrheic athletes is well founded. A higher prevalence of stress fractures has been documented in this population than in female athletes with normal menstrual cycles in a variety of sports ([Lloyd et al., 1986](#); [Maffulli, 1990](#); [Micheli, 1989](#); [Myburgh et al., 1990](#)).

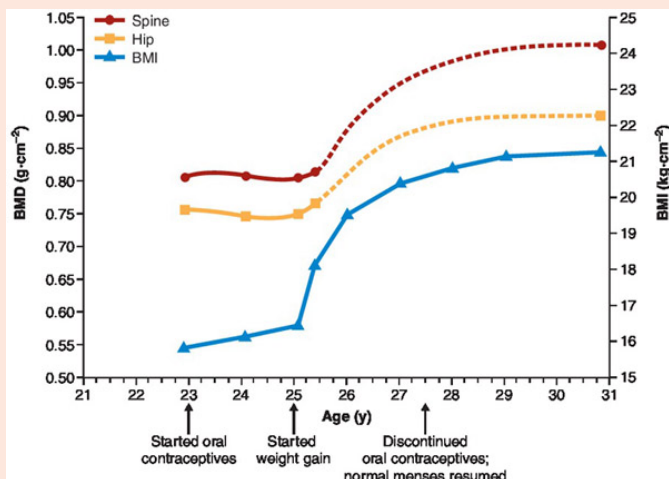
The exact incidence of microtrauma, including stress fractures, in youth athletes is difficult to document, although reports from orthopedists indicate that the number is growing as more children and adolescents participate in competitive sports ([Faulkner et al., 1993](#)). It has been reported that the highest incidence of stress fractures occurs between the ages of 10 and 15, at the time of peak growth ([Lloyd et al., 1986](#); [Sterling et al., 1992](#)). Some researchers have suggested that a normal imbalance between bone matrix formation and mineralization occurs during the growth spurt. Furthermore, during this growth spurt, muscle imbalances can occur around joints as the muscles, tendons, and ligaments are stretched and become progressively tighter when the bones elongate. A muscle that is fatigued from overuse, is weak or out of balance with its antagonist, and/or is inflexible is less able to absorb shock, allowing abnormally high stress to be transmitted to the bone. This situation increases the risk of stress fractures, other repetitive stress reactions, and impact injuries ([Jones et al., 1989](#); [Kibler et al., 1992](#); [Maffulli, 1990](#)). The treatment of stress fractures relies on modifying activity and allowing the body to rest in order to halt progressive damage while the bone heals ([Wang et al., 2015](#)). The situation is more complex, of course, if the reduced energy availability in sport (RED-S) syndrome is the underlying causal factor.

Reversing the Female Athlete Triad Portion of the Relative Energy Deficiency in Sport Syndrome

The athlete in this case study was an elite Caucasian female distance runner. She started running competitively at age 12 (before menarche) and competed until age 25. Her personal best at the 26.2 mi marathon distance was 2:41. She began restrictive eating at age 13 years. At times, her training volume was 90 mi·wk⁻¹. She exhibited primary amenorrhea until age 23, when she began calcium supplementation and estrogen replacement in the form of oral contraceptives. At that time her BMD (shown in the graph accompanying) was only 74% (T-score = -2.5) and 80% (T-score = -1.54) of normal peak for her spine and hip, respectively. Her body weight was 107 lb (48.6 kg), and her body mass index (BMI) (shown in the graph accompanying) was 15.8 kg·m⁻². Over the next 2 years, her body weight increased 4 lb (1.8 kg), and menarche occurred. However, not until she increased her energy availability by adopting better nutritional habits and decreasing her training mileage did she gain weight and increase her BMD (see graph). By age 31, she weighed a healthy 144 lb (BMI = 21.3 kg·m⁻²), and her BMD values were almost in the normal range (spine = 94%; T-score = -0.63 and hip = 96%; T-score = -0.33). As the authors state, convincing competitive athletes to gain weight is sometimes a “formidable challenge.” Indeed, in this case, the individual was 25 years old before finally acting on the seriousness of her situation. It would, of course, have been better to avoid the disordered eating and resultant female athlete triad/RED-S in the first place.

The extent to which bone deficits can be reversed is unknown, especially in athletes with secondary amenorrhea. In the case presented here, the positive changes started to occur within 2 years of menarche. Hormone replacement (in conjunction with calcium intake) was insufficient to regain bone density. Improved nutrition, weight gain, and resumption of normal menstrual cycles were necessary for

successful treatment.



Source: Reprinted with permission from Fredericson, M., & K. Kent: Normalization of bone density in a previously amenorrheic runner with osteoporosis. *Medicine & Science in Sports & Exercise*. 37(9):1481–1486 (2005). Copyright ©2005 The American College of Sports Medicine.

Complete the [Check Your Comprehension 2 Case Study](#).

CHECK YOUR COMPREHENSION 2-CASE STUDY

Allison is a sophomore in college. She ran cross country in high school but did not participate on the team in her first year of college. The summer before her sophomore year, she decided to improve her fitness so she could try out for the college cross-country team. About 3 weeks into her training program, she began to experience pain in her shins when she running. What is the most likely cause of this pain? What should she do about it?

Check your answer in [Appendix C](#).

The primary individual risk factor for overuse injuries is training error, particularly abrupt increases in intensity, duration, and frequency (Micheli, 1989). Workload should not increase more than 10% per week in young training athletes. Other non-RED-S risk factors include the aforementioned musculoskeletal imbalances of strength, flexibility, or size; errors in technique or skills; anatomical malalignments; footwear that fits improperly; and running on hard surfaces such as concrete and asphalt. Parents and others involved in youth sports must give careful attention to these factors. For example, during periods of rapid growth, the intensity of training should be reduced and static stretching programs emphasized.

Although the skeletal system is often taken for granted, it should not be. When it is injured or malfunctioning, we quickly become aware of its importance. Adequate nutrition, reasonable training regimens, and maintenance of normal hormonal levels are keys to good bone health for both sexes and all ages.

Summary

1. The skeletal system serves a number of important functions, including support, protection, movement, mineral storage, and hematopoiesis (blood cell formation).
2. Bone tissue is dynamic, living tissue that is constantly undergoing change. Bone remodeling is a continual process of bone resorption and formation of new bone.
3. Osteoclasts are bone cells that cause the resorption (breakdown) of bone tissue. Osteoblasts are bone-forming cells. Osteocytes are mature osteoblasts surrounded by calcified bone.
4. The two major types of bone tissue are cortical and trabecular bone; they differ in their microscopic appearance.
5. Studies generally show that physical activity has a positive effect on bone health. Exercise training can enhance the attainment of peak bone mass during late adolescence and early adulthood, can slow the rate of age-related bone loss in later adulthood, and may offset menopausal-related bone

loss.

6. Weight-bearing and impact-loading activities lead to an acute osteogenic effect in bone.
7. The mechanostat theory of bone response posits that a minimal effect strain is necessary to elicit normal remodeling of bone tissue and that a higher threshold exists for the enhancement of bone strength. This is consistent with the overload principle of training.
8. Weight-bearing or impact-loading activities produce greater changes in bone mineral density than do non-weight-bearing or weight-supported activities.
9. An appropriate exercise prescription for skeletal health should take into account the individual's current skeletal health. On the basis of current status and individual desires, activities should be chosen from a continuum of impact-loading activities.
10. Osteoporosis means “porous bones,” a condition characterized by a loss of bone mineral density, resulting in bones that are weak and susceptible to fracture. It is clinically defined as a bone mineral density greater than -2.5 standard deviations below young normal adult averages.
11. The female athlete triad is a portion of the relative energy deficiency in sport syndrome (RED-S). The underlying factor responsible for the female athlete triad is low energy availability resulting from disordered eating (DE) or a diagnosed eating disorder (ED). The two linked health consequences are menstrual dysfunction and skeletal demineralization. Low energy availability is directly linked to menstrual dysfunction through low estrogen levels and to skeletal demineralization by affecting metabolic substrates and hormones.

Review Questions

1. Compare and contrast cortical and trabecular bone.
2. Diagram the stages of bone remodeling, citing the specific

role of the different types of bone cells.

3. What is the relationship between the hormonal control of blood calcium levels and the hormonal control of bone remodeling?
4. Why are osteoporotic fractures more likely in bones with a higher percentage of trabecular than cortical bone?
5. Why are women more likely than men to suffer osteoporotic fractures?
6. What can be done during the growth years to optimize the attainment of peak bone mass? Why is the attainment of peak bone mass important?
7. Describe the acute changes in bone tissue that result from mechanical loading.
8. Explain how the mechanostat theory of bone relates to the following training principles: specificity, overload, and retrogression/reversibility.
9. What factors influence skeletal adaptations to exercise?
10. Describe how the female athlete triad relates to the relative energy deficiency in sport syndrome and why that is particularly relevant for bone health.
11. Describe how physical activity helps prevent osteoporosis.
12. Defend or refute the following statements:
 - a. Disturbances in bone growth frequently result from overtraining in young athletes.
 - b. Young athletes are more susceptible to stress fractures during the time of peak growth than at other times.

Literature Search

1. The skeletal system provides the framework for the body, and skeletal health is foundational to health and fitness. To better understand the research that has been done on this important issue, do a literature search using a search engine such as PubMed, Google scholar, or Web of Science.
 - a. Search Exercise (or Training) and Skeletal. This search

will yield many articles.

- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Resistance training and changes in BMD
 - ii. Training-induced skeletal changes in postmenopausal women
 - iii. Mechanostat theory applied to pre-pubescent children
 - iv. Continue your search for aspects of this topic that are of particular interest to you

For further review and study tools, visit Lippincott Connect.

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17 Skeletal Muscle System



CHAPTER OUTLINE

Introduction

Overview of Muscle Tissue

- Functions of Skeletal Muscle

- Characteristics of Muscle Tissue

Macroscopic Structure of Skeletal Muscles

- Organization and Connective Tissue

- Architectural Organization

Microscopic Structure of a Muscle Fiber

- Muscle Fibers

Molecular Structure of the Myofilaments

- Thick Filaments

- Thin Filaments

Contraction of a Muscle Fiber

The Sliding Filament Theory of Muscle Contraction

Excitation-Contraction Coupling

Structural Muscle Proteins

All-or-None Principle

Muscle Fiber Types

Contractile (Twitch) Properties

Metabolic Properties

Integrated Nomenclature

Assessment of Muscle Fiber Type

Distribution of Fiber Types

Fiber Type in Athletes

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Describe the functions of skeletal muscle tissue.
- Identify the characteristics of muscle tissue that make movement possible.
- Describe the macroscopic and microscopic organization of skeletal muscle tissue.
- Relate the molecular structure of myofilaments to the sliding filament theory of muscle contraction.
- Identify muscle proteins that determine the structural properties of the sarcomere.
- Discuss the role of titin as a spring-like protein and its contributions to residual force enhancement.
- Identify the regions of a sarcomere, and explain the changes that occur in these regions during contraction.
- Discuss the importance of specialized organelles, specifically, the sarcoplasmic reticulum, the T tubules, and the myofibrils.

- Explain the events involved in excitation-contraction coupling.
- Describe the sequence of events in the generation of force within the contractile elements.
- Differentiate muscle fiber types based on their contractile and metabolic properties.
- Discuss the ramifications of fiber-type distribution on the likelihood of success in a given athletic event.

Introduction

Muscle contractions provide the basis for all human movement. Movement also involves interactions among different body systems. For instance, the muscle cells (fibers) produce and utilize ATP to provide the energy for contraction and force production. The digestive, respiratory, endocrine, and cardiovascular systems must be operating effectively to provide muscle cells with the oxygen and nutrients needed to produce the energy. For the purposes of this chapter, it is assumed that these other body systems are functioning properly.

Overview of Muscle Tissue

Muscle tissue produces force through the interaction of its basic contractile elements—the myofilaments—which are composed primarily of protein. The three types of muscle tissue (skeletal, smooth, and cardiac) have different general functions. The force of contraction may be used for movement such as locomotion (skeletal muscle), the movement of materials through hollow tubes such as the digestive tract or blood vessels (smooth muscle), or the pumping action of the heart (cardiac muscle). Regardless of type, all muscle tissue can produce force because of certain basic characteristics. This chapter focuses on skeletal muscle.

Because skeletal muscles have various characteristics, they are often referred to by different names. Skeletal muscles are under conscious control and are often called *voluntary muscles*. Skeletal muscles are also sometimes referred to as *striated muscle* because

of the repeating pattern of light and dark bands seen in their microscopic structure. Additionally, to differentiate skeletal muscle fibers from intrafusal fibers found in sensory organs of the muscle (proprioceptors; see [Chapter 20](#)), physiologists sometimes refer to skeletal muscle fibers as *extrafusal muscle fibers*.

Functions of Skeletal Muscle

Although movement is the primary function of muscle tissue, the muscular system also has other important roles. In addition to locomotion and manipulation, skeletal muscles maintain body posture, assist in the venous return of blood to the heart, and produce heat (thermogenesis). Heat is a by-product of cellular respiration; because muscles use a great deal of energy for movement, they also generate a great deal of heat. Additionally, muscles act as energy transducers by converting biochemical energy from ingested food into mechanical and thermal energy. Skeletal muscles also help protect internal organs. Because muscles make up most of the protein in the body, they constitute a potential but rarely used form of stored energy. The use of protein as an energy substrate is discussed in the metabolism unit.

Characteristics of Muscle Tissue

The unique characteristics of muscle tissue are specifically suited to its primary function: converting an electrical signal into a mechanical event (contraction of muscle fibers). These characteristics include irritability, contractility, extensibility, and elasticity.

Irritability refers to the ability of a muscle to receive and respond to stimuli. The stimulus is usually a chemical message (from a neurotransmitter), and the response is the generation of an electrical current (action potential) along the cell membrane.

Contractility refers to the ability of a muscle to shorten in response to a stimulus. This shortening produces force. Muscle tissue is the only body tissue that can generate force.

Extensibility refers to the ability of a muscle to be stretched or lengthened. Stretching occurs when a muscle is manipulated by

another force. **Elasticity** refers to the ability of a muscle to return to its resting length after being stretched. Together, these characteristics of muscles allow for human movement. Skeletal muscle also contains satellite cells, located beneath the basal lamina but outside the muscle fibers. These cells are typically quiescent and play no known role in normal cell function. But, they are central to muscle fiber recovery from injury. Satellite cells are stem cells that can differentiate and form new muscle fibers, they are essential to regenerating new muscle cells after injury ([Lieber, 2010](#)).

Irritability The ability of a muscle to receive and respond to stimuli.

Contractility The ability of a muscle to respond to a stimulus by shortening.

Extensibility The ability of a muscle to be stretched or lengthened.

Elasticity The ability of a muscle to return to resting length after being stretched.

Macroscopic Structure of Skeletal Muscles

The human body has over 400 skeletal muscles, which account for 40–45% of the adult male body weight and 23–25% of the adult female body weight ([Hunter, 2000](#)). These muscles function together in remarkable ways to provide smooth, integrated movement for a wide variety of activities, many of which require

little conscious thought. Muscle action is also the basis of sport and fitness activities. To understand how muscles function in various sports and exercise activity, or in any other activity, it is necessary to look beneath the skin.

Organization and Connective Tissue

Skeletal muscles are organized systematically, as shown in **Figure 17.1**. Some of this organization is apparent to the naked eye, but other aspects are apparent only when muscle fibers are viewed through a microscope.

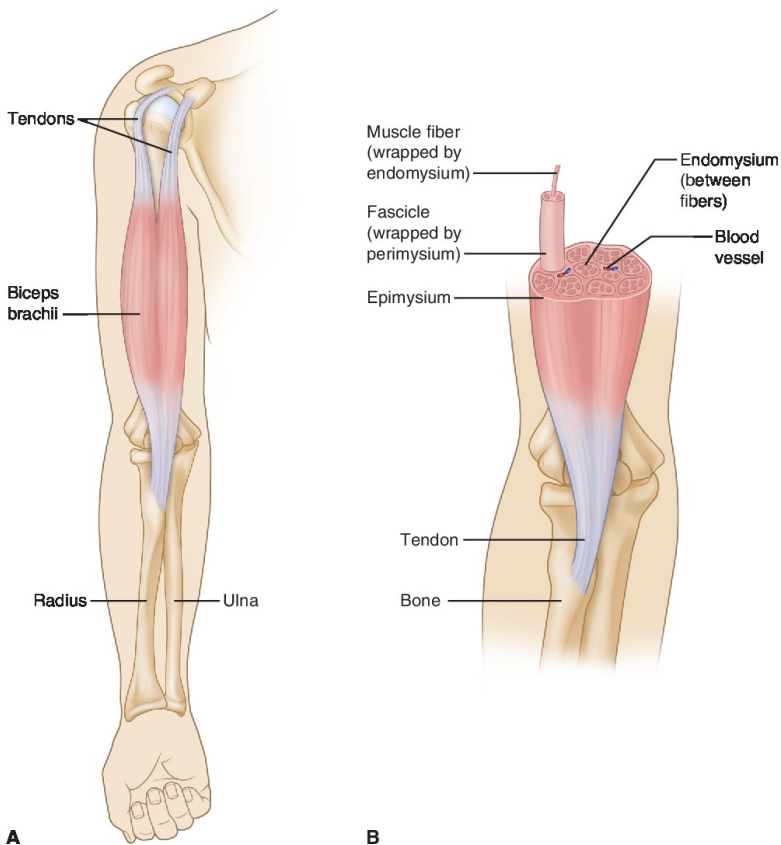


Figure 17.1 Organization of Skeletal Tissue.

A. Intact skeletal muscle. Biceps brachii is attached to bones through tendons. **B.** Connective tissue. The entire muscle is

surrounded by connective tissue called epimysium. The muscle is organized into bundles called fasciculi, which are surrounded by connective tissue called perimysium. Each fasciculus contains many individual fibers surrounded by connective tissue called endomysium.

Skeletal muscles are attached to bones by tendons, which allow the contraction of a muscle to move a bone. Each muscle is bound together by a thick layer (sheath) of connective tissue called *fascia*. Just beneath the fascia is a more delicate layer of connective tissue called *epimysium* that directly covers the muscle.

The interior of the muscle is subdivided into bundles of muscle fibers called *fasciculi* (singular: *fasciculus* or *fascicle*), which are also surrounded by connective tissue. The sheath of connective tissue that separates fasciculi within a skeletal muscle is called *perimysium*. The fasciculi are composed of many individual muscle fibers (cells), each of which is surrounded by its own sheath of connective tissue called *endomysium*.

The three layers of connective tissue (the epimysium, the perimysium, and the endomysium) provide the framework that holds the muscle together. These layers of connective tissue come together at each end of the muscle to form the tendons that attach the muscle to bone (see **Figure 17.1**). As a muscle contracts, it pulls on the connective tissue in which it is wrapped, causing the tendon to pull on the bone to which it is attached. **Myofascia** refers to the muscle and connective tissue collectively and reflects the reality that these two tissues are functionally entwined.

Myofascia refers to the muscle and band or sheet of connective tissue that encloses the muscle.

The connective tissue not only binds a muscle to a tendon to permit movement of a bony lever at a single joint but also provides a seamless connection along lines of movement in a body facilitating integration of smooth, coordinated movement across multiple joints. The central role of connective tissue in

health and movement is receiving increased attention among body workers, holistic medicine practitioners, and movement specialists. **Figure 17.2** depicts the concept of an integrated myofascial tract from the top of the head (frontal bone) to the bottom of the foot (plantar surface of the toes) emphasizing how fascia wraps muscle and connects muscle and bone along an entire tract (Myer, 2014).

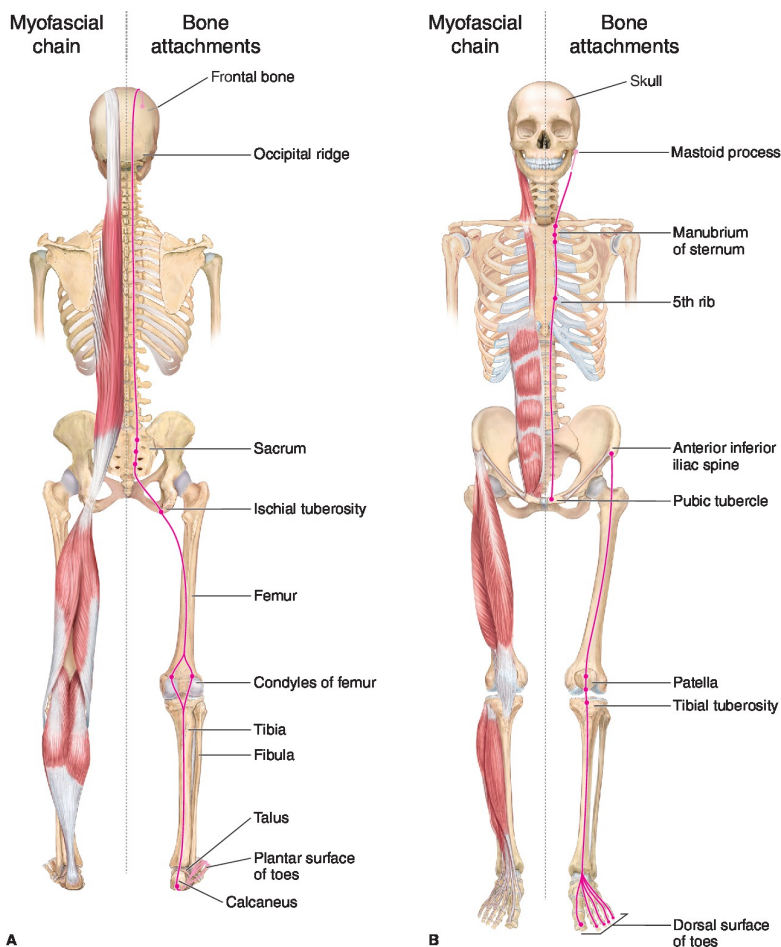


Figure 17.2 Example of a Myofascial Tract Extending along the Back (A) and Front (B) of the Body.

Source: Modified from Myer (2014).

Architectural Organization

Different arrangements of fasciculi within a muscle account for the different shapes of muscles. Muscles can be described as longitudinal, fusiform, radiate, unipennate, bipennate, or circular, as shown in **Figure 17.3**. The shape of a muscle in part determines its range of motion and influences its power production. Longer and more parallel muscle fibers, as are present in longitudinal muscles, allow for greater muscle shortening. Bipennate muscles, in contrast, shorten very little but are more powerful.


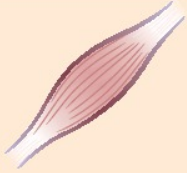
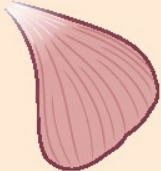

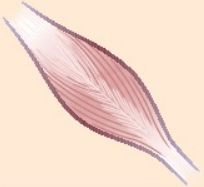

| Classification | Example | Diagram |
|----------------|-------------------------------------|---|
| Longitudinal | Sartorius |  |
| Fusiform | Biceps brachii |  |
| Radiate | Gluteus medius |  |
| Unipennate | Tibialis posterior |  |
| Bipennate | Gastrocnemius |  |
| Circular | Orbicular oculi (and sphincters) |  |

Figure 17.3 Arrangement of Fasciculi.

Microscopic Structure of a Muscle Fiber

Individual muscle fibers are composed primarily of smaller units called myofibrils, which are in turn made up of myofilaments. Refer to the organization of skeletal muscle shown in **Figure 17.4** as you read the following sections.

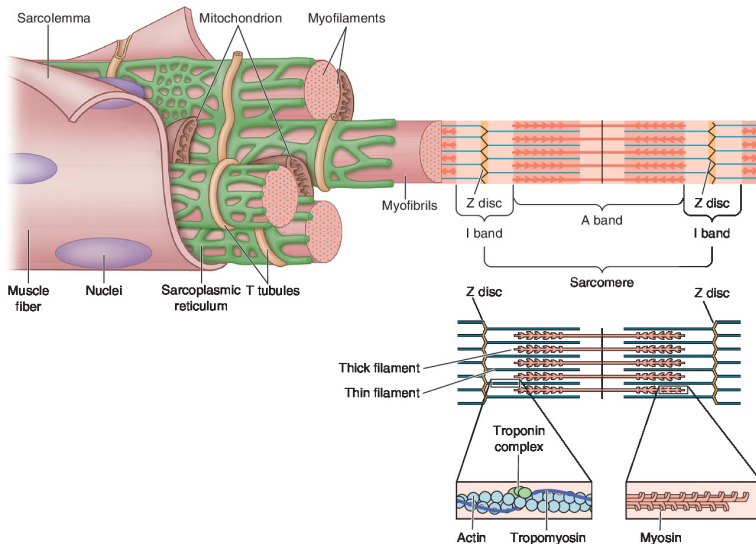


Figure 17.4 Organization of a Muscle Fiber.

There is a close anatomical relationship among the organelles, specifically the myofibrils, T tubules, and sarcoplasmic reticulum (SR). The repeating pattern of the myofibrils is due to the arrangement of the myofilaments.

Muscle Fibers

Muscle fibers, also called muscle cells, are long, cylinder-shaped cells ranging from 10 to 100 μm in diameter and 1–400 mm in length (Hunter, 2000; Marieb and Hoehn, 2019). The major structures of a muscle fiber and their functions are summarized in **Table 17.1**.

TABLE 17.1 Summary of Major Components of a Skeletal Muscle Cell

| Cell Part | Description | Function |
|------------------------|---|--|
| Nucleus | Multinucleated | Is the control center for the cell |
| Sarcolemma | Polarized cell membrane | Is capable of receiving stimuli from the nervous system |
| Sarcoplasm | Intracellular material | Holds organelles and nutrients; provides the medium for glycolytic enzymatic reactions |
| Organelles | | |
| Myofibrils | Rod-like structures composed of smaller units called myofilaments; account for 80% of muscle volume | Contain contractile proteins (myofilaments), which are responsible for muscle contraction |
| T tubules | Series of tubules that run perpendicular (transverse) to the cell and are open to the external part of cell | Spread polarization from the cell membrane into the interior of cell, which triggers the sarcoplasmic reticulum to release calcium |
| Sarcoplasmic reticulum | Interconnecting network of tubules running parallel with and wrapped around the myofibrils | Stores and releases calcium |
| Mitochondria | Sausage- or spherical-shaped organelles; numerous in a muscle cell | Are the major site of energy production |
| Cytoskeleton | Specialized proteins that provide much of the scaffolding of the muscle cell and provide structure to the sarcomere | Provides structure, elasticity, and signaling for the sarcomere and connects the sarcomere to other components of the cell |

A skeletal muscle fiber contains many nuclei, which are located just below the cell membrane. The *sarcolemma is the polarized plasma membrane* of a muscle cell, whose properties account for the irritability of muscle. The *sarcoplasm* of a muscle cell is similar to the cytoplasm of other cells, but it has specific adaptations to serve the functional needs of muscle cells, namely, increased amounts of glycogen and the oxygen-binding protein myoglobin.

A muscle fiber contains the same organelles found in other cells (including a large number of mitochondria) along with some specialized organelles. Organelles of specific interest are the transverse tubules (T tubules), the sarcoplasmic reticulum (SR), and the myofibrils. Myofibrils are composed primarily of the protein myofilaments and are responsible for the contractile properties of muscles. Skeletal muscle cells also have a highly organized complex cytoskeleton that provides the framework for the organelles and plays an important role in transmitting force from muscle tissue to bone.

Sarcoplasmic Reticulum and Transverse Tubules

Figure 17.4 illustrates the relationship among the myofibrils, the sarcoplasmic reticulum, and the transverse tubules. The **sarcoplasmic reticulum (SR)** is a specialized organelle that stores and releases calcium. It is an interconnecting network of

tubules running parallel with and wrapped around the myofibrils. (In **Figure 17.4**, the sarcolemma has been partially removed to illustrate the SR and myofibrils.) The major significance of the sarcoplasmic reticulum is its ability to store, release, and take up calcium and thereby control muscle contraction. Calcium is stored in the portion of the sarcoplasmic reticulum called the *lateral sacs* or *cisterns*.

Sarcoplasmic Reticulum (SR) The specialized muscle cell organelle that stores and releases calcium.

The **transverse tubules (T tubules)** are organelles that carry the electrical signal from the sarcolemma to the interior of the cell. T tubules are continuous with the sarcolemma and protrude into the sarcoplasm of the cell. As their name implies, T tubules run perpendicular (transverse) to the myofibril. Each T tubule runs between two lateral sacs of the sarcoplasmic reticulum, creating what is known as a triad; this ensures that the spread of an electrical signal (action potential) through the T tubules causes the release of calcium from the lateral sacs of the sarcoplasmic reticulum.

Transverse Tubules (T Tubules) Organelles that carry the electrical signal from the sarcolemma into the interior of the cell.

Myofibrils and Myofilaments

Each muscle fiber contains hundreds to thousands of smaller cylindrical units, or rod-like strands, called **myofibrils** (**Figure 17.4**). Myofibrils are specialized contractile organelles composed of myofilaments. These myofibrils, sometimes simply called fibrils, typically lie parallel to the long axis of the muscle cell and extend the entire length of the cell. Myofibrils account for approximately 80% of the volume of a muscle fiber.

Myofibril Contractile organelles composed of myofilaments.

As shown in **Figure 17.4**, each myofibril is composed of still smaller myofilaments (or filaments) arranged in a repeating pattern along the length of the myofibril. Myofilaments are contractile proteins (thick and thin) that are responsible for muscle contraction. **Myofilaments** account for most of the muscle protein. The repeating pattern of these myofilaments along the length of the myofibril gives skeletal muscle its striated appearance. Each repeating unit is referred to as a sarcomere.

Myofilaments Contractile (thick and thin) proteins responsible for muscle contraction.

Sarcomeres

A **sarcomere** is the functional unit (contractile unit) of a muscle fiber. As shown in **Figure 17.5**, each sarcomere contains two types of myofilaments. The *thick* filaments are composed primarily of the contractile protein myosin, and the *thin* filaments are composed primarily of the contractile protein actin. Thin filaments also contain the *regulatory proteins*, troponin and tropomyosin. Viewed with an electron microscope, the arrangement of myofilaments has the appearance of alternating bands of light and dark striations. The light bands are called *I bands* and contain only thin filaments. The dark bands are called *A bands* and contain thick and thin filaments, with the thick filaments running the entire length of the A band. The length of the thick filament thus determines the length of the A band.

Sarcomere The functional unit (contractile unit) of muscle fibers.

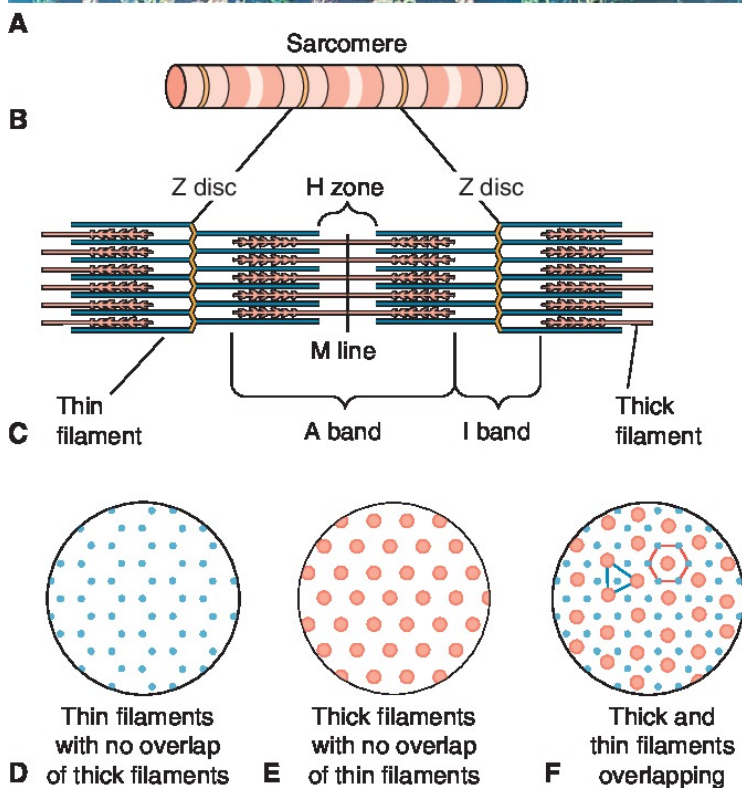
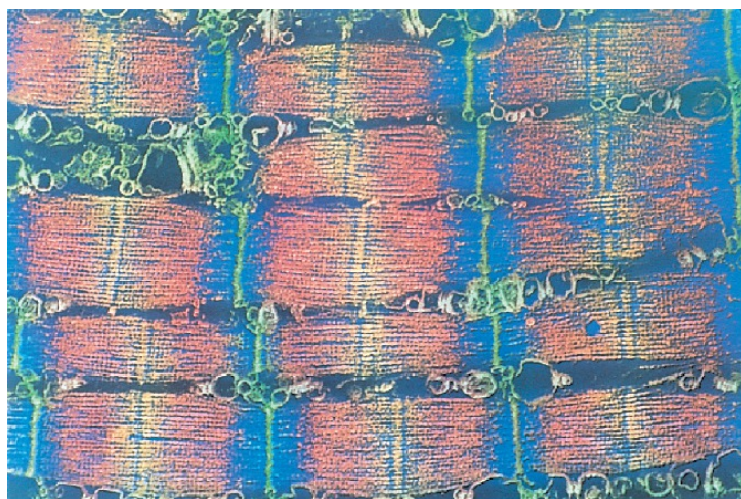


Figure 17.5 Arrangement of Myofilaments in a Sarcomere.

A. Micrograph of sarcomeres. B. Model of sarcomeres. C.

Relationship between thick and thin filaments. **D.** Cross-sectional view of thin filaments. **E.** Cross-sectional view of thick filaments. **F.** Cross-sectional view of thick and thin filaments.

The letter names of the various regions of the sarcomere derive from the first letter of the German word that describes the appearance of each. The names of the bands relate to the refraction of light through them. The I band is named for the word *isotropic*, which means that this area appears lighter because more light passes through it. The A band is named for its *anisotropic* properties, meaning that it appears darker because not as much light passes through. These properties relate to the types of filament present in the bands.

Each A band is interrupted in the midsection by an *H zone* (from the German *Hellerscheibe*, for “clear disk”), where there is no overlap of thick and thin filaments. Running through the center of the H zone is a dense line called the *M line* (from the German *Mittelscheibe*, for “middle disk”). The I bands are also interrupted at the midline by a darker area called the Z disk (from the German *Zwischenscheibe*, for “between disk”). A sarcomere extends from one Z disk to the successive Z disk. The Z disk serves to anchor the thin filaments to adjacent sarcomeres.

Myofilaments occupy three-dimensional space. The arrangement of the myofilaments at different points in the sarcomere is shown in **Figure 17.5D–F**. **Figure 17.5D** presents a cross-section of thin filaments in regions where there is no overlap with thick filaments (i.e., the I band), whereas **Figure 17.5E** presents a cross-section of thick filaments in a region where there is no overlap with thin filaments (H zone). Notice that in regions where the thick and thin filaments overlap (**Figure 17.5F**), each thick filament is surrounded by six thin filaments, and each thin filament is surrounded by three thick filaments.

In addition to contractile and regulatory proteins, a sarcomere consists of numerous structural proteins. The contractile proteins (actin and myosin) are organized by specialized cytoskeleton proteins that make up the sarcomeric cytoskeleton ([Gautel, 2011](#)). Structural proteins in the cytoskeleton bind components of

the sarcomere together and stabilize the sarcomere. Researchers have identified dozens of proteins that contribute to a highly organized and complex sarcomere cytoskeleton (Caiozzo and Rourke, 2006). **Figure 17.6A** diagrams some of the major structural proteins in the cytoskeleton of the sarcomere and indicates the relationship between the structural and contractile proteins (Boland et al., 2018). Proteins of the M line and the Z disk hold the thick and the thin filaments in place, respectively. Titin has multiple functions and will be discussed in some length later. Nebulin is actually a family of cytoskeletal proteins that binds the actin thin filament and has a role in regulating its assembly and function (Pappas et al., 2011), and the protein α -actinin links thin filaments to the Z disk. Still other structural proteins serve as mechanical links between the sarcomeres and the extracellular matrix of the muscle fiber. Collectively, these connection sites are called costameres (Caiozzo and Rourke, 2006).

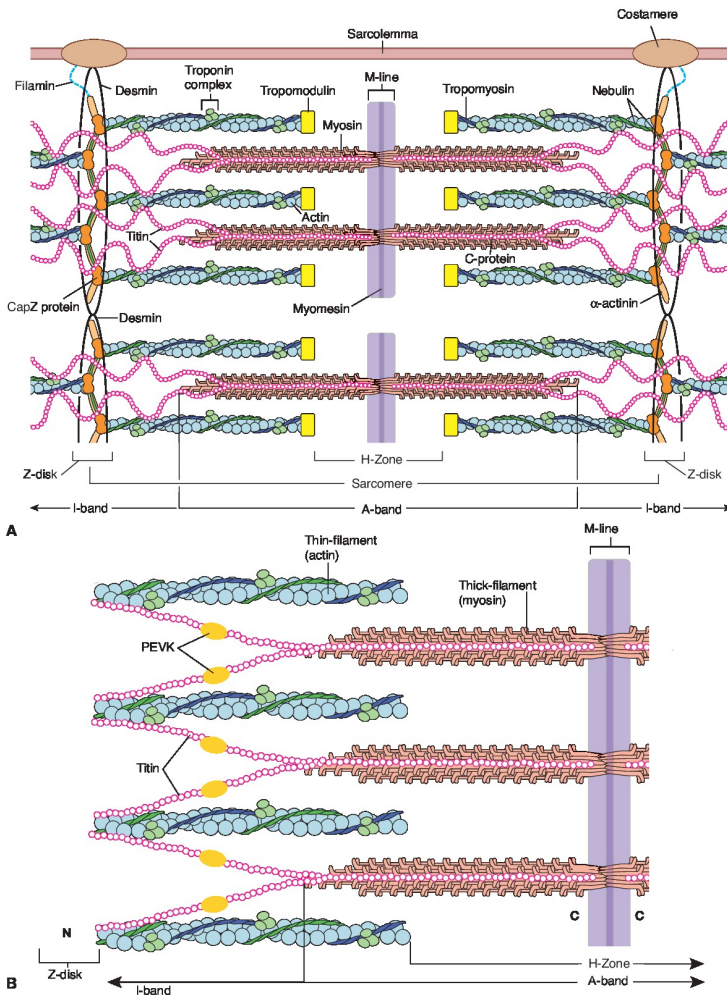


Figure 17.6

A. Representation of Auxiliary Structural Proteins in the Sarcomere. **Source:** Adapted from Boland, M., L. Kaur, F. M. Chian, T. Astruc: Muscle proteins. In Melton, L. D., F. Shahidi & P. Varelis (eds.): *Encyclopedia of Food Chemistry*. Cambridge, MA: Elsevier, 164–179 (2019). Copyright © 2019 Elsevier. With permission. **B.** Schematic illustration of a sarcomere with particular emphasis on the components of titin and their relationship to actin and myosin. **Source:** Modified with permission from Herzog, W.: Passive force

Molecular Structure of the Myofilaments

The contractile proteins of the myofilaments slide over one another during muscular contraction. Hence, the **sliding filament theory of muscle contraction** explains how muscles contract. Knowing the structure of the myofilaments is essential to understanding how muscles contract.

Sliding Filament Theory of Muscle Contraction The theory that explains muscle contraction as the result of myofilaments sliding over each other.

Thick Filaments

Thick filaments are composed of myosin molecules, primarily the contractile form, myosin heavy chain (MHC). It is primarily by differentiating MHC isoforms (different forms of a protein) that muscle fibers are delineated into muscle types (discussed later in this chapter) ([Pette, 2005](#)). Myosin light chain (MLC) molecules are also present and assist in regulating the rate of contractions ([Caiozzo and Rourke, 2006](#)). The term myosin as used in this text refers to the contractile form (MHC) unless otherwise specified. Each molecule of myosin has a rod-like tail and two globular heads (**Figure 17.7A**). A typical thick filament contains approximately 200–300 myosin molecules ([Caiozzo and Rourke, 2006](#)). These molecules are oriented so that the tails form the central rod-like structure of the filament (**Figure 17.7B**). The globular myosin heads extend outward and form *cross-bridges* when they interact with thin filaments. The myosin heads have two reactive sites: One allows it to bind with the actin filament and one binds to ATP. Only when the myosin heads bind strongly to the active sites on actin, forming a cross-bridge, can

contraction occur.

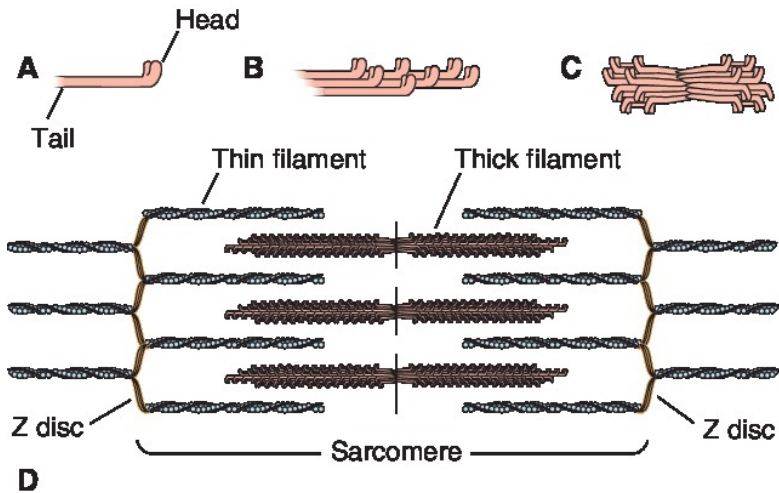


Figure 17.7 Molecular Organization of Thick Filaments.

A. Individual myosin molecules have a rod-like tail and two globular heads. **B.** Individual molecules are arranged so that the tails form a rod-like structure and the globular heads project outward to form cross-bridges. **C.** Myosin subunits are oriented in opposite directions along the filament forming a central bare zone in the middle of the filament (H zone). **D.** Thick filament (myosin) within a single sarcomere showing the myosin heads extending toward the thin filament.

The myosin subunits are oriented in opposite directions along the filament, forming a central section that lacks projecting heads (**Figure 17.7C**). The result is a bare zone in the middle of the filament, which is the H zone seen in the middle of the A band (**Figure 17.5C and D**).

Thin Filaments

Thin filaments are composed primarily of the contractile protein actin. As illustrated in **Figure 17.8A and B**, actin is composed of

small globular subunits (G actin) that form long strands called fibrous actin (F actin). A filament of actin is formed by two strands of F actin coiled about each other to form a double-helical structure; this structure, which resembles two strands of pearls wound around each other, may be referred to as a *coiled coil* (**Figure 17.8C**). The actin molecules contain active sites to which myosin heads bind during contraction.

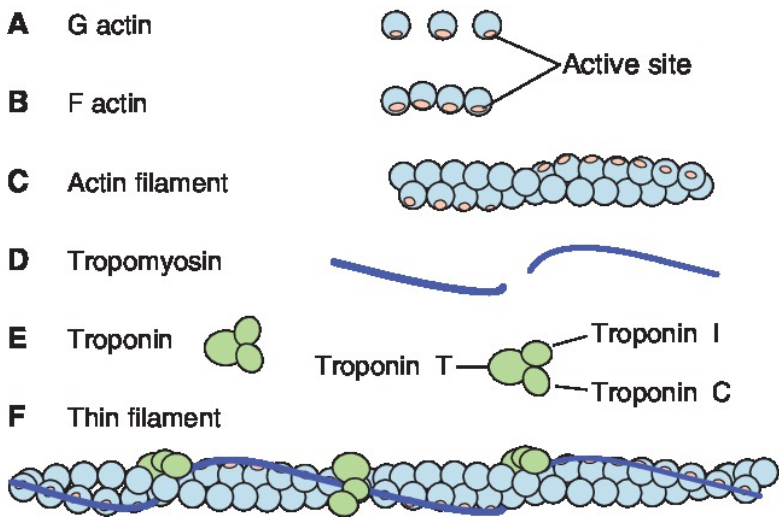


Figure 17.8 Molecular Organization of Thin Filaments.

A. Individual actin subunits (globular, G actin) shown with active site for binding to myosin heads. **B.** Fibrous actin (F actin). **C.** Actin filament with two strands of fibrous actin wound around itself to form a coiled coil. Active sites are exposed. **D.** Tropomyosin is a regulatory protein that covers the binding sites on actin. **E.** Troponin is a regulatory protein that when bound to Ca^{2+} removes tropomyosin from its blocking position on actin. **F.** The thin filament is composed of actin, tropomyosin, and troponin.

The thin filaments also contain the regulatory proteins called tropomyosin and troponin, which regulate the interaction of actin and myosin. *Tropomyosin* is a long, double-stranded, helical protein wrapped about the long axis of the actin backbone

(Figure 17.8D). Tropomyosin blocks the active site on actin, thereby inhibiting actin and myosin from binding under resting conditions.

Troponin is a small, globular protein complex composed of three subunits that control the position of the tropomyosin (**Figures 17.8E and 17.9**). The three units are troponin C (TnC), troponin I (TnI), and troponin T (TnT). TnC contains the calcium-binding sites, TnT binds troponin to tropomyosin, and TnI inhibits the binding of actin and myosin in the resting state (**Figure 17.9B**). When calcium binds to the TnC subunit, the troponin complex undergoes a configurational change. Because troponin is attached to tropomyosin, the change in the shape of troponin causes tropomyosin to be removed from its blocking position, thus exposing the active sites on actin ([Marieb and Hoehn, 2019](#)). Once the active sites are exposed, the myosin heads can bind to the actin, forming the cross-bridges (**Figure 17.9C**). Thus, calcium is key to controlling the interaction of the filaments and, in turn, muscle contraction.

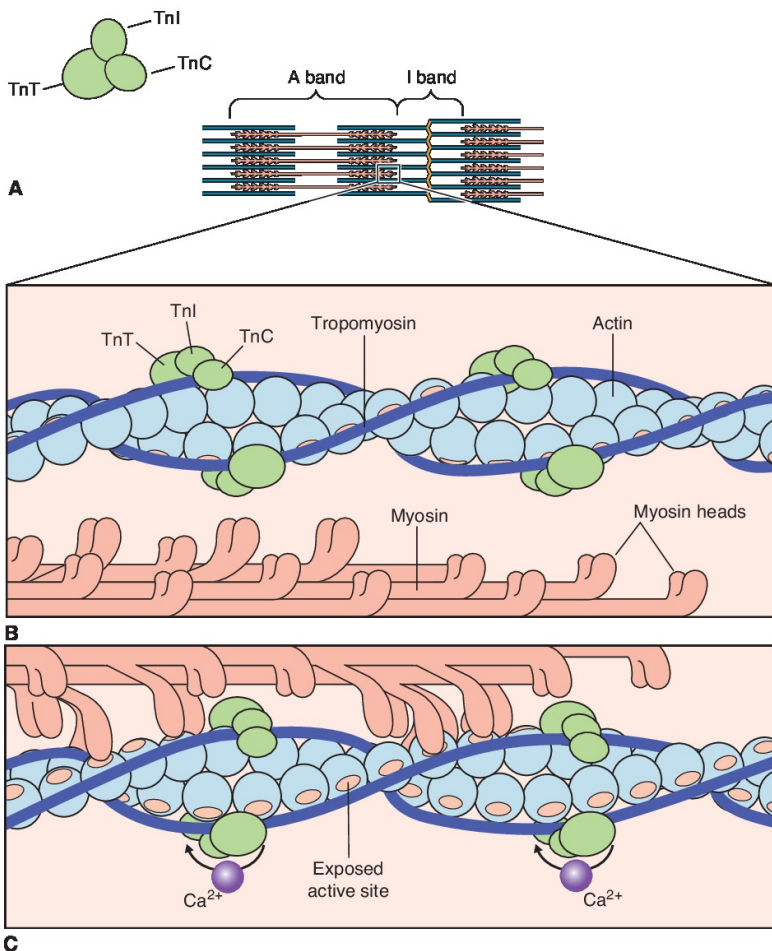


Figure 17.9 Regulatory Function of Troponin and Tropomyosin.

A. Troponin is a small globular protein with three subunits. **B.** Resting condition: Tropomyosin blocks the active sites on actin, preventing actin and myosin from binding. **C.** Contraction: When troponin binds with Ca^{2+} , it undergoes a configurational change and pulls tropomyosin from the blocking position on the actin filament, allowing myosin heads to form cross-bridges with actin.

Increasing Protein Synthesis: Interaction of Training and Nutrition

Many athletes, especially those engaged in resistance training, are interested in increasing protein synthesis. Increased protein synthesis increases the amount of contractile proteins and thus makes the muscles larger and stronger. Protein synthesis is enhanced in several circumstances: (1) following resistance exercise and (2) when amino acid availability is increased. Research by [Rasmussen et al. \(2000\)](#) suggests that when these conditions occur together, their effect on protein synthesis is additive. Participants in this study ingested a drink containing six essential amino acids and 35 g of sucrose following a bout of resistance training. The participants consumed the drink at either 1 or 3 hours after training, and the results were compared to a control group that consumed a flavored placebo drink. Those consuming the dietary drink had a significantly higher level of muscle protein synthesis compared to placebo.

Long-term interventional studies also show a benefit of protein supplementation around training for muscle mass accrual. [Willoughby et al. \(2007\)](#) showed that consuming a protein beverage containing 20 g of protein pre- and postexercise increases lean mass to a greater extent than a control drink (20 g dextrose) over 10 weeks. There is some evidence that the addition of carbohydrates postexercise may have an additional benefit on protein synthesis. However, more recent data suggest that when protein consumption is adequate, additional carbohydrate does not augment protein synthetic rates ([Staples et al., 2011](#)).



Source: Rasmussen et al. (2000); Staples et al. (2011); Willoughby et al. (2007).

Contraction of a Muscle Fiber

For a muscle to contract, three major events must happen:

1. An *action potential* must be generated in the motor neuron

that innervates the muscle fibers.

2. The motor neuron must release a neurotransmitter that travels across the neuromuscular junction and binds to receptors on the muscle cell membrane (sarcolemma).
3. An action potential in the muscle fiber must lead to the sliding of the myofilaments—thus shortening the muscle cell.

The first two of these necessary events are discussed in detail in [Chapter 20](#). The following section addresses the third event in the sequence: how a muscle fiber produces force when stimulated. The process whereby electrical events in the sarcolemma of the muscle fiber are linked to the movement of the myofilaments is called *excitation-contraction coupling*.

Before detailing the physiological changes within the muscle fiber (and their myofilaments) as a result of electrical stimulation, however, it is useful to consider the major tenets of the sliding filament theory as revealed through microscopic studies.

The Sliding Filament Theory of Muscle Contraction

The sliding filament theory of muscle contraction is commonly used to describe how muscle contraction generates force. A great deal of data have been amassed from x-ray, light microscopic, and electron microscopic studies to support the sliding filament theory of muscle contraction. This theory accounts for force production during concentric (shortening) contractions very well. However, there is some concern about the extent to which the sliding filament theory adequately explains force generation during eccentric (lengthening) contractions. One particular example is the increased force production during eccentric muscular contraction, which is described in more detail under the titin section in this chapter. It is plausible that other structural proteins, such as titin, contribute to the contractile properties of muscle, and their actions have yet to be fully elucidated. The basic principles of the sliding filament theory are as follows:

1. The force of contraction is generated by the process that slides the actin filament over the myosin filament. The

sliding of actin filaments leads to decreased space between adjacent actin molecules and subsequent shortening of the muscle fibers, thus, producing force.

2. The lengths of the thick and the thin filaments do not change during muscle contraction.
3. The length of the sarcomere decreases as the actin filaments slide over the myosin filaments and pull the Z disks toward the center of the sarcomere.

Changes in the Sarcomere during Contraction

Much of the evidence for the sliding filament theory comes from observed changes in the length of a sarcomere during muscular contraction. **Figure 17.10** diagrams the sarcomere during rest (**Figure 17.10A**) and during shortening with contraction (**Figure 17.10B**). Notice the following changes in the sarcomere:

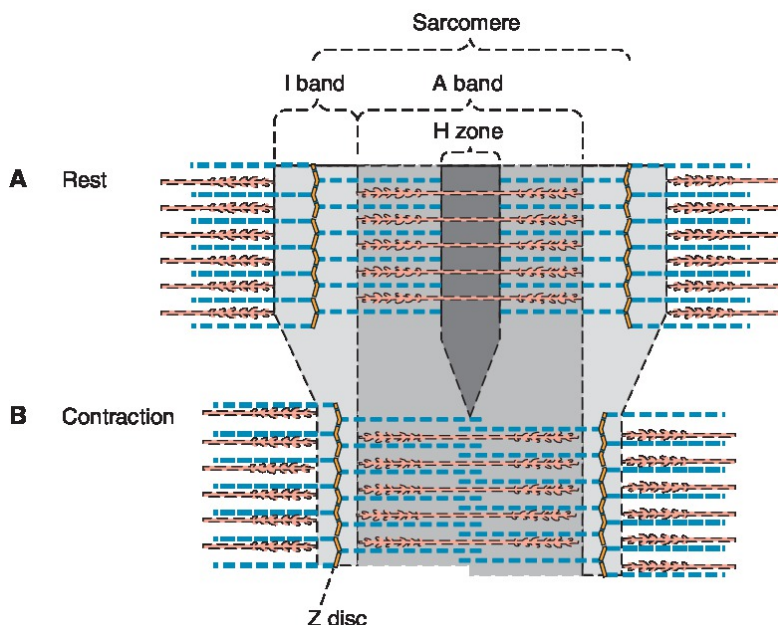


Figure 17.10 Changes in a Sarcomere during Contraction.

A. Sarcomere at rest. B. During contraction of the sarcomere, the lengths of actin and myosin filaments are

unchanged. Sarcomere shortens because actin slides over myosin, pulling Z disks toward the center of the sarcomere. The H zone disappears, the I band shortens, and the A band remains unchanged.

1. The A band does not change length, but the Z disks do move closer together. The length of the A band is preserved because the thick filament length does not change.
2. The I band shortens and may disappear. The I band shortens because the thin filaments are pulled over the thick filaments toward the center of the sarcomere. Thus, there is little or no area where the thin filaments do not overlap the thick filaments.
3. The H zone shortens and may disappear because the thin filaments are pulled over the thick filaments toward the center of the sarcomere. If the thin filament overlaps the thick filament for the entire length of the thick filament, there is no H zone.

As detailed in the next section, the sarcomere shortens as the result of the attachment of the myosin heads with the active site on actin and the subsequent release of stored energy that swivels the myosin cross-bridges. This step causes the actin to pull the Z disk toward the center of the sarcomere, which in turn causes the sarcomere, and thus the muscle fiber, to shorten. The ability of the sarcomere to deform elastically during contraction is due primarily to the structural cytoskeleton proteins whose modular architecture and flexibility permit change in length (Tskhovrebova and Trinick, 2012).

Excitation-Contraction Coupling

Excitation-contraction coupling is the sequence of events by which an action potential (AP; an electrical event) in the sarcolemma of the muscle cell initiates the sliding of the myofilaments, resulting in contraction (a mechanical event). Excitation-contraction coupling occurs in three phases:

Excitation-Contraction Coupling The sequence of events by which an action potential in the sarcolemma initiates the sliding of the myofilaments, resulting in contraction.

1. The spread of depolarization
2. The binding of calcium to troponin
3. The generation of force (cross-bridge cycling)

Figure 17.11 summarizes what occurs in each phase of excitation-contraction coupling. In the resting state, the regulatory protein tropomyosin is covering the active sites on actin. Excitation-contraction coupling begins with depolarization and the spread of an action potential (AP) along the sarcolemma (labeled 1 in **Figure 17.11**) and continues with the propagation of the action potential into the T tubules. The action potential in the T tubules causes the release of calcium from the adjacent lateral sacs of the sarcoplasmic reticulum (labeled 1a in **Figure 17.11**).

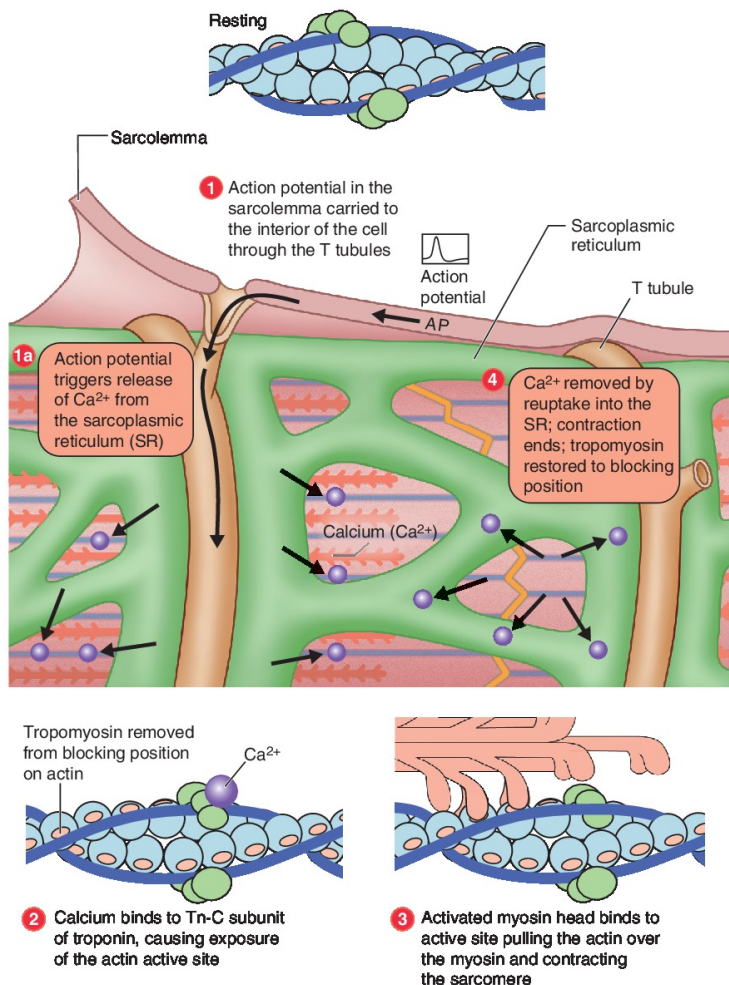


Figure 17.11 Phases of Excitation-Contraction Coupling.

The calcium that is released from the SR binds to the troponin molecules (TnC subunit) on the thin filament during the second phase. This causes troponin to undergo a configurational change, thereby removing tropomyosin from its blocking position on the actin filament (labeled 2 in **Figure 17.11**).

The third phase of excitation-contraction coupling is the cross-bridging cycle (labeled 3 in **Figure 17.11** and detailed fully in **Figure 17.12**). The **cross-bridging cycle** involves myosin heads binding to the active sites on actin and a series of cyclic events

necessary for the generation of tension within the myosin heads during muscle contraction. The tension within the contractile elements results from the binding of the myosin heads to actin and the subsequent release of stored energy in the myosin heads.

Cross-Bridging Cycle The cyclic events necessary for the generation of force or tension within the myosin heads during muscle contraction.

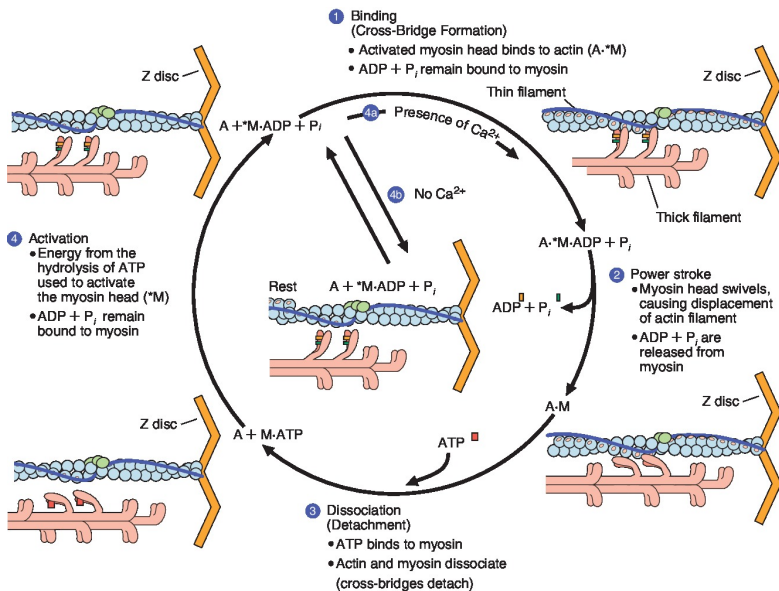


Figure 17.12 Force Generation of the Contractile Elements: The Cross-Bridging Cycle.

As detailed in **Figure 17.12**, the cross-bridging cycle occurs in four steps (Marieb and Hoehn, 2019; Vander et al., 2001):

1. Binding of myosin heads to actin (cross-bridge formation)
2. Power stroke
3. Dissociation of myosin and actin
4. Activation of myosin heads

The first step in the *cross-bridge cycle* is the binding of activated myosin heads (*M) with the active sites on actin, forming cross-bridges. In **Figure 17.12**, a centered dot (·) indicates binding and an asterisk (*) indicates activated myosin heads. Thus, A·*M means that the activated myosin heads are bound to actin (A), whereas A + M indicates that actin and myosin are unbound.

The second step in the cross-bridging cycle is the power stroke. During this step, activated myosin heads swivel from their high-energy, activated position to a low-energy configuration (M with no*). This movement of the myosin cross-bridges results in a slight displacement (sliding) of the thin filament over the thick filament toward the center of the sarcomere. As shown in **Figure 17.12**, during the second step, ADP and P_i are released from the myosin heads, resulting in myosin bound only to actin (A·M).

The third step involves the binding of ATP to the myosin heads and the subsequent dissociation (detachment) of the myosin cross-bridges from actin, thus producing A + M·ATP.

Note the role of ATP in steps 3 and 4. The *binding of ATP* molecules to the myosin head in step 3 allows the myosin heads to detach from actin. In the fourth step, the *breakdown of ATP* provides the energy to activate the myosin heads (*M). The activation of the myosin head is extremely important because it provides the cross-bridges with the stored energy to move the actin during the power stroke. The breakdown of ATP at this step depends on the presence of myosin ATPase (also known as myofibrillar ATPase), as depicted in the following reaction:



Notice that the products of ATP hydrolysis, ADP + P_i , remain bound to the myosin heads and that the myosin is now in its high-energy or activated state.

The cross-bridging cycle continues as long as ATP is available and calcium is bound to troponin (TnC), causing the active sites on actin to be exposed. On the other hand, activated myosin will remain in the resting state awaiting the next stimulus if calcium is not available in sufficient concentration to remove tropomyosin from its blocking position on actin (labeled 4b in **Figure 17.12**).

Because each cycle of the myosin cross-bridges barely displaces the actin, the myosin heads must bind to the actin and be displaced many times for a single contraction to occur. Thus, myosin makes and breaks its bond with actin hundreds or even thousands of times during a single muscle twitch. For this make-and-break cycle to occur, the myosin heads must detach from actin and then be reactivated. This detaching and reactivating process requires the cycle to be repeated and requires the presence of ATP (step 3).

An analogy helps explain the role of ATP in providing energy to activate the myosin head. Visualize a spring-loaded mousetrap. It takes energy to set the trap, just as it takes the splitting of ATP to set or activate the myosin head. Once set, however, the trap will release energy when it is sprung. In a similar manner, the myosin head possesses stored energy that is released when the myosin heads bind to actin and swivel.

It may be useful to review the cycle of events in **Figure 17.12** several times, paying attention to a different aspect (the symbols, the wording, the diagrams, the role of ATP) in each review. Also, keep in mind that ATP plays several important roles in muscle contraction:

1. ATP breakdown provides the energy to activate and reactivate the myosin cross-bridge prior to binding with actin.
2. ATP binding to the myosin head is necessary to break the cross-bridge linkage between the myosin heads and the actin so that the cycle can repeat.
3. ATP is used for the return of calcium into the sarcoplasmic reticulum and restoration of the resting membrane potential once contraction has ended.

The final phase of muscular contraction is a return to muscular relaxation. Relaxation occurs when the nerve impulse ceases and calcium is pumped back into the sarcoplasmic reticulum by active transport (labeled 4 in **Figure 17.12**). In the absence of calcium, tropomyosin returns to its blocking position on actin, and myosin heads are not able to bind to actin. While emphasis is usually placed on muscle contraction, relaxation of a

muscle following contraction is just as important.

Structural Muscle Proteins

Myosin and actin are the most commonly discussed proteins when it comes to muscle structure and function. However, their actions are not possible without the function of other structural proteins within the sarcomere. Although a comprehensive review of these proteins is outside of the scope of this textbook, general information regarding the role of these proteins is helpful to fully understand muscle physiology (**Figure 17.6A**).

Filamins: structural proteins that bind to actin proteins via different actin binding sites. Filamins exist in various isoforms and mainly serve as scaffolds that hold several proteins in proper spatial arrangement ([Nakamura et al., 2011](#)).

Desmin: a small protein that is located on the periphery of z-disks in skeletal muscle and is involved in the linking of myofibrils to the sarcolemma. Like filamins, desmin plays an important role as a scaffold protein ([Paulin and Li, 2004](#)).

Nebulin: a large protein involved in regulating the length and mechanical properties of thin filaments as well as interacting with other structural proteins within the Z-disk ([Yuen and Ottenheijm, 2020](#)).

α -Actinin: the main component of the z-disks and plays an integral role in the binding of actin to the z-disk ([Sjöblom et al., 2008](#)).

Tropomodulin: present at the end of actin molecules near the M-line and acts as a “cap,” which regulates the length of actin molecules by inhibiting further growth of actin ([Kostyukova, 2008](#)).

Myomesin: the main protein that composes the M-line. Structurally, it is partially responsible for the binding of myosin and titin. Additionally, there are some data suggesting that myomesin may play a role in the assembly and integrity of the sarcomere ([Prill et al., 2019](#)).

Myosin and actin receive much attention due to their important role in muscle contraction. However, without the presence and actions of these additional structural proteins, the sarcomere would not properly function. The following section is

dedicated to titin, a very large structural protein which plays a key role in muscle contraction.

Titin

The cross-bridge and sliding filament theories are widely accepted as the explanation for muscle contraction mechanisms. However, not all experimental observations and basic muscle properties can be fully explained by these actions alone. The primary examples involve the active lengthening of muscle known as eccentric contraction. For example, the lowering action when performing a biceps curl is an eccentric contraction. During eccentric contraction, force is increased compared with isometric or concentric contractions, the energy requirement is decreased, and forces following the contraction are increased. This enhanced force production is known as residual force enhancement (RFE). *Residual force enhancement* is defined as the increase in active, steady-state, isometric force in a muscle following active stretching (eccentric action) compared with the corresponding purely isometric force. RFE is associated with a decrease in metabolic cost per unit of force produced compared with the metabolic cost of a purely isometric contraction. Experimental evidence indicates that such force enhancement is not associated with a change in muscle stiffness nor a greater proportion of attached cross-bridges (Freundt and Linke, 2019; Fukutani and Herzog, 2019a, 2019b; Herzog, 2014, 2019; Montesano, et al., 2020; Nishikawa et al., 2018). In addition, there is an inherent limit on the amount of useful mechanical work that ATP-driven action can perform in striated muscle. If a great enough load is applied, the power output will drop to zero (Eckels et al., 2018). The explanations for these previously unexplained observations are pointing to the unique protein, titin.

There is now strong evidence that the muscle sarcomere is composed not of two (actin-based thin and myosin-based thick), but of three main filaments that work together to store elastic energy and perform mechanical work. The third is the titin-based elastic filament (Eckels et al., 2018; Freundt and Linke, 2019).

Titin is the largest known protein in the human body. It is a single chain of approximately 34,000 amino acids that connects the Z line to the M line in the sarcomere such that a single titin

molecule spans half the length of a sarcomere (**Figure 17.6B**) and another spans the other half of the sarcomere. Titin is arranged as a sequence of individually folded domains (~244) connected by unstructured regions sometimes labeled proximal, differentially spliced, and distal domains. The N terminal segment is composed of immunoglobulin-like (Ig) domains and anchors titin to the Z-band. The I band segment is the elastic component and contains tandem Ig-domains and intrinsically disordered structures (PEVK). The A band segment is composed of Ig and fibronectin domains and binds titin tightly to the thick filament. The C-terminal segment contains 10 Ig domains and anchors titin to the proteins of the M line. Six titin filaments are associated with each thick filament, and each titin filament attaches to a single thin filament (Eckels, et al., 2018; Freundt and Linke, 2019; Herzog, 2019; Nishikawa et al., 2018).

Titin performs a variety of functions in skeletal muscle including the following:

1. Acting as a scaffold. The function of titin as a scaffold in maintaining the structural integrity of the striated muscle sarcomere is generally well accepted. Titin regulates the lattice spacing between the thick and thin filaments. Titin also prevents longitudinal misalignment of the thick filaments and maintain A band centering, thereby contributing to active muscle force (Granzier and Labeit, 2006; Nishikawa et al., 2018; Nishikawa, 2020).
2. Acting as a molecular spring. The compliant Ig domains near the Z-line straighten at low force, whereas the stiffer PEVK region extends under much higher force. Thus, titin is both a compliant and a stiff spring in series. It is bidirectional. This spring both prevents overstretching of the sarcomere and provides a recoil of the sarcomere after it has been stretched (Freundt and Linke, 2019; Granzier and Labeit, 2006; Herzog, 2014; Nishikawa, 2020).
3. Causing passive force enhancement. Resting skeletal muscles at lengths less than normal do not exert any force. If the length is extended somewhat, passive force is evident. This passive force is developed in noncontracting muscles by the elastic elements of the muscle and can be increased or

enhanced. There is strong evidence that such *passive force enhancement* is caused by titin. Details of the molecular mechanism underlying the enhancement remain unknown; however, it likely involves the shortening of titin's free spring length and binding to actin in the proximal (near the Z band) segment leaving the distal titin segments unbound for accomplishing the increases in I-band length with increasing sarcomere length. The binding of titin to actin (possibly as a result of the configurational changes in troponin and tropomyosin) results in increased forces in the actively stretched muscle. When the muscle is deactivated, titin remains attached to actin providing the long-lasting increase in passive muscle force. This passive force is typically smaller than the total RFE (Herzog, 2019; Nishikawa, 2018).

4. Possibly contributing to residual force enhancement. As noted previously, RFE is defined as the increase in active, steady-state, isometric force in a muscle following active stretching (eccentric action) compared with the corresponding purely isometric force. Although unequivocal evidence for titin's contribution to RFE is still lacking, there is accumulating evidence for its involvement. As with passive force enhancement, it is likely linked to a shortening of titin's free spring length by its binding to actin. However, it extends to additional actin-binding sites. If the proximal Ig domain of titin is bound to actin, that proximal segment cannot be elongated during active stretch. To compensate for this, the distal segment is elongated more. This results in an increased force at a given sarcomere length that is RFE (Fukutani et al., 2021).
5. Serving as a modulator of contraction. When a muscle stretches, the mechanical tension increases and titin domains unfold. The Ig domains near the Z-line straighten at low force, whereas the stiffer PEVK region extends under much higher force. When muscles contract, myosin heads engage with actin and decrease the force on titin, allowing the Ig domains to refold and shorten. This process delivers stored elastic energy and assists in the muscle contraction. Titin cannot fold without formation of the cross-bridge but also completion of the power stroke relies on titin folding. Thus,

titin works as an active component in the sarcomere and is a main determinant of muscle force as both a passive spring and a generator of active contractile work via Ig domain folding contraction. Titin Ig domains are capable of folding against a pulling force (Eckels et al., 2018; Freundt and Linke, 2019; Nishikawa, 2018).

At this point in time, it seems very likely that titin plays a major role in explaining the experimental observations and basic muscle properties that cannot be explained solely by the sliding filament theory and the basic crossbridging cycle. However, work remains to fully explain how titin functions at the molecular level.

All-or-None Principle

According to the **all-or-none principle**, when a motor neuron is stimulated, all the muscle fibers in that motor unit contract to their fullest extent or do not contract at all. A **motor unit** is defined as a motor neuron ($\alpha 1$ or $\alpha 2$) and the muscle fibers it innervates. The minimal stimulus necessary to initiate that contraction is referred to as the *threshold stimulus*. When the threshold is reached, a muscle fiber contracts to its fullest extent. This principle involves the electrical properties of the stimulated cell membrane and thus applies to a motor unit or a single muscle fiber only, not to the entire muscle. Consider the analogy of a light switch. When enough pressure (threshold) is applied to the switch to flip it on, the light will turn on to its fullest extent. When the switch controls a group of lights (like a motor neuron innervating multiple muscle fibers), all the lights will turn on to their fullest extent. You cannot make the lights brighter by pushing the switch harder, because it is an all-or-none response. The same is true for an individual muscle fiber or a motor unit: either a threshold stimulus is reached and contraction occurs or a threshold stimulus is not reached and contraction does not occur.

All-or-None Principle When a motor neuron is stimulated, all of the muscle fibers in that motor unit contract to their fullest

extent or do not contract at all.

Motor Unit A motor neuron and the muscle fibers it innervates.

Muscle Fiber Types

Muscle fibers are typically described by two characteristics: their contractile (twitch) properties and their metabolic properties (**Figure 17.13**).

| Muscle Fibers | | | |
|---|------------|-----------------------|--------------------|
| Twitch properties | Slow | Fast | |
| Metabolic properties | Oxidative | Oxidative/ glycolytic | Glycolytic |
| Name based on twitch and metabolic properties | SO | FOG | FG |
| Other nomenclature | ST, Type I | FTa, FTA, Type IIA | FTx, FTX, Type IIX |
| Motor Neurons | | | |
| Neuron type | α_2 | α_1 | α_1 |
| Neuron size | Small | Large | Large |
| Conduction velocity | Slow | Fast | Fast |
| Recruitment threshold | Low | High | High |

Figure 17.13 Properties of Motor Units.

Contractile (Twitch) Properties

Based on differences in *contractile (twitch) properties*, human muscle fibers can be categorized as *slow-twitch (ST)* or *fast-twitch (FT) fibers*. Slow-twitch fibers are sometimes called Type I fibers, and fast-twitch fibers are called Type II fibers. The difference between ST and FT fibers appears to be absolute—like the difference between black and white. Some FT fibers contract and relax slightly faster than other fast-twitch fibers, but both of them are clearly much faster than ST fibers. Understanding the difference between twitch speeds begins with understanding the integration of muscles and nerves.

Skeletal muscle fibers are innervated by alpha (α) motor neurons, which exist in two categories, α_1 and α_2 . The α_1 motor neurons innervate FT fibers, and the α_2 motor neurons innervate ST fibers. **Figure 17.14** depicts the results of an experiment that manipulated the innervation of muscle fibers. The α_1 motor neuron was severed from the FT fibers and connected to the ST fibers, and the α_2 motor neuron was cut from the ST fibers and connected to the FT fibers. Importantly, the ST fibers became FT fibers when the α_1 motor neuron replaced the α_2 motor neuron, and vice versa. Therefore, it is logical to conclude that the contractile property of muscle depends on the type of motor neuron that innervates the muscle fibers (Buller et al., 1960; Noth, 1992).

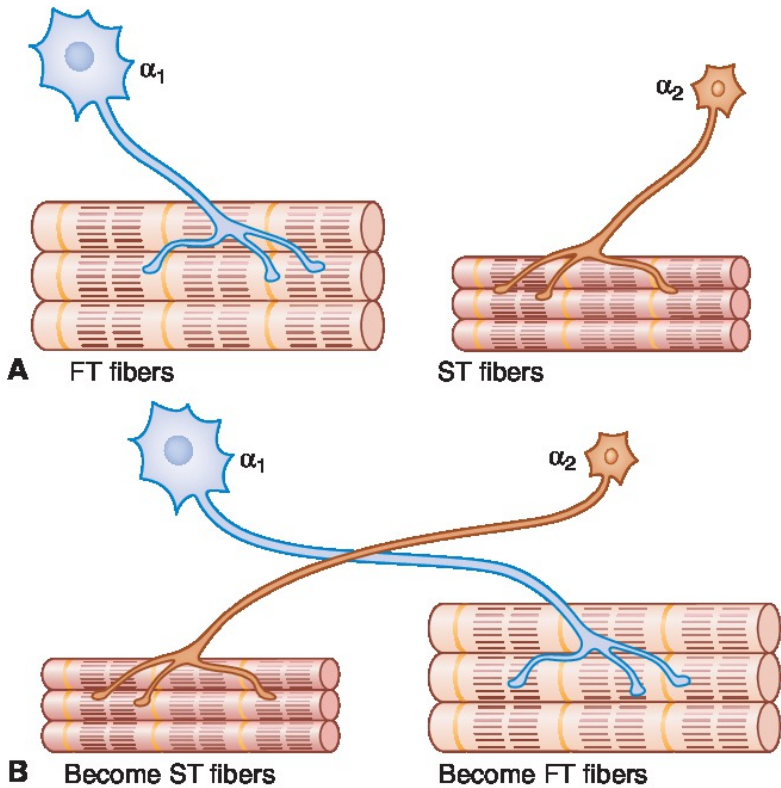


Figure 17.14 Results of Cross-Innervation.

A. Under normal conditions, α_1 motor neurons innervate FT fibers and α_2 motor neurons innervate ST fibers. **B.** If the neurons supplying the muscles are switched (cross-innervated), the muscle fibers acquire the properties of the new motor neuron.

Other elements in the muscle, especially the contractile enzyme myosin ATPase, also contribute to the variation in twitch speed. Indeed, when biopsied muscle fibers are typed, the amount of stain for myosin ATPase is often used to distinguish twitch speed, since the motor neurons are not typically biopsied.

Note that α_2 motor neurons are the smaller of the two nerves and innervate the ST muscle fibers; the α_1 motor neurons are the larger nerves and innervate the FT muscle fibers. The size difference is important because small motor neurons have low

excitation thresholds and slow conduction velocities and are thus recruited at low workloads. In contrast, larger motor neurons have a higher excitation threshold and are not recruited until high force output is needed. Thus, motor neurons are recruited according to the *size principle*. Smaller motor units ($\alpha 2$ motor neurons innervating ST fibers) are recruited during activities that require low force output, such as maintaining posture. As the need for force production increases, such as lifting heavy weights, larger motor units ($\alpha 1$ motor neurons innervating FT fibers) are recruited.

Metabolic Properties

On the basis of differences in *metabolic properties*, human muscle fibers can be described as *glycolytic*, *oxidative*, or a combination of both, *oxidative/glycolytic*. All muscle fibers can produce energy both anaerobically (without oxygen, labeled as glycolytic) and aerobically (with oxygen, labeled as oxidative). These processes and terms are fully explained in the unit on metabolism.

Despite the ability of all muscle fibers to produce energy by both glycolytic and oxidative processes, one or the other type of energy metabolism may predominate or the production may be balanced. Thus, the metabolic properties of muscle fibers are not absolute characteristics as much as a continuum (oxidative to glycolytic). This continuum involves shades of gray, unlike the black-or-white typing of slow- or fast-twitch fibers. The metabolic properties of a muscle specimen are determined by staining for key enzymes (often phosphofructokinase [PFK] for glycolytic processes and succinate dehydrogenase [SDH] for oxidative processes) ([Saltin et al., 1977](#)).

Integrated Nomenclature

Slow-twitch fibers rely primarily on oxidative metabolism to produce energy and are therefore referred to as **slow oxidative (SO) fibers** (Type I fibers). Fast-twitch fibers that can work under both oxidative and glycolytic conditions are called **fast oxidative glycolytic (FOG) fibers**; these fibers are also referred to as Type IIA or Type Ila fibers. Other fast-twitch fibers that perform

predominantly under glycolytic conditions are called **fast glycolytic (FG) fibers** and in humans are also known as Type IIX or Type IIX fibers.

Slow Oxidative (SO, Type I) Fibers Slow-twitch muscle fibers that rely primarily on oxidative metabolism to produce energy

Fast Oxidative Glycolytic (FOG, Type IIA or IIA) Fibers Fast-twitch muscle fibers that can work under oxidative and glycolytic conditions.

Fast Glycolytic (FG, Type IIX, or IIX) Fibers Fast-twitch muscle fibers that perform primarily under glycolytic conditions.

Skeletal muscle has been classified in numerous ways based on the composition of the myosin molecules, particularly the isoforms of the myosin heavy chain (MHC) and to a lesser extent on the myosin light chain (MLC) isoform (Caiozzo and Rourke, 2006). This chapter and the text as a whole primarily rely on the integrated nomenclature of SO, FOG, and FG for clarity, but occasionally, the designations Type I, IIA (or IIA), and IIX (or IIX) are used.

A motor unit consists of a motor neuron (α_1 or α_2) and the muscle fibers it innervates. As previously described, the twitch speed of a muscle fiber depends largely on the motor neuron that innervates it. Thus, all muscle fibers within a motor unit will be either FT or ST. In addition, because all muscle fibers in a motor unit are recruited to contract together, they have the same metabolic capabilities. Therefore, a motor unit is composed exclusively of SO, FOG, or FG muscle fibers. That means that references to muscle fiber types also refer to motor unit types. **Figure 17.13** summarizes the properties of motor units and muscle fibers (Caiozzo and Rourke, 2006; Harris and Dudley,

2000).

Muscle fiber size differs across species, gender, muscle group, and training status (Chalmers and Row, 2011; Staron et al., 2000). **Figure 17.15** presents cross-sectional area measurements of the fiber types in the vastus lateralis muscle (part of the quadriceps group) of untrained men and women (Staron et al., 2000). These data are based on multiple studies performed on over 150 college-aged males and females over a 10-year period. In general, men have larger cross-sectional area than women. Interestingly, Type II fibers tend to be larger than Type I fibers in men, whereas Type I fibers tend to be larger than Type II fibers in women.

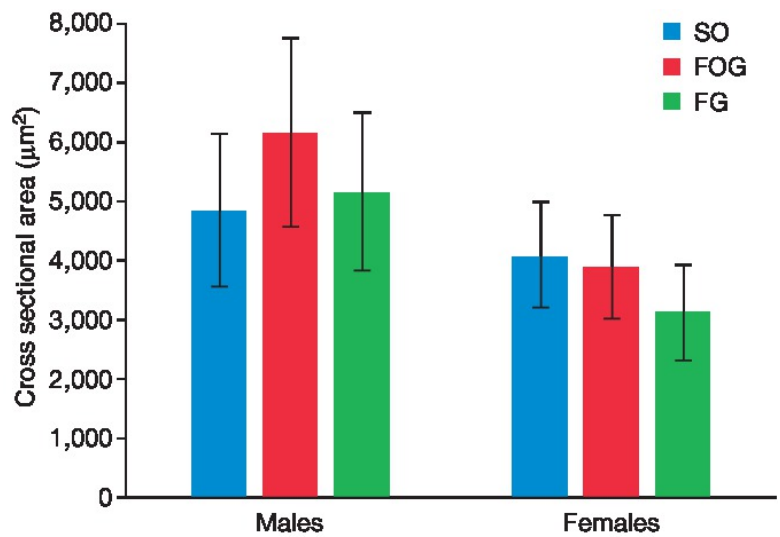


Figure 17.15 Fiber-Type Cross-Sectional Area of Vastus Lateralis.

Source: Data from Staron et al. (2000).

Table 17.2 further compares the different muscle fiber types based on important structural, metabolic, and functional characteristics (Harris and Dudley, 2000; Zierath and Hawley, 2004). Many differences among fiber types are related directly to their predominant metabolic pathway for energy production. The SO fibers, which rely mainly on oxidative pathways for energy

production, have a high number of mitochondria, high capillary density, high myoglobin content, and high oxidative enzyme activity. The FG fibers, which rely primarily on glycolytic pathways for energy production, have few mitochondria, low capillary density, low myoglobin content, and high glycolytic enzyme activity. The FOG fibers share characteristics of both SO and FG but also have some unique characteristics. Specifically, the FOG fibers have intermediate mitochondrial density, capillary density, myoglobin content, and oxidative enzyme activity and high PC stores, glycogen stores, and glycolytic enzyme activity.

TABLE 17.2 Characteristics of Muscle Fibers

| | Type I | Type II | |
|----------------------------|--------|--------------|------|
| Contractile (Twitch) | ST | FTa | FTx |
| Metabolic | SO | FOG | FG |
| Structural Aspects | | | |
| Mitochondrial density | High | Intermediate | Low |
| Capillary density | High | Intermediate | Low |
| Myoglobin content | High | Intermediate | Low |
| Metabolic Aspects | | | |
| Phosphocreatine stores | Low | High | High |
| Glycogen stores | Low | Intermediate | High |
| Triglyceride stores | High | Intermediate | Low |
| Myosin-ATPase activity | Low | Intermediate | High |
| Glycolytic enzyme activity | Low | Intermediate | High |
| Oxidative enzyme activity | High | Intermediate | Low |
| Functional Aspects | | | |
| Twitch (contraction) time | Slow | Fast | Fast |
| Relaxation time | Slow | Fast | Fast |
| Force production | Low | Intermediate | High |
| Fatigability | Low | Intermediate | High |

The metabolic differences among muscle fibers both require and reflect differences in energy substrate availability. All muscles store and utilize glycogen, but since glycogen is the only

substrate (along with its constituent parts—glucose) that can be used to fuel glycolysis, it makes sense that the FOG and FG fibers would have higher glycogen stores than SO fibers have. Conversely, since triglycerides can only be broken down and used oxidatively, it would be anticipated that SO fibers would have more triglyceride storage than either FG or FOG fibers. Furthermore, FOG fibers would have an intermediate amount of triglycerides—more than FG but less than SO fibers.

Because SO fibers are so well supplied by the cardiovascular system and have ample fuel supplies (energy substrate), particularly from triglycerides, they are very resistant to fatigue. Because the FOG fibers have a substantial oxidative capability and the FG fibers do not, the FG fibers are the quickest to fatigue. The FOG fibers are somewhat less resistant to fatigue than the SO fibers and somewhat more resistant to fatigue than the FG fibers. **Figure 17.16** shows functional differences in the three types of muscle fibers including different force production curves (**A**), different fatigue curves (**B**), and different contractile force and power curves at various speeds (**C**).

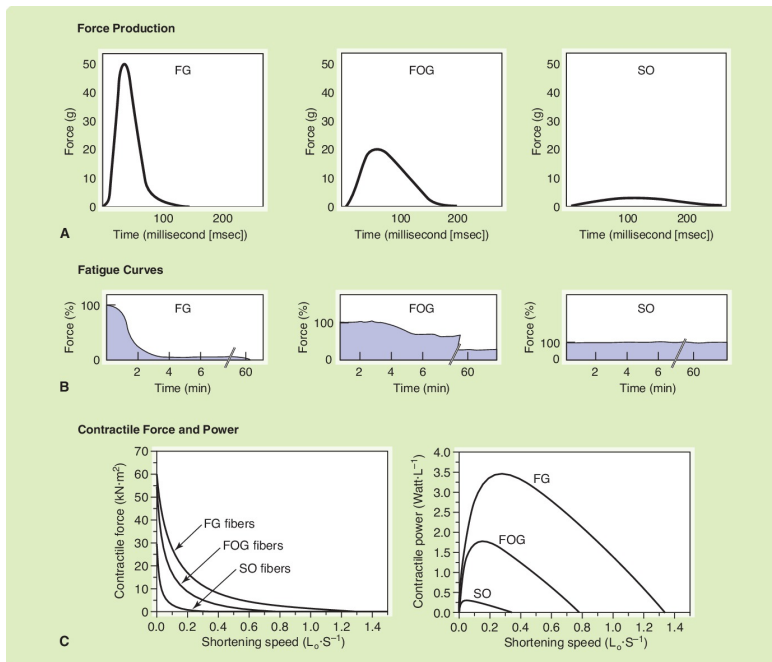


Figure 17.16 Fiber Types Have Different Properties.

A. Force production. B. Fatigue curves. C. Contractile force and power at different contraction speeds. **Sources:** Adapted from Edington and Edgerton (1986); Caiozzo and Rourke (2006).

Table 17.2 summarizes key information about fiber types. Take a few minutes now to study this table and check your understanding of how this information is interrelated. Remember that although the fiber types have been labeled at the top of the columns separately for their contractile and metabolic properties, in practice, the designations ST, SO, and Type I; Type IIa and FOG; and Type IIx and FG are used interchangeably.

Also take a few minutes and complete the [Check Your Comprehension—Case Study 1](#) to evaluate your understanding of muscle fiber types.

CHECK YOUR COMPREHENSION 1-CASE STUDY 1

Jackamo and Andreas have been best friends throughout high school. Both are on the track and field team, but Jackamo has always excelled at long-distance events, whereas Andreas routinely places in the top three finishers for the sprint races. Who do you think has a higher percentage of SO fibers? Discuss metabolic differences among fiber types and indicate how energy substrate availability differs in those with a high percentage of SO fibers versus FG or FOG fibers.

Check your answer in Appendix C.

Assessment of Muscle Fiber Type

Muscle fiber type is typically determined by a needle biopsy, an invasive procedure that involves collecting a small sample of skeletal muscle (**Figure 17.17**). Muscle biopsy samples are most commonly obtained from the gastrocnemius, vastus lateralis, or deltoid muscles. First, the skin is thoroughly cleaned, and a

topical anesthetic is applied to numb the area. A small incision is made through the skin, subcutaneous tissue, and fascia. The biopsy needle is then inserted into the belly of the muscle to extract a small amount of skeletal muscle tissue (~20–40 mg). This sample is then frozen in liquid nitrogen and sliced into very thin cross-sections.



Figure 17.17 Muscle Biopsy.

A biopsy needle is inserted into a small incision to obtain a sample of skeletal tissue.

The cross-sections are chemically stained so that the muscle fibers can be differentiated into categories. Muscle samples may be stained for the enzyme myosin ATPase and for glycolytic and oxidative enzymes. When stained muscle fibers are viewed in cross-section under a microscope, different muscle fiber types appear in different colors. **Figure 17.18** shows a skeletal muscle sample that has been histochemically stained, revealing ST (dark) and FT (light) fibers. Notice that the fiber types are intermingled, creating a mosaic pattern. Counting fibers of each type allows calculating the percentage of each fiber type (see and complete the [Check Your Comprehension 2 box](#)). In addition to these percentages, researchers often measure the diameter of the muscle fibers.

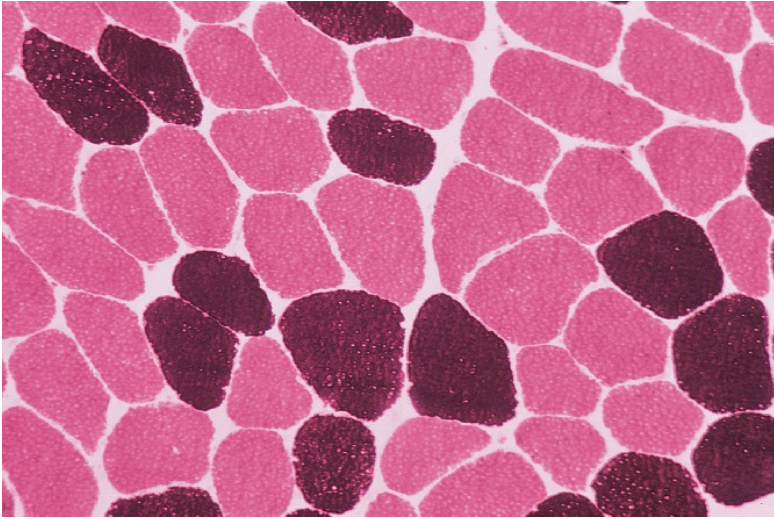


Figure 17.18 Mosaic Pattern of FT and ST Muscle Fibers Seen in a Microphotograph of Skeletal Muscle.

The darker-stained fibers are the ST fibers; the lighter-stained fibers are the FT fibers.

Muscle fibers also can be typed noninvasively by nuclear magnetic resonance spectroscopy (NMR), but this laboratory technique has not yet gained widespread acceptance ([Baguet et al., 2011](#)). Attempts to use the vertical jump as a field measure of fiber type have met with varying success (see the Focus on Application) ([Costill, 1978](#); [Fry et al., 2003](#)).

Knowledge about fiber types is important for at least three reasons:

1. Fiber-type differences help explain individual differences in performance and response to training.
2. Fiber-type differences help explain what training can and cannot do.
3. The relationship between fiber types and training and performance in elite athletes helps in the design of training programs for others who wish to be successful in specific events even if they do not know their exact fiber-type percentages or distribution.

FOCUS ON APPLICATION

The Relationship between Muscle Fiber Characteristics and Physical Performance

Athletes in strength or power sports have higher percentages of fast-twitch (FT) fibers than athletes in endurance events. Additionally, the cross-sectional area of FT fibers is larger in weightlifters and strength athletes than in sedentary individuals or endurance-trained athletes. Based on the structure-function relationship that underlies physiology, a strong relationship would be expected between fiber-type percentage and performance variables. Indeed, when a group of researchers investigated the relationship (correlations) between fast oxidative glycolytic (FOG) fibers (both percentage of fibers and area) and performance, they found several statistically significant correlations (see table below).

Correlations between Muscle Fiber Characteristics and Performance Variables

| | % FOG Fibers | % Area of FOG Fibers |
|---------------|-----------------|-------------------------|
| 1 RM snatch | 0.94 | 0.83 |
| Vertical jump | 0.83 | 0.75 |

The high percentage and large cross-sectional area of fast oxidative fibers in weightlifters are strongly related to performance in both weightlifting and in vertical jump performance. These results support the theoretical relationship between muscle fiber characteristics and actual physical performance. The results also suggest that a simple test for lower-body power, the vertical jump, may be a useful

field test to provide a noninvasive indicator of muscle fiber characteristics.

Source: Fry et al. (2003).

Complete the [Check Your Comprehension 2](#) to test your understanding of how muscle fiber typing is performed.

CHECK YOUR COMPREHENSION 2

1. Fiber typing involves determining the percentage of a sample's fast-twitch or slow-twitch fibers. Count how many total fibers are visible in **Figure 17.18**. Now count how many of those fibers are darkly stained (indicating, in this example, that they are ST fibers). Based on these two numbers, what is the percentage of ST fibers in this muscle sample? What is the percentage of FT fibers in this sample? Using the information provided in [Check Your Comprehension 1](#), whose biopsy do you think is represented in **Figure 17.18**?

Check your answer in Appendix C.

Distribution of Fiber Types

All muscles in humans are composed of a combination of slow-twitch and fast-twitch muscle fibers arranged in a mosaic pattern. This arrangement is thought to reflect the variety of tasks that human muscles must perform. The relative distribution, or percentage, of these fibers, however, may vary greatly from one muscle to another. For example, the soleus muscle may have as much as 85% ST fibers, and the triceps and ocular muscles may have as few as 30% ST fibers. The distribution may also vary considerably among individuals for the same muscle group ([Saltin et al., 1977](#)). The following are general characteristics of the distribution of fiber types:

1. Although distribution of fiber type varies within and between individuals, most individuals possess between 45 and 55% ST fibers.
2. The distribution of fiber types is not different for males and females, although males tend to show greater variation than females.
3. After early childhood, the fiber distribution does not change significantly as a function of age.
4. Fiber-type distribution is primarily genetically determined.
5. Muscles involved in sustained postural activity have the highest number of slow-twitch muscle fibers.

Fiber Type in Athletes

Few topics in exercise physiology evoke more interest and debate than issues of fiber type in athletes. **Figure 17.19** shows the distribution of fiber types in male and female athletes. Athletes in endurance activities typically have a higher percentage of slow-twitch fibers, while athletes in power activities have a higher percentage of fast-twitch muscle fibers. The distribution of fiber type ranges widely in each group, however, indicating that athletic success is not determined solely by fiber type.

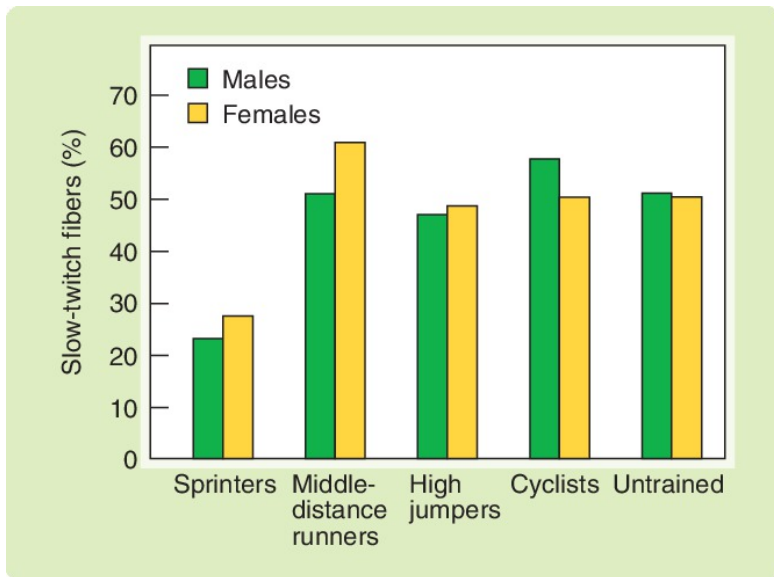


Figure 17.19 Fiber-Type Distribution among Athletes.

Source: Data from [Fox et al. \(1993\)](#).

Not only do endurance athletes differ in general fiber type from power or resistance athletes but often these differences relate to specific muscles within these athletic groups. The results of one study of fiber-type distribution are reported in **Figure 17.20** ([Tesch and Karlsson, 1985](#)). According to this study, the vastus lateralis muscles of the legs possess a greater percentage of ST fibers in endurance athletes primarily using the legs for their activity (such as runners). In contrast, athletes whose sport requires endurance of the upper body possess a greater percentage of ST fibers in the deltoid muscle.

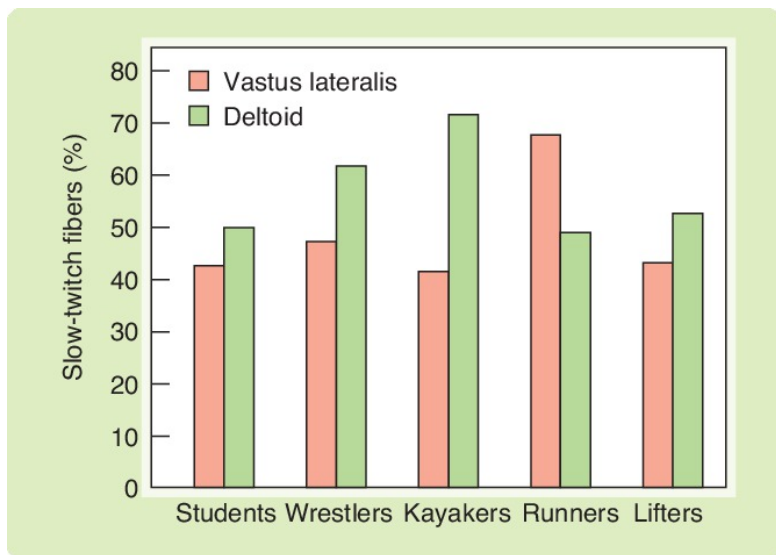


Figure 17.20 Fiber-Type Distribution of Different Muscle Groups among Athletes.

Source: Data from [Tesch and Karlsson \(1985\)](#).

FOCUS ON RESEARCH

Does Fiber-Type Distribution Affect Maximal Oxygen Uptake?

Researchers have long been interested in the distribution of fiber type in athletes. At the time that Bergh et al. undertook this classic study, researchers knew that aerobically trained individuals were characterized by a high percentage of ST fibers (and hence a lower percentage of FT fibers) and that anaerobically trained individuals (e.g., sprinters) were more likely to have a high percentage of FT fibers. Researchers also knew that aerobic training was associated with a high

$\dot{V}O_2\text{max}$ **$\dot{V}O_2\text{max}$** is the greatest amount of oxygen an individual can take in, transport, and use during strenuous work; it is considered the best measure of an individual's

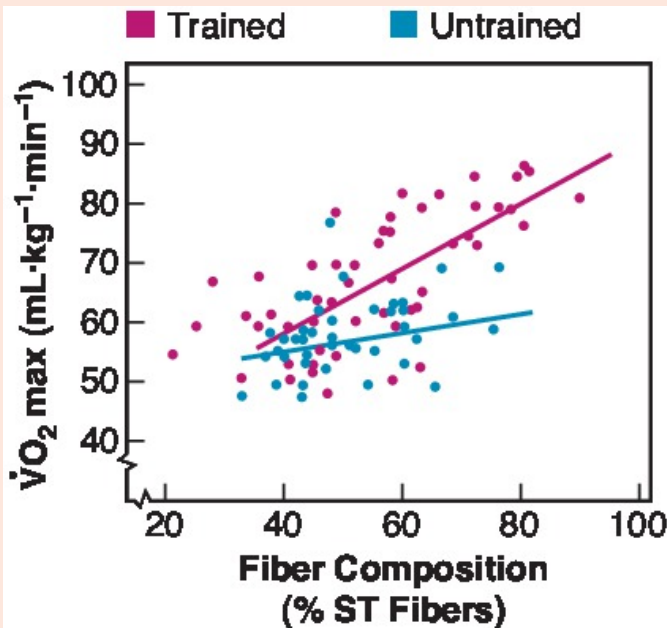
aerobic (or cardiovascular) fitness. Thus, Bergh et al. proposed that there would be a relationship between the percentage of slow-twitch fibers and a person's $\dot{V}O_2\text{max}$. Their results, shown here in the graph, support their hypothesis.

These data lead to two important conclusions:

1. There is a strong linear relationship between $\dot{V}O_2\text{max}$ and %ST fibers. This makes sense because the ST fibers have the greatest oxidative ability, that is, the ability to use oxygen to produce large amounts of ATP to support long-duration activities.

2. At any given %ST (above ~40%), an athlete has a greater $\dot{V}O_2\text{max}$ than a nonathlete. This is consistent with what we know about the trainability of muscle fibers. Endurance training increases the oxidative capacity of muscle, thereby allowing the muscle to use more oxygen and thus achieve a higher $\dot{V}O_2\text{max}$.

Source: Bergh, U., A. Thorstensson, B. Sjodin, B. Hulten, K. Piehl, & J. Karlsson: Maximal oxygen uptake and muscle fiber types in trained and untrained humans. *Medicine and Science in Sports*. 10(3):151–154 (1978).



An interesting question arises from such comparisons of fiber-type distribution in various athletes: did training and participation in a given sports influence the fiber type or did fiber type influence the type of athletic participation? Although some researchers have theorized that changes are possible in the contractile properties of muscle, most available evidence indicates that the distribution of ST and FT fibers (the types involving contractile properties) is genetically determined and cannot be altered in humans by exercise training ([Kraemer, 2000](#); [Saltin et al., 1977](#); [Williams, 1994](#)). Evidence does show, however, that training can alter the metabolic properties of the cell (enzyme concentration, substrate storage, and so on). These changes may lead to a conversion of FT fiber subdivisions. Indeed, with endurance training, the oxidative potential of FOG and FG fibers can exceed that of SO fibers of sedentary individuals ([Saltin et al., 1977](#)). Research continues to confirm the plasticity of muscle fiber type and provide evidence that different stimuli can cause a muscle to change fiber type. But, considerably more research is necessary to understand whether such changes are the exception to the rule ([Caiozzo, 2012](#)).

In summary, the distribution of fiber types varies considerably within the muscle groups of an individual and among individuals. The basic distribution of fiber type appears to be genetically determined. It is generally thought that exercise training does not alter the contractile properties of muscle fibers. The possibility remains, however, that training adaptations can alter the metabolic capabilities of muscle fibers sufficiently to change the classification of fiber types within the FT fibers (i.e., from FG to FOG or vice versa).

FOCUS ON RESEARCH

Are There Sex Differences in Muscle Fiber Power Production in Older Adults?

It is well known that males are generally stronger than females. The purpose of this study was to determine if differences in power at the single muscle fiber level contribute to the sex difference in whole muscle power production in elderly individuals. Sixteen older adults (mean age = 72 years) participated in the study. A muscle biopsy procedure was performed to obtain muscle fibers.

As expected, the males were stronger in a double-knee press and had greater right knee extension power than females (although some measures of strength and power did not differ between these males and females). However, the slow oxidative (SO) and fast oxidative glycolytic (FOG) fibers of these males and females did not differ significantly in power production.

Thus, it appears that power-generating capacity differs by muscle fiber type ($SO < FOG$), but not by sex. Rather, it appears that the primary reason that males are stronger than females is that they possess greater muscle mass.



Source: Krivickas, L. S., R. A. Fielding, A. Murray, D. Callahan, A. Johansson, D. J. Dorer, & W. R. Frontera: Sex differences in single muscle fiber power in older adults. *Medicine & Science in Sports & Exercise*. 38(1):57–64 (2006).

Summary

1. Skeletal muscles provide for locomotion and manipulation,

maintain body posture, and play an important role in heat generation.

2. The muscle characteristics that allow production of movement include irritability, contractility, extensibility, and elasticity.
3. A motor neuron along with the muscle fibers it innervates is called a motor unit. Because each muscle fiber in a motor unit is connected to the same neuron, the electrical activity in the motor neuron controls the contractile activity of all the muscle fibers in a given motor unit.
4. Skeletal muscle fibers are bundled together into groups of fibers called fasciculi. A muscle fiber is itself composed of smaller units called myofibrils, which are made up of myofilaments.
5. The two types of myofilaments are the thick and thin filaments. The repeating pattern of these myofilaments along the length of the myofibril gives skeletal muscle its striated appearance.
6. The repeating unit, or sarcomere, is the functional unit of the muscle.
7. Tropomyosin is a regulatory protein that blocks the active site on actin, thereby inhibiting actin and myosin from binding under resting conditions. The position of tropomyosin is controlled by troponin.
8. Structural muscle proteins include, among others, filamins, desmin, nebulin, α -actinin, tropomodulin, myomesin, and titin. These proteins are required for the sarcomere to function properly.
9. Titin is a large protein that connects the Z line to the M line in the sarcomere. It functions as a molecular spring that prevents overstretching of the sarcomere and provides a recoil of the sarcomere after it has been stretched.
10. Excitation-contraction coupling is the sequence of events by which an action potential in the sarcolemma initiates the contractile process of the myofilaments.
11. Excitation-contraction coupling has three phases: the spread of depolarization, the binding of calcium to troponin, and the generation of force (cross-bridge cycling).

12. Force is generated in the cross-bridging cycle. This cycle consists of the binding of myosin to actin, the power stroke, the dissociation of myosin and actin, and the activation of myosin heads.
13. The spread of depolarization (action potential) is carried into the interior of the muscle fiber by the T tubules. As the electrical signal moves into the cell, it causes the release of calcium, which is stored in the lateral sacs of the sarcoplasmic reticulum.
14. Calcium released from the sarcoplasmic reticulum binds to the troponin molecules, which undergo a configuration change, thereby removing tropomyosin from its blocking position on the actin filament. This allows the myosin cross-bridges (heads) to bind with the actin filaments.
15. The generation of tension within the contractile elements results from the binding of actin and myosin, which causes the release of stored energy in the myosin heads.
16. ATP plays several important roles in muscle contraction. The hydrolysis of ATP provides the energy to activate or reactivate the myosin head before binding with actin. ATP binding is also necessary to break the linkage between the myosin cross-bridge and actin so that the cycle can repeat. ATP is also used to return calcium to the sarcoplasmic reticulum and to restore the resting membrane potential.
17. During relaxation, calcium is pumped back into the sarcoplasmic reticulum (by active transport), and troponin no longer keeps tropomyosin from its blocking position.
18. When a muscle fiber or motor unit is stimulated to contract, it contracts to its fullest extent or does not contract at all. This is known as the all-or-none principle.
19. Human muscle fibers are categorized as two different types, ST and FT, based on their contractile properties. The FT fibers can be further classified as FOG or FG fibers based on their metabolic properties. ST fibers are metabolically oxidative or SO.
20. Athletes involved in endurance activities typically have a high percentage of slow-twitch fibers. Athletes involved in power activities typically have a high percentage of fast-twitch muscle fibers.

21. Training alters the metabolic capabilities of muscle fibers, but not their contractile properties. It is possible that metabolic alterations could be significant enough to change the classification of fibers within the FT fibers (FOG to FG and vice versa).

Review Questions

1. List, in order of largest to smallest, the major components of the whole muscle.
 2. What causes the striated appearance of skeletal muscle fibers?
 3. What are the T tubules and the sarcoplasmic reticulum? What is the function of each?
 4. Relate each region of the sarcomere to the presence of thick and thin myofilaments.
 5. Diagram a sarcomere at rest and at the end of a contraction, and identify each of the areas.
 6. Identify the key structural proteins to the sarcomere and what role they play in muscular contraction.
 7. Describe the role of the regulatory proteins in controlling muscle contraction.
 8. Describe the sequence of events in excitation-contraction coupling.
 9. Identify the role of ATP in the production of force within the contractile unit of muscle.
 10. What is the role of calcium in muscle contraction?
 11. Explain the five functional roles of titin in skeletal muscle.
 12. Describe the all-or-none principle as it relates to the contraction of a single muscle fiber.
 13. Diagram the force production, twitch speed, and fatigue curve for the different fiber types.
 14. Discuss the possibility of influencing fiber-type distribution by exercise training.
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Literature Search

In this chapter, we discussed the anatomy, physiology, as well as functional and structural properties of skeletal muscle. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

1. Search skeletal muscle, this will yield a huge selection of articles.
2. Refine your search using key terms that may reflect your interest in this area. For example,
 - a. Skeletal muscle sarcomere proteins
 - b. Skeletal muscle function
 - c. Skeletal muscle microscopic structure
 - d. Sliding filament theory
 - e. Continue your search for aspects of this topic that are of particular interest to you.

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18 Muscle Contraction and Movement



CHAPTER OUTLINE

Introduction: Exercise—the Result of Muscle
Contraction591

Muscular Force Production

- Tension versus Load
- Classification of Muscle Contractions
- Force Development

Muscular Fatigue and Soreness

- Muscular Fatigue
- Type of Activity and Muscle Fatigue
- Muscle Soreness

Measurement of Muscular Function

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The Influence of Age and Sex on Muscle Function

Male-Female Differences

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Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Differentiate between force and load.
- Identify the different types of muscle contraction and the terms to describe muscle fiber contraction or whole-muscle contraction.
- Compare and contrast concentric and eccentric dynamic contractions.
- Describe neural and mechanical factors that affect force development.
- Differentiate between the length-tension relationship as it applies to a muscle fiber and as it applies to a whole muscle.
- Identify possible causes of muscle fatigue, and indicate the most probable cause of muscle fatigue for various types of activities.
- Outline the integrated model of delayed-onset muscle soreness and explain why DOMS can be a concern.
- Refute the popular belief that delayed-onset muscle soreness is caused by an accumulation of lactic acid in the muscle.
- Identify the different laboratory and field methods for measuring muscular function.
- Describe the basic pattern of strength development.
- Compare and contrast the expression of strength in males and

females.

- Describe the impact of age on strength expression in children/adolescents and older adults, and explain the factors affecting older adults.

Introduction: Exercise—the Result of Muscle Contraction


Dictionaries generally define exercise as an activity performed using muscles. In other words, the contraction of muscles is exercise. Therefore, it is impossible to discuss the exercise response of the neuromuscular system in the same way that it is discussed for the cardiovascular-respiratory or metabolic systems.

Although not all muscle action results in exercise, all exercise is done by muscle action. An almost endless number of exercises result from the many ways different body muscles can contract. For example, imagine a basketball player taking off from the foul line for a slam dunk, a bodybuilder flexing in a pose, a swimmer doing a front crawl stroke, and the movement of your eyes across the pages of this book. All of these activities require different muscle actions—explosive dynamic power; controlled static activity; force against a resistance through a large range of motion; and highly coordinated, rapid, but minimal force movement. Although the individual actions involving muscle contractions vary widely, they can be classified in general ways. These classifications help us understand differences in how muscle contractions produce force.

Muscular Force Production

Tension versus Load

The force developed when a contracting muscle acts on an object is called **muscle tension**. The force exerted on the muscle by the object is called the **load**. The load and muscle tension are opposing forces.



Muscle Tension Force developed when a contracting muscle acts on an object.

Load Force exerted on the muscle.

The term **contraction** refers to the tension-producing process of the contractile elements within muscle. However, not all contractions produce movement; movement depends on the magnitude of the load exerted and the tension produced by the muscle. In order for a muscle to move a load, the force of muscle tension must exceed the force of the load. Furthermore, muscle tension developed experimentally in an isolated muscle fiber, a motor unit, or even a whole muscle is not necessarily the same as the force developed by an intact muscle in the human body.

Contraction Tension-producing process of the contractile elements within muscle.

All human motion involves rotation of body segments about their joint axes. The capability of a force to produce rotation is referred to as **torque**. Thus, torque is force applied at some distance away from the center of the joint, causing the limb to rotate around the joint. Torque changes as a muscle moves bone through the range of motion.

Torque The capability of a force to produce rotation of a limb around a joint.

Classification of Muscle Contractions

A classification of muscle contractions is presented in **Table 18.1**. This classification is based on three component characteristics:

time (as duration and/or velocity), displacement (length change), and force production. Throughout this discussion, it is important to distinguish whether a contraction is being described in an isolated muscle fiber/motor unit or in intact, whole muscles.

TABLE 18.1 Classification of Contractions

| Type of Contraction | | | |
|--|--|--|------------------------------|
| Muscle Fiber or Motor Unit | Intact Muscle in Humans | External Work in Intact Muscle in Humans | Function of Contraction |
| Isotonic: constant force production: shortening, or lengthening | Dynamic concentric: muscle force varies as muscle shortens to accommodate change in muscle length and/or joint angles as limb moves through ROM while moving a constant external load | Positive: (work = force \times distance); external load can be overcome | Acceleration |
| | Dynamic eccentric: see above, except muscle lengthens | Negative: (work = force \times distance); external load assists lengthening | Deceleration |
| Isokinetic: constant velocity of lengthening or shortening | Isokinematic (dynamic): rate of limb displacement or joint rotation is constant. Velocity varies with joint angle | Positive or negative: see above | Acceleration or deceleration |
| Isometric: constant muscle length | Static: limb displacement or joint rotation does not occur; little muscle fiber shortening occurs | Zero: (work = force \times distance, where distance = 0); external load cannot be overcome | Fixation |

Note: ROM, range of motion.

Sources: Komi (1984); Williams (1994).

As shown in **Table 18.1**, at the muscle fiber or motor unit level, the three basic types of contraction are isotonic, isokinetic, and isometric. At the whole-muscle level, contractions may be defined as dynamic, isokinematic, or static. The following paragraphs describe these different contractions in a muscle fiber and the corresponding types of contraction in an intact muscle.

An **isotonic contraction** is a muscle fiber contraction in which the tension generated by the muscle fiber is constant throughout the range of motion. The term isotonic indicates that the force production (tonus) is unchanged (iso means “same”) when the muscle fiber contracts, causing movement of an external load. In the intact human system, such a contraction is practically impossible. What happens in intact whole muscle is that the muscle force varies as the muscle contracts. This variation is necessary to accommodate changes in muscle length and/or joint angles as limbs move through their ranges of motion. Thus, the load is constant, but the force produced to move it through the range of motion is not. Therefore, the term dynamic is more accurate to describe contraction within the intact human.

Isotonic Contraction A muscle fiber contraction in which the tension generated by the muscle fiber is constant through the range of motion.

A **dynamic contraction** is a whole-muscle contraction that produces movement of the skeleton. If the movement results from a dynamic muscle contraction that produces tension during shortening, it is a **concentric contraction**. Concentric contractions result in positive external work and are primarily responsible for acceleration in movement. The lifting action of the biceps in curling a barbell is an example of a concentric dynamic contraction. The sliding filament theory describes this type of contraction (see [Chapter 17](#)).

Dynamic Contraction A muscle contraction in which the force exerted varies as the muscle shortens to accommodate change in muscle length and/or joint angle throughout the range of motion while moving a constant external load.

Concentric Contraction A dynamic muscle contraction that produces tension during shortening.

If movement occurs as a result of a dynamic muscle contraction that produces tension while lengthening, the contraction is referred to as an **eccentric contraction**. Eccentric contractions result in negative work and are primarily responsible for deceleration in movement. The lowering action of the biceps in curling a barbell is an example of an eccentric dynamic contraction. During an eccentric contraction, the cross-bridges are cycling as the filaments are being pulled apart. Because of this action of being pulled apart, strenuous eccentric activities can cause mechanical disruption of the sarcomeres, muscle damage, and soreness ([Peake et al., 2005](#)). Strenuous eccentric contractions also allow for a greater force of contraction at a

lower energy cost than do concentric contractions (Stauber, 1989). Skeletal muscle fibers produce 1.5–1.9 times more force during maximal eccentric contraction than during maximal isometric contraction, whereas an eccentric contraction in whole muscle is slightly greater than a concentric contraction (Aagaard and Bangsbo, 2006; Byrne et al., 2004).

Eccentric Contraction A dynamic muscle contraction that produces tension (force) while lengthening.

Why can an eccentric contraction produce more force than a concentric one? During an eccentric contraction, some of the cross-bridges do not cycle but are continually pulled backward. Because of this, the myosin heads do not rotate forward, the actin and myosin remain bound (Stauber, 1989) and more tension can be produced. Additionally, titin is also involved in this phenomenon. As previously described in Chapter 17, titin works as an active component in the sarcomere and is a main determinant of muscle force as both a passive spring and a generator of active contractile work (Freundt & Linke, 2019; Fukutani and Herzog, 2019; Herzog, 2019; Hessel et al., 2017). It is now clear that both cross-bridges and titin facilitate the increased force capable during eccentric contractions (Hessel et al., 2017).

Why does eccentric (negative) work have a lower energy cost than does concentric (positive) work? The answer to this question is not completely known, but three possibilities have been suggested (Stauber, 1989). First, fewer muscle fibers are recruited during eccentric contractions than during concentric contractions; fewer fibers use less oxygen, which translates to a smaller energy cost. Second, because some of the cross-bridges do not cycle, less ATP is broken down and used as energy. The third possibility may be due to the structure of titin. As detailed in Chapter 17, titin has spring-like properties. When a muscle stretches, titin domains unfold, lengthening the “spring” and storing energy. When muscles contract, myosin heads engage and decrease the force on titin. This allows the titin to refold and shorten which, in turn, delivers folding contractile (recoil) energy without any

additional ATP being needed (Eckels et al., 2018). Researchers estimate that positive work uses three to nine times more energy than does negative work. To appreciate the applicability of this information, mentally compare climbing up stairs in a high-rise building with walking down them.

An **isokinetic contraction** is a muscle fiber contraction in which the velocity of contraction is kept constant. Isokinetic contractions are also dynamic contractions in that movement occurs; the force generated is sufficient to overcome the external load. In the intact human, the velocity of the movement actually varies with the joint angle; however, the rate of limb displacement can be held constant with special exercise equipment. Hence, the term **isokinematic contraction** is more technically correct, although seldom used. What differentiates isokinetic contractions from other forms of dynamic contractions is that the rate of shortening or lengthening (kinesis means “motion”) is constant (iso means “same”).

Isokinetic Contraction A muscle fiber contraction in which the velocity of the contraction is kept constant.

Isokinematic Contraction A muscle contraction in which the rate of limb displacement or joint rotation is held constant with the use of specialized equipment.

An **isometric contraction** is a muscle fiber contraction without a length change in the muscle fiber. In an isometric contraction, the cross-bridges are cycling—hence producing tension—but sliding of the filaments does not occur. The actual length (metric derives from meter and refers to length) is constant. This kind of action is possible in an isolated fiber because the fiber can be stabilized at both ends in an experimental apparatus before being stimulated. However, an intact fiber has an elastic element (connective tissue and joints) to contend with, and so some fiber shortening actually does occur even though no limb displacement or joint rotation occurs. A

static contraction is a muscle contraction that produces an increase in muscle tension but does not cause meaningful limb displacement or joint rotation and, therefore, does not result in movement of the skeleton. Thus, the term static, meaning “not in motion,” is better than isometric for describing such a contraction that occurs in an intact muscle in humans. No external work is done by static contractions, but they often perform the important task of fixation or stabilization. Gripping a tennis racket or barbell is an example of a static contraction.

Isometric Contraction A muscle fiber contraction that does not result in a length change in muscle fiber.

Static Contraction A muscle contraction that produces an increase in muscle tension but does not cause meaningful limb displacement or joint displacement and, therefore, does not result in movement of the skeleton.

Force Development

All types of contractions exert tension; however, the amount of tension that can be generated is not the same for all contractions. In whole muscles, eccentric dynamic contractions produce the greatest force, followed by static contractions and then by dynamic concentric contractions.

The amount of force produced by each type of contraction depends on neural and mechanical factors (Komi, 1984). This is true on the level of the muscle fiber and also in the intact, whole muscle. The following sections discuss how neural and mechanical factors influence force development. Each section first discusses the muscle fiber and then the more complex environment of whole muscles.

Neural Activation

A single muscle fiber or motor unit that is stimulated to contract

will contract maximally or not at all (according to the all-or-none principle of muscle contraction described in [Chapter 17](#)). When stimulated, a muscle fiber produces a *twitch* and then relaxes (**Figure 18.1A**). If a second stimulus is applied before the fiber has completely relaxed, *temporal summation* results, producing slightly greater tension. If the frequency of stimulation is increased enough to prohibit relaxation, the individual contractions blend together to form first an *irregular* (or *unfused*) *tetanus* and then a *smooth* (*fused*) *tetanus* (**Figure 18.1B**). Unfused tetanic contractions produce greater tension than do either single twitches or summations, and fused tetanic contractions produce the highest tension.

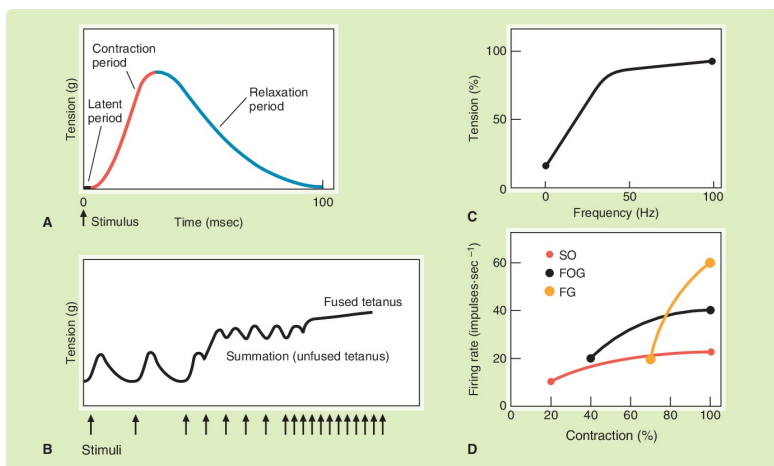


Figure 18.1 Muscle Response to Stimulation.

A. Myogram of a single muscle fiber twitch. **B.** Myogram of a series of muscle fiber twitches. **C.** Effect of frequency of stimulation on muscle tension in whole muscle. **D.** Firing rate and muscle tension in different fiber types in whole muscle. SO, slow oxidative; FOG, fast oxidative glycolytic; FG, fast glycolytic.

In intact muscle, contractions occur with tetanic contractions of motor units. Unlike single muscle fiber or motor unit contractions, whole-muscle contractions do not occur in an all-or-none fashion. Whole-muscle contractions can be graded, meaning

a muscle contraction can produce little force (for instance, to move your hand while turning a page) or a lot of force (for instance, to lift a heavy barbell). Two neural factors determine force production in whole muscle. The first factor is the frequency of stimulation, or *rate coding* (Enoka, 1988). As shown in **Figure 18.1C**, as the frequency of stimulation increases, so does the force that the muscle produces. Notice that at frequencies less than 50 Hz, a small increase in frequency of firing can produce a large increase in muscle tension (Sale, 1992).

The second neural way of controlling force output is by varying the number of motor units activated, which is called recruitment, or *number coding*. In the intact human, some motor units are always contracting in an alternating manner. These contractions maintain what is called *muscle tone*, or *tonus*. When greater tension is needed, the processes of rate coding and number coding occur in tandem. As the firing of a given motor unit reaches its maximum, new motor units are recruited. Such activation or recruitment, and the subsequent deactivation or derecruitment, occurs according to the size principle. Remember that smaller motor neurons are easier to stimulate than larger ones. Therefore, in accordance with the size principle, the small $\alpha 2$ motor neurons innervating slow oxidative (SO) motor units are recruited first (**Figure 18.1D**) (Sale, 1992). As the stimulus increases, the larger $\alpha 1$ motor neurons that innervate the fast oxidative glycolytic (FOG) muscle fibers are activated, followed by the recruitment of fast glycolytic (FG) fibers as the stimulus increases. For example, the speed continuum of walk, jog, and run involves a continuum of SO, FOG, and FG muscle fiber recruitment. Once the neural stimulation ends, deactivation proceeds in reverse sequence. The larger, faster fibers (FG), then FOG, are inactivated first; finally, the slower, smaller fibers are deactivated until only muscle tonus remains.

Mechanical Factors Influencing Muscle Contractions

Several mechanical factors influence the force produced during muscle contractions. This section discusses four mechanical factors: length-tension-angle relationships, force-velocity relationships, the elasticity-force relationship, and cross-sectional

area/architectural design. Force production in a muscle fiber is discussed first, followed by the more complex situation in whole muscles.

LENGTH-TENSION-ANGLE RELATIONSHIPS Within a muscle fiber, the amount of tension that can be exerted is related to the initial length of the sarcomeres. As shown in **Figure 18.2A**, this relationship forms an inverted U (Edman, 1992). The amount of tension produced is directly related to the degree of overlap of the thick and thin filaments. In elongated fibers, there is little overlap of the actin and myosin filaments, making it hard for cross-bridges to form. In shortened fibers, where the thick and thin filaments already overlap almost completely, there is little room for further shortening. Thus, less force is produced in both the elongated and shortened positions. In contrast, the maximum number of cross-bridges coincides with the highest force production, which occurs at approximately 100–120% of the resting sarcomere length (Edman, 1992).

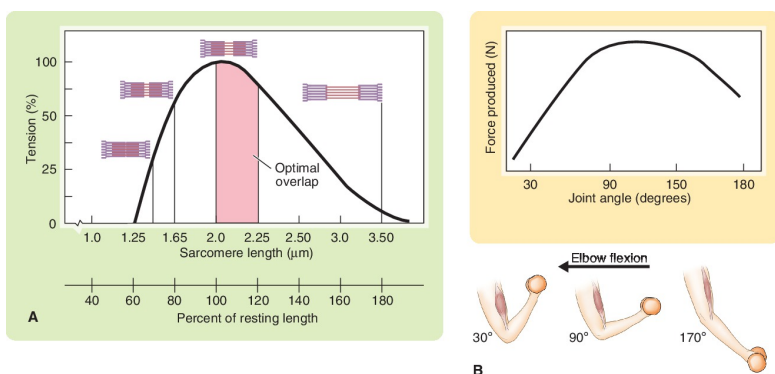


Figure 18.2 Length-Tension Relationship in Muscle.

A. Length-tension relationship in skeletal muscle fibers. **B.** Example of a strength curve illustrating the length-tension relationship in whole muscle. **Source:** Republished with permission of John Wiley & Sons from Edman, K. A. P.: Contractile performance of skeletal muscle fibers. In Komi, P. V. (ed.): *Strength and Power in Sport*. Champaign, IL: Human Kinetics, 96–114 (1992); permission conveyed through Copyright Clearance Center, Inc.

In whole muscle, this length-tension relationship also holds, but its expression is complicated by many factors. These factors include the cross-sectional area of the muscle, the arrangement of the sarcomere in relation to the line of pull, the level of neural muscle activation, the degree of fatigue, the involvement of the elastic components of muscle (which, in part is due to the properties of titin), and the biomechanical aspects of how a muscle exerts force at a joint. The biomechanical aspects are most notable, as described below.

When whole-muscle tension (measured as muscle force or torque) is plotted against the joint angle at which it occurs, strength curves are generated (**Figure 18.2B**). Imagine the muscle action involved in performing a bicep curl. As the barbell is lifted through the joint's range of motion, different amounts of force are generated to compensate for biomechanical differences related to joint angle. Of course, the force produced depends on the length of the sarcomere, but force production is also greatly influenced by biomechanical aspects of the joint. **Figure 18.2B** shows the strength curve for this bicep curl (elbow flexion) example. The joint angle is plotted along the horizontal axis, and the force of the contracting muscle is plotted along the vertical axis. In this graph, the action of raising the barbell proceeds from right to left because the joint angle is greatest in the lowered position (~170 degrees) and smallest with the barbell in the raised position (~30 degrees). In this example, peak force occurs at approximately 100–120 degrees.

As indicated in **Figure 18.3**, strength curves occur in three forms: ascending, descending, or ascending and descending. An *ascending strength curve* is characterized by an increase in torque as the joint angle increases. A *descending strength curve* is characterized by a decrease in torque as the joint angle increases. In an *ascending and descending strength curve*, the force initially increases and then decreases as a function of joint angle.

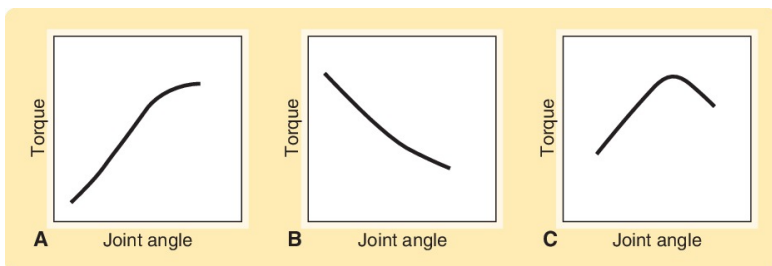


Figure 18.3 Classification of Strength Curves.

A. Ascending. **B.** Descending. **C.** Ascending and descending.

Source: Reprinted with permission from Kulig, K., J. G. Andrews, & J. G. Hay: Human strength curves. In Terjung, R. (ed.): *Exercise and Sport Sciences Reviews* (Vol. 12). Lexington, MA: Collamore Press, 417–466 (1984).

Figure 18.4A presents a generalized strength curve for knee flexion. Most studies describe this as an ascending curve, although some findings disagree ([Kulig et al., 1984](#)). As shown in the figure, when the joint angle is greatest, the greatest force is exerted. This point also corresponds to the point at which the muscles of the hamstrings are longest.

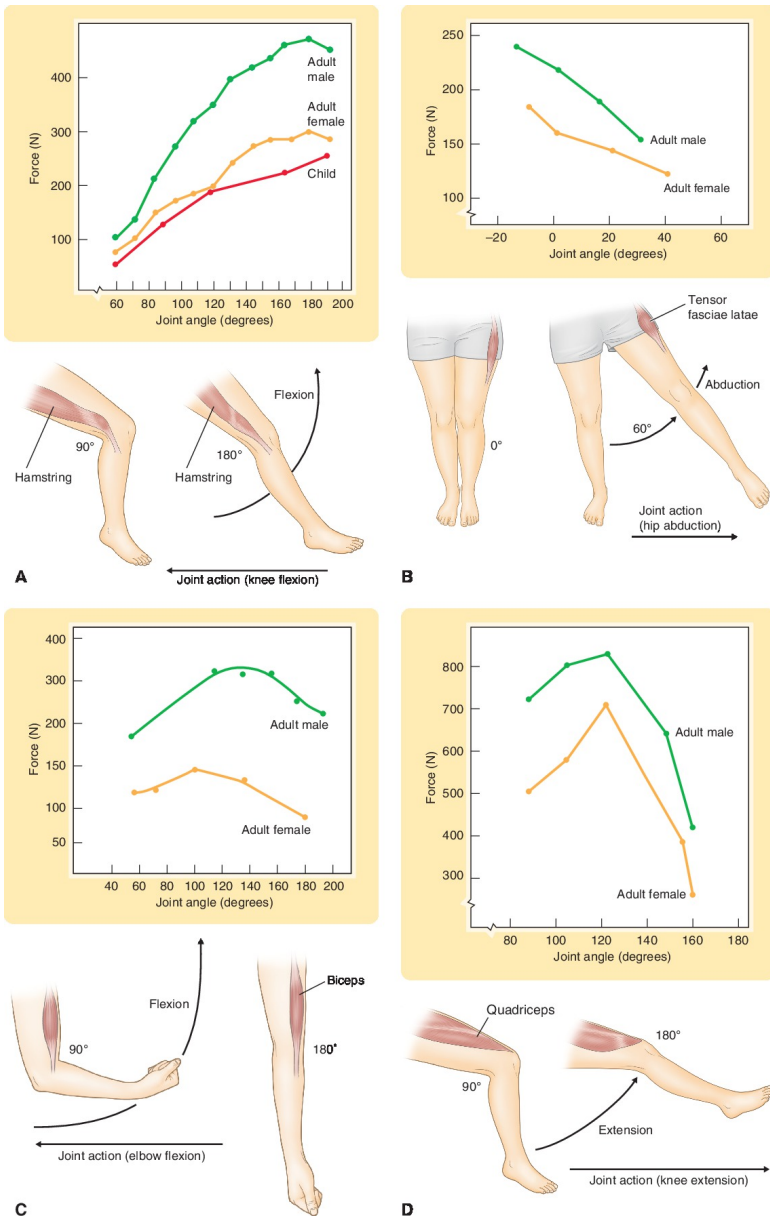


Figure 18.4 Strength Curves.

A. Knee flexion. **B.** Hip abduction. **C.** Elbow flexion. **D.** Knee extension.

Source: Reprinted with permission from Kulig, K., J. G.

Andrews, & J. G. Hay: Human strength curves. In Terjung, R. (ed.): *Exercise and Sport Sciences Reviews* (Vol. 12). Lexington, MA: Collamore Press, 417–466 (1984).

Figure 18.4B presents a generalized strength curve for hip abduction (Kulig et al., 1984). This curve is generally described as descending. In this case, larger joint angles correspond to lower forces. In this example, the tensor fasciae latae (the muscle responsible for hip abduction) is longest at the lowest joint angle.

Both of these situations—**Figure 18.4A and B**—are in reality consistent: The longer the muscle length, the greater the force exerted. However, remember that it is not just, or even primarily, the length of the muscle itself that causes this variation.

There is general agreement that both elbow flexion (**Figure 18.4C**) and knee extension (**Figure 18.4D**) exhibit ascending and descending strength curves (Kulig et al., 1984). The strongest angles for elbow flexion seem to be between 90 and 130 degrees; for knee extension, the strongest angles are between 100 and 130 degrees. As with the other configurations, the joint angle that coincides with the greatest force production is partially, but not entirely, a result of the muscle length and degree of overlap of the thick and thin filaments.

Although there are some differences, the strength curves are similar for males and females, and the young and the old. Injuries typically result in low strength curve values; thus, preinjury strength curves or strength curves from a comparable healthy individual are sometimes used as goals in rehabilitation. Standard norms for strength curves are not generally available.

Knowledge of strength curves has another practical application. When lifting an external load—for example, a barbell through the range of elbow flexion (bicep curl exercise)—the individual is limited by the weight that can be handled at the weakest point. The stronger angles are not overloaded as much as the weaker angles, and the muscle is taxed maximally only at its “sticking point.”

Finally, strength curves have been used to design strength training equipment—specifically, variable-resistance equipment (Kulig et al., 1984). Nautilus developed the variable-radius cam

to mimic the strength curve of selected single-joint exercises. The aim is to compensate for changes in mechanical leverage throughout the range of motion of a joint, thereby matching the load to the strength curve so that the muscles are taxed maximally at all angles, not just at the sticking point. Although this reasoning seems logical, training studies have not demonstrated that greater benefits are derived from variable-resistance training programs than from traditional-free weight programs.

FORCE-VELOCITY AND POWER-VELOCITY RELATIONSHIPS

The second major mechanical factor that influences the expression of muscle force or torque is the velocity at which the shortening occurs.

The relationship between force and velocity is similar for a single muscle fiber (**Figure 18.5A**) and an intact whole muscle (**Figure 18.5B**). The single muscle fiber does not exhibit the smooth hyperbolic curve of an intact muscle, but both show the basic relationship: The shortening velocity of a muscle increases as the force developed by the muscle decreases, which means that a muscle can shorten fastest when the load is the lightest. Maximal velocity occurs in an unloaded (zero force) situation, and a maximal load results in no movement (zero velocity). Between these two extremes, the velocity gradually decreases in a curvilinear fashion as the load increases. To apply this information, think about swinging a baseball bat. As the weight of the bat increases, the speed with which it can be swung decreases.

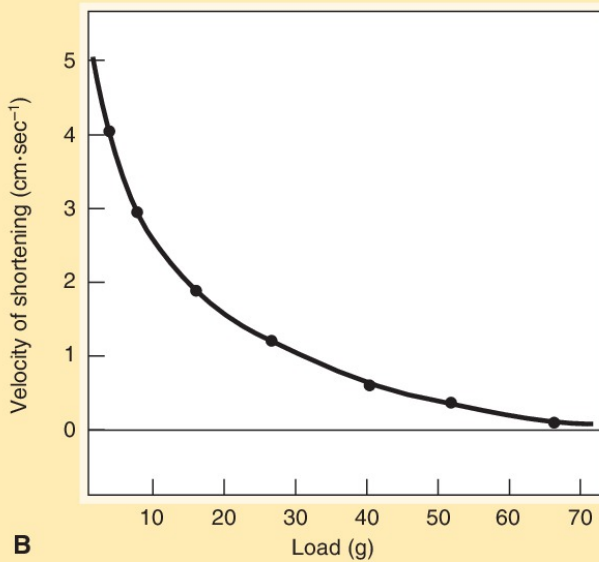
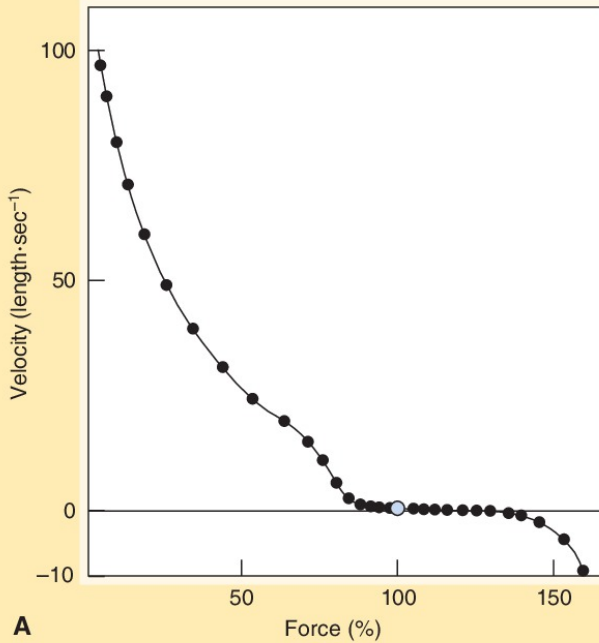


Figure 18.5 Force-Velocity Curves.

A. In a single muscle fiber. **B.** In a whole muscle. **Source:** Republished with permission of John Wiley & Sons from

Edman, K. A. P.: Contractile performance of skeletal muscle fibers. In Komi, P. V. (ed.): *Strength and Power in Sport*. Champaign, IL: Human Kinetics, 96–114 (1992); permission conveyed through Copyright Clearance Center, Inc.

If the external force overcomes the ability of the muscle to resist it, the muscle lengthens (eccentric contraction) due to a certain degree of neural inhibition, which prevents muscle contraction (Hahn, 2018). The eccentric contraction is seen in **Figure 18.5A** where the force curve dips below the horizontal axis. Notice that the force produced continues to increase during this eccentric phase. This result is consistent with earlier statements made about eccentric dynamic contractions.

It is interesting to note that on the cellular level, the maximal velocity of shortening varies among individual muscle fibers, but within the same fibers, maximal velocity of shortening is constant regardless of the sarcomere length (**Figure 18.6**) (Edman, 1992).

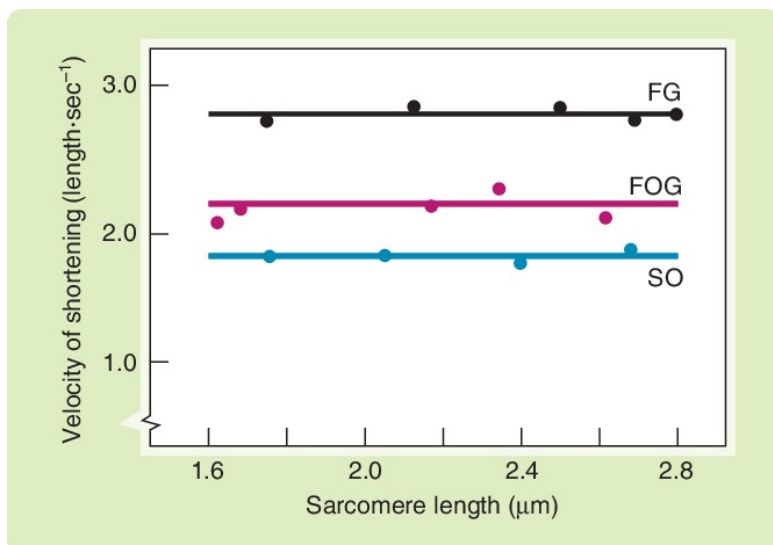


Figure 18.6 Velocity-Length Curves.

Source: Republished with permission of John Wiley & Sons from Edman, K. A. P.: Contractile performance of skeletal muscle fibers. In Komi, P. V. (ed.): *Strength and Power in*

Sport. Champaign, IL: Human Kinetics, 96–114 (1992); permission conveyed through Copyright Clearance Center, Inc.

Compare **Figure 18.6** with **Figure 18.2** (the length-tension curve). This comparison shows that the length of the sarcomere influences force production but does not affect the velocity of force production. The maximum velocity of shortening does not depend on the number of cross-bridges between the thick and thin filaments but on the maximal cycling rate of the cross-bridges ([Edman, 1992](#)).

Figure 18.7 shows the relationship between power and velocity in whole muscles. These data were derived from a study of two different groups of subjects: one with more than 50% FT muscle fibers (average = 58.5%) and the other with less than 50% FT fibers (average = 38.3%) ([Coyle et al., 1976](#)). Several conclusions can be drawn from this graph.

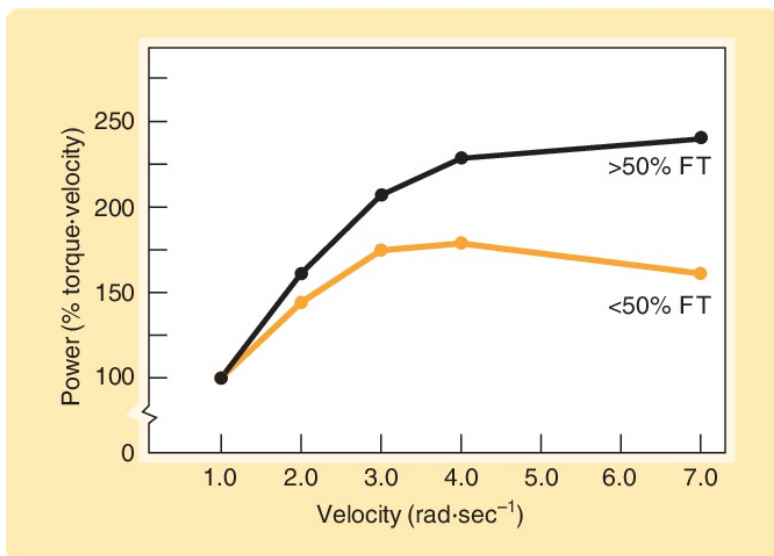


Figure 18.7 Influence of Fiber Type on Velocity-Power Relationship.

Source: Reprinted with permission from Coyle, E. F., D. L. Costill, & G. R. Lesmes: Leg extension power and muscle

1. Power is positively related to velocity.
2. The shape of the relationship is curvilinear. Consider what this power-velocity relationship would mean if a heavier (rather than lighter) bat can be swung quickly enough to contact a fast pitch.
3. The graph in **Figure 18.7** shows that the power that can be generated at any given velocity varies with the predominant fiber type. At any given velocity, the resulting power is higher in individuals with more than 50% FT fibers than in individuals with less than 50% FT fibers. Thus, individuals with high percentages of FT fibers would seem to be genetically predisposed, given sufficient training and practice, to be more successful in power events such as sprinting, power lifting, and jumping and in field events such as shot, javelin, and discus.
4. The power curve of the predominantly ST fiber individuals (those with <50% FT) not only levels off but also makes a downturn at the higher velocities. Although such a downturn is not evident at the tested speeds for the FT group, it is likely that there is a fiber-type-specific optimal speed of movement for the generation of power.

ELASTICITY-FORCE RELATIONSHIP Elasticity is the third mechanical factor involved in the force development capabilities of muscles. Both the muscle fibers and their tendinous attachments contain elastic components. The most common mechanisms to explain the elasticity-force relationship are stretch-reflex activation and the storage of energy in tendons. There is, however, a third possibility. That is, events in the actin-myosin-titin complex of the sarcomeres may be involved as well. When a muscle fiber is stretched and then contracted, the resultant contraction is stronger than it would have been without prestretching. In the intact human, the relationship between prestretch and force of contraction is expressed as a stretch-

shortening cycle (SSC), where the preactivated muscle is first stretched (eccentric action) and then followed by the shortening (concentric) action. Titin is an elastic component and probably responsible for this production of residual force enhancement (RFE) induced by stretching an active muscle. Therefore, it seems likely that RFE is also induced during SSC ([Fukutani et al., 2021](#); [Komi, 1992](#); [Nicol et al., 2006](#)).

Stretch-shortening cycles do not occur in all human movements, however. They are most evident in activities such as running, jumping, and (to a lesser extent) bicycling. **Figure 18.8** shows that the calf muscles and Achilles tendon are stretched while contracting eccentrically as a runner's foot impacts the ground ([Komi, 1992](#)). The push-off, or concentric-shortening phase, is aided by the release of the potential energy stored during the stretch phase. To feel the impact of the SSC, stand up and see how high you can jump with as little flexion as possible and then with moderate flexion at your hip, knee, and ankle joints. Finally, try a rebound jump from a small stool. This latter task is an example of a *plyometric exercise*. A plyometric exercise is one in which a concentric contraction is immediately preceded by an eccentric contraction (such as the eccentric contraction upon landing and absorbing the force from jumping off a stool before the concentric contraction of jumping up). Plyometrics will be discussed more thoroughly in the section on flexibility in [Chapter 20](#), because more than just elasticity is involved in the physiological basis for their execution. As a result of additional power generation with increasing stretch-proceeding contraction, you should be able to jump successively higher in the three examples.

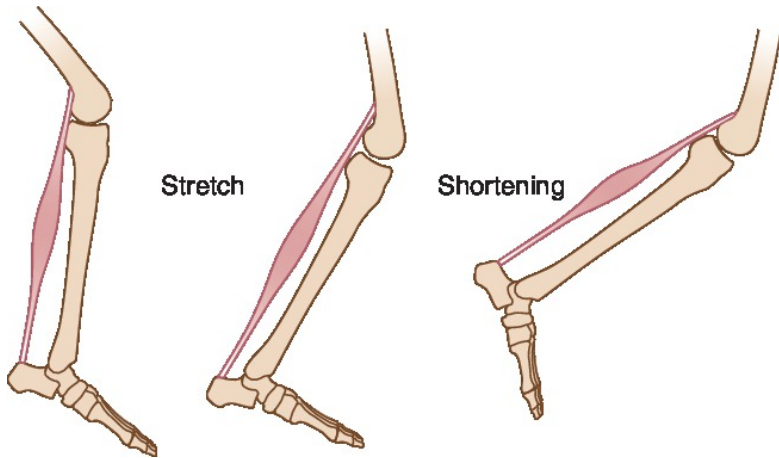


Figure 18.8 Stretch-Shortening Cycle.

Source: Komi, P. V.: Physiological and biomechanical correlates of muscle function: Effects of muscle structure and stretch-shortening cycle of force and speed. In Terjung, R. (ed.): *Exercise and Sport Sciences Reviews* (Vol. 12). Lexington, MA: Collamore Press, 81–122 (1984).

The stretch-shortening cycle not only leads to more powerful muscle contractions but also affects mechanical efficiency. Efficiency is discussed at length in [Chapter 4](#). The point here is that more work can be done with less energy expenditure following a prestretch.

CROSS-SECTIONAL AREA/ARCHITECTURAL DESIGN The maximal force that can be developed within a muscle fiber is related to its cross-sectional area. The force that can be developed by a whole muscle is also related to its cross-sectional area. However, this relationship is not as strong as the one within a single muscle fiber, largely owing to different arrangements of the muscle fibers within a muscle. Muscles that are designed for a high force generation (pennate, bipennate, and multipennate muscles) are arranged anatomically to maximize their cross-sectional area. Muscles designed for high-velocity shortening have parallel fibers and are arranged in a fusiform manner ([Goldspink, 1992](#)).

Muscular Fatigue and Soreness

Despite all the benefits and enjoyment of exercise, unaccustomed muscular activity can also lead to some unpleasant outcomes. The following section discusses two such consequences: muscular fatigue and muscular soreness.

Muscular Fatigue

Anyone who has participated in vigorous activity is familiar with muscular fatigue. Although fatigue is a familiar experience, pinpointing a definition of fatigue has been difficult. The research literature contains multiple definitions including a motor deficit, a gradual decrease in the force capacity of a muscle or the endpoint of a sustained activity. Such a broad usage of the term fatigue has made it more difficult to understand the physiological mechanisms responsible for the phenomenon ([Enoka and Duchateau, 2008](#)). The National Heart Lung and Blood Institute (NHLBI) defines fatigue as “a condition in which there is a loss in the capacity for developing force and/or velocity of a muscle, resulting from muscle activity under load which is reversible by rest” ([NHLBI, 1990](#)). This means that fatigue results in a transient loss in the ability of the muscle to develop force and or velocity, often leading to a cessation of muscular work or an inability to maintain a given intensity of work. The muscle is not damaged, and restoration and recovery are possible ([Fitts, 2008](#)). Fatigue is most often quantified as the magnitude of fall of force or power in response to a specific protocol designed to induce fatigue or as the duration of a contraction or exercise that can be maintained at a specified level ([Kent-Braun et al., 2012](#)).

But what causes muscle fatigue? Muscle fatigue is a complex and still controversial phenomenon that appears to include failure at one or more of the sites along the chain of events that leads to muscular contraction. Fatigue can be caused by many different mechanisms, and the cause of fatigue is often specific to the task being performed ([Enoka and Duchateau, 2008](#)). Some of the mechanisms that contribute to muscle fatigue during exercise include, but are not limited to, neural inhibition, calcium availability, blood flow and oxygen delivery, ATP availability,

and other metabolic factors such as pH changes, and the presence of reactive oxygen species (ROS) (Wan et al., 2017). As diagrammed in **Figure 18.9**, fatigue can be classified as central or peripheral based on the site of fatigue. Central sites refer to the nervous system (brain and spinal cord), whereas peripheral sites include specific sites in the skeletal muscle. **Table 18.2** lists the possible sites of fatigue and summarizes the proposed mechanisms for the muscle fatigue at each site. In reality, fatigue is a complex phenomenon that likely results from an interaction of these mechanisms at one or more sites. Fatigue depends on the type of exercise performed, the fiber type of the muscle involved, and the exerciser's fitness and nutritional status.

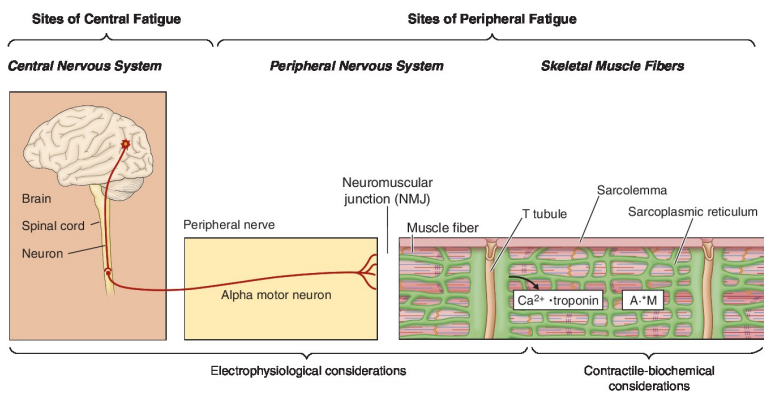


Figure 18.9 Possible Sites of Muscle Fatigue.

TABLE 18.2 Site of Muscle Fatigue and Proposed Mechanism

| Site of Fatigue | Proposed Mechanism |
|----------------------|---|
| Central | |
| CNS | Malfunction of neurons Inhibition of voluntary effort (motor cortex) Psychological factors |
| Peripheral | |
| NMJ | Inhibition of axon terminal Depletion of neurotransmitter Altered neurotransmitter binding to receptors |
| T tubule/SR | Inability to release Ca^{2+} Inability of Ca^{2+} to bind to troponin |
| Contractile elements | Depletion of ATP Depletion of PC Depletion of glycogen Accumulation of lactate, H^+ , PO_4^- , P_i , etc. |

Central fatigue may be related to neurons in the brain or spinal cord, inhibitory input from muscle afferents innervating neurons in the brain, or alterations in motor neurons. Central fatigue may also be influenced by psychological factors and motivation involving how much effort the individual makes, particularly when continuing the activity causes pain.

Peripheral fatigue refers to fatigue at a site beyond the central nervous system (CNS), anywhere from the neuromuscular junction (NMJ) to the skeletal muscle. Peripheral fatigue can occur at several specific sites: the NMJ, the sarcolemma-T tubules-sarcoplasmic reticulum system, and the myofilaments. Failure of neuromuscular transmission is a possible cause of fatigue. This failure could include depletion of the neurotransmitter and/or problems with the binding of the neurotransmitter to the receptors on the motor end plate. Fatigue could also result from a failure of the electrical excitation along the sarcolemma and T tubules and an inability of the sarcoplasmic reticulum (SR) to release sufficient calcium. Likewise, fatigue could result from any alteration in the ability of calcium to bind to troponin and thereby remove tropomyosin from its blocking position on actin. Fatigue might also result from biochemical and metabolic changes within the contractile

elements (myofilaments of the muscle cell).

Notice in **Figure 18.9** that fatigue can also be categorized in relation to electrophysiological or contractile biochemical considerations. Electrophysiological considerations involve steps within the CNS, the peripheral nervous system, or the muscle fiber leading up to the binding of the actin and myosin filaments (A.*M). These include the initial neural impulse from the motor cortex, transmission of the impulse across the synapse, propagation of the neural impulse along the motor neuron, transmission of the impulse across the NMJ, and subsequent spread of an action potential along the sarcolemma and into the T tubules. Contractile considerations involve the ability of actin and myosin to continue their cross-bridge attachment-detachment cycles, thus producing force. The reduced peak force with fatigue can be explained by both a decline in the force per cross-bridge and the number of cross-bridges that are formed ([Fitts, 2008](#); [Nocella et al., 2011](#)).

Two primary hypotheses attempt to explain the causes of fatigue within the contractile elements of the muscle: the depletion (or exhaustion) hypothesis and the accumulation hypothesis ([MacLaren et al., 1989](#); [Wan et al., 2017](#)). The depletion hypothesis suggests that fatigue results from decrease in certain metabolites, specifically, ATP, phosphocreatine (PC), and glycogen. Thus, the muscle fibers are no longer able to produce force. The accumulation hypothesis suggests that fatigue is caused by the accumulation of certain metabolites that are known to impair force generation within muscles, specifically, lactate, hydrogen ions, ammonia, and phosphate.

FOCUS ON APPLICATION

The Effects of Caffeine on Exercise and Sports Performance

Caffeine has been used for many centuries and is well known for increasing wakefulness. Caffeine is also used as an

ergogenic aid to enhance sport performance. Caffeine is readily absorbed through the gastrointestinal tract and appears in the blood stream 15–45 minutes after ingestion, with peak blood concentrations occurring approximately 1 hour after ingestion. The effectiveness of caffeine as an ergogenic aid varies with the type of activity performed.

Caffeine has a well-documented beneficial effect on endurance performance. It is acknowledged that caffeine's effect on endurance exercise is multifactorial, and there is evidence that caffeine has an effect on both the CNS and the excitation contraction coupling of skeletal muscle (Tarnopolsky, 2008). Caffeine also stimulates the delivery to and utilization of free fatty acids by the working muscles, thereby sparing glycogen. Improvements in sustained endurance events are apparent after low-to-moderate doses of caffeine (3–6 mg·kg⁻¹ BW).

More recently, the impact caffeine has on short-term, high-intensity performance, such as sprinting, team sports, and resistance training, has been studied with varying results. The discrepancies in published studies may be due to differences in testing protocols, the caffeine dose, training status, and habitual caffeine use. Astorino and Roberson reviewed the impact of acute caffeine use on short-term, high-intensity exercise and found that a majority of the studies reported an increase in performance for team sport exercise and power-related sports. However, the results were more pronounced in elite athletes who did not regularly use caffeine. Similarly, the benefit of caffeine ingestion was noted in the studies that employed resistance training. A study conducted by Beck et al. (2006) tested 37 resistance trained men (mean age 21 years) on the Wingate Anaerobic Test, a one-repetition max test, and a muscle endurance test after ingesting 2.5 mg·kg⁻¹ BW of caffeine or placebo. There was a 2.1% increase in the one-repetition max test for the caffeine group, while there was no change for the placebo group. There were no changes in the Wingate Anaerobic Test or the muscle endurance test for either group. In general, research suggests that moderate caffeine ingestion (4–6 mg·kg⁻¹ BW) is advantageous for short-term, high-intensity performance in trained athletes.

While research indicates a benefit of acute caffeine ingestion for various types of activity, the exact mechanism of action remains unknown. As with other ergogenic aids, it should be noted that it is rare that a single mechanism fully explains physiological effects of a supplement (Goldstein et al., 2010). It has been proposed that caffeine exerts its beneficial effects on sports performance through several potential mechanisms. Possible mechanisms include stimulation of the CNS and alteration of substrate utilization, β -endorphin release, and neuromuscular function. Astorino and Roberson (2010) have proposed a model to explain the impact caffeine has on the CNS and skeletal muscles to improve short-term, high-intensity performance. This model, presented in the accompanying figure, reinforces the fact that there are multiple ways that caffeine may affect both the CNS and the muscular system to improve performance and prevent fatigue. The model also notes areas where research evidence is more robust.

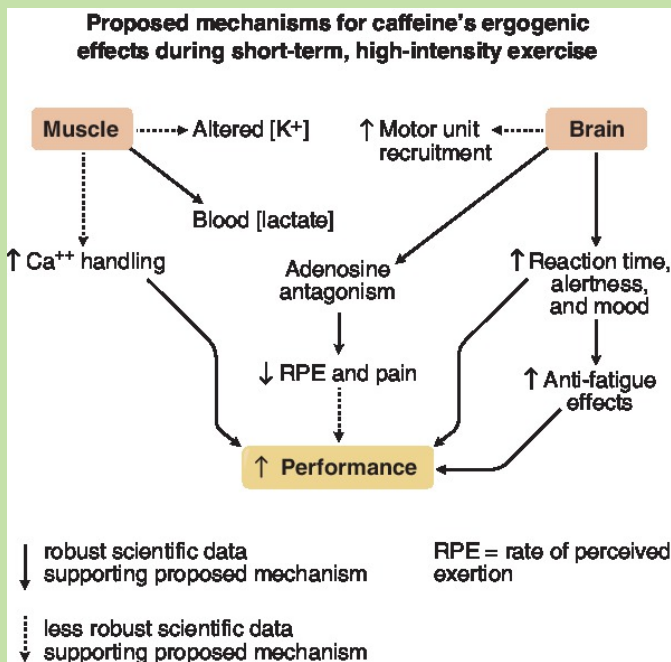
Given that caffeine supplementation can significantly enhance athletic performance, the International Olympic Committee (IOC) has mandated an upper limit on caffeine ($12 \mu\text{g}\cdot\text{mL}^{-1}$ in the urine). Caffeine ingestion of $9\text{--}13 \text{ mg}\cdot\text{kg}^{-1}$ BW (6–8 cups of brewed coffee) approximately 1 hour before testing would correspond to maximal urinary concentrations. The National Collegiate Athletic Association (NCAA) has set a value of greater than $15 \mu\text{g}\cdot\text{mL}^{-1}$ as illegal.

The International Society of Sports Nutrition (ISSN) has concluded that caffeine has an ergogenic affect that is specific to the condition of the athlete and the type of exercise being performed (Guest et al., 2021). Based on the available scientific literature, they have drawn the following conclusions:

1. Caffeine should be ingested 60 minutes prior to the onset of activity to ensure optimal absorption. The source of caffeine needs to be considered as caffeine gums, gels, and mouth rinses may require less than 60 minutes of wait time prior to exercise.
2. Caffeine should be ingested in low-to-moderate doses

($\sim 3\text{--}6 \text{ mg}\cdot\text{kg}^{-1} \text{ BW}$). There is no further benefit when consumed in higher amounts and higher amounts are related to higher incidence of side effects ($\geq 9 \text{ mg}\cdot\text{kg}^{-1} \text{ BW}$).

3. Caffeine has been shown to be an effective ergogenic aid in maximal endurance and time trial events.
4. Caffeine has been shown to be an effective ergogenic aid in prolonged, high-intensity team sports, such as soccer, rowing, or field hockey.
5. Caffeine is effective as an ergogenic aid in strength and power performance; however, the impact is likely very small and most likely advantageous for sports such as powerlifting and weightlifting ([Grgic, 2021](#))
6. Research has shown a positive benefit of caffeine on strength/power and performance in women.



Sources: [Astorino and Roberson \(2010\)](#); [Beck et al. \(2006\)](#); [Ganio et al. \(2009\)](#); [Guest et al. \(2021\)](#); [McLellan et al. \(2016\)](#); [Tarnopolsky \(2008\)](#).

Current research suggests that the primary site of fatigue, in the absence of disease, is within the contractile machinery of the muscle itself (Kent-Braun et al., 2012). The factors that contribute to fatigue are complex and interrelated and depend on the muscle fibers involved and the type of activity being performed. The depletion of PC leads to fatigue in FG fibers that rely on this substrate to regenerate ATP for explosive-type events. The depletion of glycogen leads to fatigue in SO fibers that rely on this substrate to produce ATP aerobically during prolonged activity. The accumulation of lactate and the associated H^+ may lead to fatigue by interfering with the contractile process in several places, decreasing the amount of calcium released, interfering with calcium-troponin binding, disrupting the Na^+-K^+ pump, inhibiting anaerobic glycolysis (by inhibiting the rate-limiting enzyme PFK), or interfering with cross-bridging. The detrimental effects of H^+ accumulation appear most evident in FOG fibers. But the role lactate and acidosis play in muscular fatigue is far from clear. Several studies suggest that lactate and acidosis during exercise are beneficial; acidosis may cause greater release of oxygen from hemoglobin for working muscle (Bohr effect) and enhance blood flow, while lactate released from working muscle may be taken up and utilized by other cells as a metabolic fuel (see Chapter 3). While the controversy about the role or roles of lactate remain to be resolved, what is certain is that lactate/ H^+ may contribute to fatigue but is not the sole cause of muscular fatigue (Bangsbo et al., 2006; Cairns, 2006; Grassi et al., 2015; Lamb et al., 2006).






Because many types of activity involve more than one fiber type and different recruitment patterns, several of the mechanisms described in this section may be involved in muscle fatigue for any given activity (Conley, 2000; Fitts, 2012).

Type of Activity and Muscle Fatigue

Fatigue results from different mechanisms in different types of activity. Furthermore, the fiber type involved in a given activity greatly influences the most probable mechanism of fatigue for that activity. **Table 18.3** summarizes the most probable causes of fatigue for different types of activity. Note that any given activity may have several possible causes and that the proposed

mechanisms often interact. Investigation in this area is ongoing.

TABLE 18.3 Most Probable Causes of Muscle Fatigue

| Type of Activity | Probable Causes of Fatigue |
|---|---|
| Long-term, moderate to heavy, submaximal aerobic  | Depletion of glycogen, low blood glucose Accumulation of H^+ Inhibits glycolysis Decreases Ca^{2+} release from SR Interferes with Ca^{2+} -troponin binding La^- or H^+ interferes with cross-bridging |
| Incremental aerobic exercise to maximum  | Depletion of glycogen Accumulation of H^+ Inhibits glycolysis Decreases Ca^{2+} release from SR Interferes with Ca^{2+} -troponin binding Depletion of PC La^- or H^+ interferes with cross-bridging |
| Static  | Depletion of PC Accumulation of H^+ Inhibits glycolysis Decreases Ca^{2+} release from SR Interferes with Ca^{2+} -troponin binding Occlusion of blood flow Inhibition of motor cortex via sensory fibers in muscle La^- or H^+ interferes with cross-bridging |
| Dynamic resistance | Depletion of PC stores |
| Low repetitions | Depletion of glycogen |
| High repetitions  | Accumulation of H^+ Inhibits glycolysis Decreases Ca^{2+} release from SR Interferes with Ca^{2+} -troponin binding La^- interferes with cross-bridging |
| Anaerobic (sprint)  | Depletion of PC stores Accumulation of H^+ Inhibits glycolysis Decreases Ca^{2+} release from SR Interferes with Ca^{2+} -troponin binding La^- or H^+ interferes with cross-bridging |

Long-Term, Moderate to Heavy, Submaximal

Aerobic Exercise

Steady-state activities that are performed at moderate workloads rely almost exclusively on SO fibers to produce ATP. As a result, lactate and H^+ levels do not increase substantially during these activities. The most probable cause of fatigue during steady-state activities is the depletion of glycogen stores, resulting in peripheral fatigue (Hargreaves, 2015; Hawley and Leckey, 2015). During prolonged activities performed at an intensity high enough to recruit FOG fibers, a significant amount of lactate and H^+ are formed. In this case, the high levels of H^+ likely contribute to fatigue (Conley, 2000).

Central fatigue is also thought to play a significant role in fatigue during prolonged activities. The “central fatigue” hypothesis suggests that an alteration in brain neurotransmitters (specifically the ratio of serotonin to dopamine) is associated with reduced motor unit recruitment and feelings of tiredness and lethargy during prolonged activity (Meeusen et al., 2006; Newsholme et al., 1987). Central fatigue may be especially important in prolonged activities in warm environments (Nybo and Nielsen, 2001).

Incremental Aerobic Exercise to Maximum

Incremental aerobic exercise to maximum involves recruiting all muscle fiber types in the order of SO, FOG, and FG corresponding to the increasing intensity of effort. All of the mechanisms described above likely play a role in fatigue during this type of exercise. Furthermore, the cardiovascular system may be unable to provide blood supply to the working muscle adequate to support aerobic energy production (Conley, 2000).

Static Exercise

When a person maintains a static contraction at a given intensity (expressed as a percentage of maximal voluntary contraction, %MVC), force decreases over time because of fatigue. **Figure 18.10** depicts the force a muscle can produce statically as a function of time. This figure suggests that if the individual sustains a static contraction (in this case, a handgrip exercise) at less than 20% MVC, the load can be maintained indefinitely.

However, as the %MVC increases, the time that the contraction can be maintained decreases rapidly. A force equal to 50% MVC can be maintained only for approximately 1 minute in a handgrip exercise. The precise shape of the fatigue curve varies among muscle groups, reflecting differences in fiber-type distribution and architectural design.

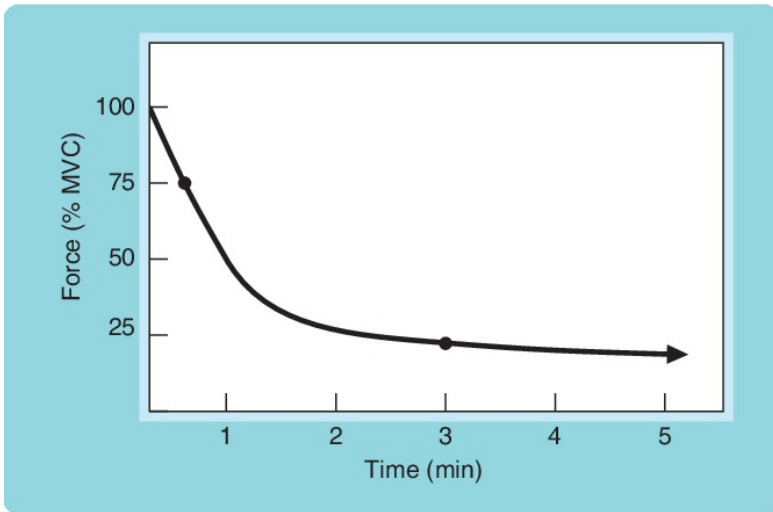


Figure 18.10 Hypothetical Fatigue Curve for Handgrip.

Fatigue likely results from several factors: depletion of PC in FG fibers (if the intensity of effort is great enough to recruit them), the accumulation of H^+ , and inhibition of the CNS by afferent fibers that are sensitive to the buildup of H^+ and other metabolites (Conley, 2000).

Dynamic Resistance Exercise

Dynamic resistance exercises that involve very few repetitions employ FG fibers. Fatigue is, therefore, likely due to a depletion of PC. In resistance exercise involving a high number of repetitions or a high total volume of work, fatigue is more likely caused by an accumulation of H^+ (and perhaps P_i and ADP) acting at the cross-bridges to reduce force, velocity, and power (Conley, 2000).

Very Short-Term, High-Intensity Anaerobic Exercise

When an individual performs a high-intensity exercise, the amount of force that can be developed decreases rapidly due to fatigue. **Figure 18.11** depicts the change in force production on a Wingate Anaerobic Test (described fully in [Chapter 3](#)). During this test, the participant pedaled as hard as possible for 30 seconds. Despite continued maximal effort, the force that could be developed decreased dramatically over this period.

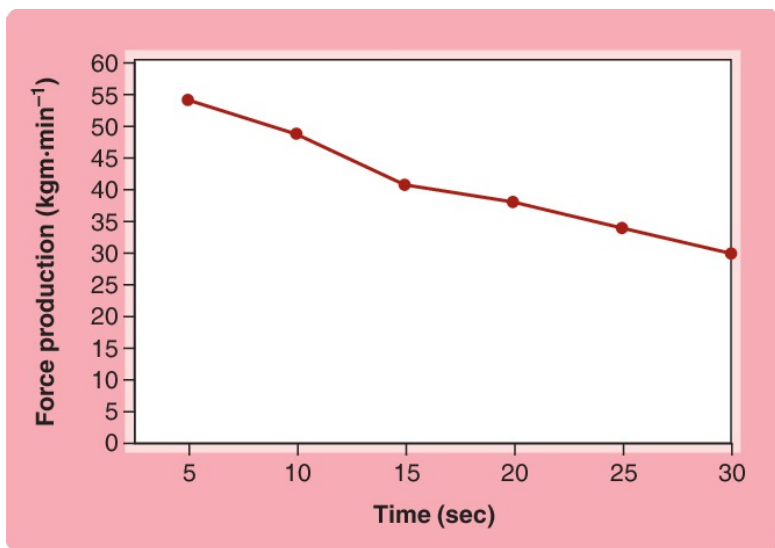


Figure 18.11 Force Decrement during Short-Term, High-Intensity Exercise (30 Seconds. WAT).

Very short-term, high-intensity anaerobic activities rely largely on FT fibers to produce ATP. Explosive power events rely more heavily on FG, whereas activities that continue 1–3 minutes rely more heavily on FOG fibers. During explosive-type activities involving a predominance of FG fibers, fatigue most likely results from the depletion of PC stores and the subsequent inability to regenerate ATP. During anaerobic activities that rely on anaerobic metabolism and use FOG fibers, the primary factor limiting performance appears to be the accumulation of H⁺

associated with increased lactate. The accumulation of H^+ interferes with the contractile process (see explanation above) and inhibits the production of ATP by anaerobic glycolysis (Conley, 2000). A recent study has shown that ingestion of sodium bicarbonate improves high-intensity intermittent exercise performance in trained young men (Krustrup et al., 2015). Because sodium bicarbonate increases the buffering capacity in the blood, this evidence supports that importance of increased $[H^+]$ (decreased pH) in fatigue during high-intensity intermittent exercise. However, other lines of research suggest that inorganic phosphate, produced because of the hydrolysis of phosphocreatine (PC), is a major cause of muscle fatigue (Westerblad et al., 2002).

Muscle Soreness

Muscle soreness is a familiar consequence of overexertion. The two generally recognized types of muscle soreness are immediate and delayed onset. *Immediate-onset soreness* is characterized by pain during and immediately after exercise, which may persist for several hours. This type of soreness is thought to be caused by stimulation of the pain receptors by metabolic by-products of cellular respiration, especially the H^+ associated with increased lactate levels. It is generally relieved by discontinuing exercise or subsides shortly thereafter.

Delayed-onset muscle soreness (DOMS) is characterized by muscle tenderness, pain on palpitation, and mechanical stiffness that appears approximately 8 hours after exercise, increases and peaks over the next 24–48 hours, and usually subsides within 96 hours (Byrne et al., 2004). Delayed-onset muscle soreness is generally considered an adult phenomenon. Children experience less muscle damage from excessive exercise than do adults and may recover more quickly (Rowland, 2005). The age in adolescence when this changes has not been determined. However, Lin et al., have shown that prepubescent females (9–10 years of age) experience less soreness and muscle damage compared to pubescent (14–15 years of age) or postpubescent females (20–24 years of age) after a bout of eccentric exercise (Lin et al., 2018). Additionally, the pubescent females also experienced less muscle damage and soreness than postpubescent

females. Therefore, it seems that muscle damage and muscle soreness are characteristic of aging and progress gradually with time.

Delayed-Onset Muscle Soreness (DOMS) It is a condition characterized by muscle tenderness, pain on palpitation, and mechanical stiffness that appears approximately 8 hours after exercise, increases and peaks over the next 24–48 hours, and usually subsides within 96 hours.

DOMS is of concern to exercise professionals because it affects athletic performance and exercise participation. Despite its importance and considerable research attention, the precise causative factors and cellular mechanisms of DOMS remain elusive. However, there is widespread agreement that muscle damage and inflammation play important roles in DOMS. Eccentric muscle action has been documented as the principle factor responsible for muscle damage (Byrne et al., 2004).

Etiology and Mechanisms

The model most likely to explain DOMS combines at least three theories: the mechanical trauma, muscle damage, and inflammation theories. The sequence of events in this integrated model is still hypothetical but is backed by considerable research; (Byrne et al., 2004; Cheung et al., 2003; Hotfiel et al., 2018):

1. Unaccustomed high-force activity, particularly during eccentric muscle contraction, causes disruption of structural proteins in muscle fibers, especially along the Z lines of the sarcomeres. At the same time, damage occurs to the connective tissue at the muscle-tendon junction.
2. Damage to the sarcolemma of the cell leads to an accumulation of calcium, which in turn inhibits ATP production and causes a disruption in calcium homeostasis. (Remember that ATP is needed to return calcium to its storage locations.) High calcium concentrations lead to further degradation of the Z discs, troponin, and tropomyosin

and ultimately tissue necrosis (death).

3. Structural damage initiates inflammation and activation of the immune system. Fluid moves into the muscle and causes swelling (edema).
4. The accumulation of by-products and debris from the cellular deaths and the immune system response in addition to increased pressure from the edema ultimately stimulate pain nerve endings, resulting in the sensations associated with DOMS.

FOCUS ON RESEARCH | *Clinically Relevant*

Effect of Glucose Supplementation on Fatigue

Glucose is an important substrate during muscular work, and decreased levels of blood glucose during prolonged activity have been linked to fatigue. Ingestion of carbohydrates during prolonged exercise may improve performance, whereas an insufficient supply of glucose may lead to hypoglycemia and fatigue. Fatigue is known to involve both central and peripheral factors. This study investigated the effects of glucose supplementation during 3 hours of cycling and subsequent force production during a 2-minute sustained maximal static contraction of the knee extensors. During the

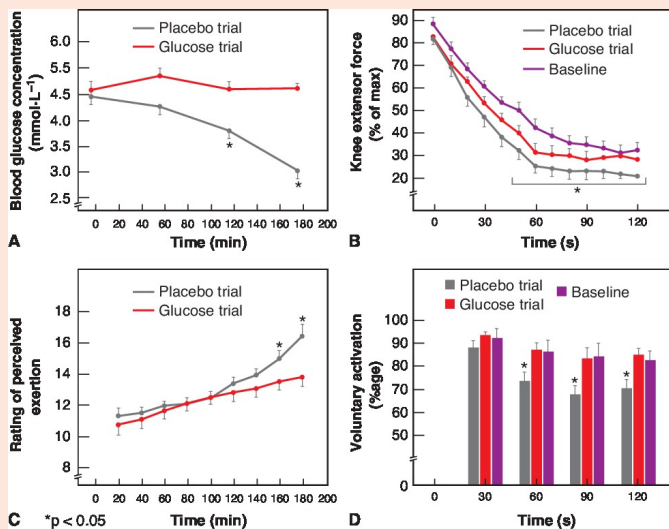
trial, subjects cycled at 60% of their $\dot{V}O_2 \text{ max}$. In the glucose trial, they ingested a 6% glucose drink, and in the placebo trial, they ingested an equal volume of a noncaloric placebo drink. A total of 200 g of carbohydrate was ingested in the glucose trial, and a total of 3.3 L of fluid was ingested in both trials.

During the maximal knee extension task, subjects were instructed to attempt to maintain maximal contraction throughout the 2-minute period. An electrical stimulation was superimposed every 30 seconds to assess the subjects' ability to voluntarily produce activation of the motor

neurons.

This study demonstrated that prolonged exercise was associated with decreased blood glucose levels and elevated RPE and that glucose supplementation was able to maintain blood glucose and lessen perceived exertion during the exercise bout. As expected, maximal force generation decreased during the 2 minutes of sustained maximal voluntary contraction in all conditions (baseline, after glucose ingestion, and after a placebo trial). At the onset of the maximal contraction, force was similar in the glucose and placebo trials. However, in the last minute of sustained contraction, the glucose ingestion trial subjects maintained a higher percentage of maximal voluntary force than did the placebo subjects. Furthermore, the glucose subjects achieved a higher voluntary activation percentage at 60, 90, and 120 seconds of contraction.

This study found that the development of hypoglycemia during prolonged exercise is associated with impaired neuromuscular performance, and the lower force generated during a subsequent sustained maximal contraction seems to be related to “central fatigue.” This suggests that hypoglycemia impairs the ability to maintain high neural drive to the muscles. The observed “central fatigue” seems to be alleviated by carbohydrate supplementation.



Source: Adapted with permission from Nybo, L.: CNS fatigue and prolonged exercise: Effect of glucose supplementation. *Medicine & Science in Sports & Exercise*. 35(4):589–594 (2003). Copyright ©2003 The American College of Sports Medicine.

Figure 18.12 summarizes the integrated model of DOMS. The sequence of events whereby muscle trauma leads to inflammation and activation of the immune system is described fully in [Chapter 22](#). In fact, the cytokine hypothesis of overtraining proposes that muscle damage, inflammation, and the immune response are central to explaining the symptoms of overtraining ([Smith, 2000](#)). In addition to the well-established theories that contribute to the integrated model of DOMS, researchers have also provided evidence that oxidative stress ([Ascensão et al., 2008](#)), the release of bradykinin ([Murase et al., 2010](#)), and restricted blood flow ([Umbel et al., 2009](#)) may play a role in DOMS.

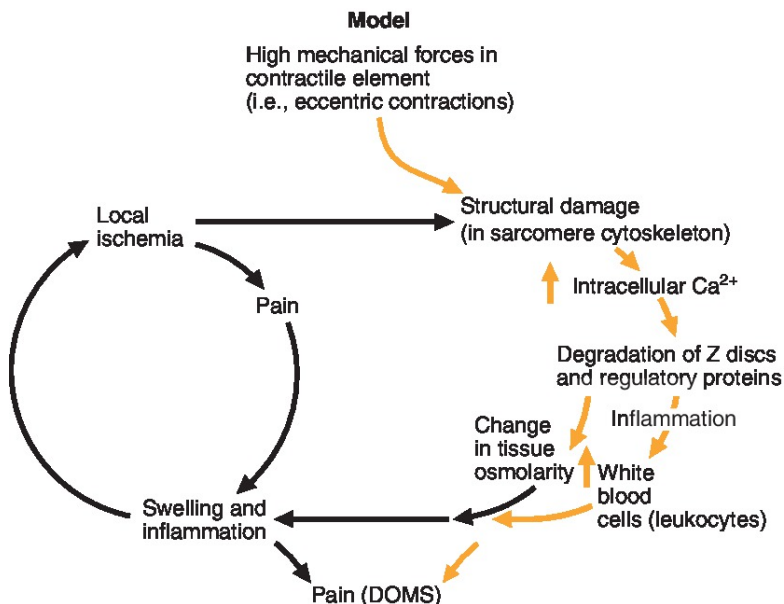


Figure 18.12 An Integrated Model to Explain Delayed-Onset Muscle Soreness (DOMS).

This integrated model of DOMS recognizes that mechanical trauma leads to muscle damage, resulting in an inflammatory response that causes swelling and pain.

One of the popular misconceptions about DOMS is that it is caused by the accumulation of lactic acid. Although this theory was originally proposed by researchers (Asmussen, 1956; Edington and Edgerton, 1976), it remains popular only with the lay public. Considerable research evidence now argues against this theory, including the fact that individuals with McArdle's syndrome, who do not produce lactic acid, also suffer from DOMS. Additional evidence against lactic acid as the cause of muscle soreness is that the type of activity that produces the greatest degree of soreness, eccentric contractions, produces lower lactic acid levels than concentric contractions of the same power output (Armstrong et al., 1983; Bonde-Petersen et al., 1972; Davies and Barnes, 1972). Perhaps the most compelling evidence is that lactic acid/lactate has a half-life of 15–25 minutes and is fully cleared from muscle within an hour (see

Chapter 3). Since lactic acid/lactate is not present at elevated levels 24–48 hours later, it cannot cause soreness then.

Effect on Muscle Function

While the sensation of soreness (DOMS) is important, many athletes are equally concerned with the functional impairments related to muscle damage. Exercise-induced muscle damage, especially with eccentric exercise, has a detrimental effect on muscle function. Muscle damage reduces strength, power, and performance. Unusual patterns of muscle recruitment during any given movement may include alteration in temporal sequencing of muscles. Altered joint mechanics and reduced joint range of motion have also been reported. These impairments are immediate but may persist for a prolonged period even though the athlete or exerciser may perceive no change. Reduced strength and power may lead to an individual working at a higher intensity than normal. Altered coordination or segment motion as well as reduced force output may lead to compensatory recruitment of muscles unused to such work. An inaccurate perception of impairment may lead to a premature return to high-intensity exercise (Cheung et al., 2003).

Evidence suggests that the time course of functional impairment is not the same as the time course of the sensation of DOMS (Byrne et al., 2004). The severity of muscle function impairment and time course of recovery greatly depend on the muscle group studied and the exercise protocol. An eccentric exercise protocol resulted in a 57% loss in strength immediately after exercise with strength measures remaining 33% below baseline after 5.5 days of recovery (Sayers and Clarkson, 2001). Performing the eccentric phase of a barbell squat exercise resulted in a 30–40% reduction in knee extensor strength immediately after exercise, which recovered to 95% of baseline strength by day 7 of recovery (Byrne and Eston, 2002). Marathon running has been shown to result in a 30% reduction in ankle extensor strength, which recovered fully 2 days after the race (Avela et al., 1999). A soccer match has also resulted in evidence of muscle damage and impaired function (Ascensão et al., 2008). Importantly, the time course of recovery varies substantially among individuals.

An athlete, coach, or clinician must be sensitive to individual differences when planning workouts after a stressful bout of exercise, particularly when it includes a large eccentric component, because recovery varies considerably. DOMS may otherwise lead to more substantial injury.

Treatment for Relief and Prevention

Several different treatment strategies have been researched to prevent DOMS or decrease the severity of the symptoms and restore function to the affected muscles as quickly as possible. Anti-inflammatory drug use has been suggested to prevent or treat muscle soreness, since inflammation is clearly part of the DOMS cycle. Although nonsteroidal anti-inflammatory drugs (NSAIDs) (such as aspirin, naproxen, and ibuprofen) do help relieve DOMS symptoms, their effect on muscle damage is less clear because muscle damage generates a local proinflammatory response, but the systemic response is tightly regulated by anti-inflammatory mediators (Peake et al., 2005). Current evidence suggests that NSAIDs may be beneficial for short-term recovery from muscle damage and soreness in healthy individuals (Cheung et al., 2003; Baldwin Lanier, 2003). Research has also found that branched-chain amino acid supplementation administered before heavy resistance exercise can lessen the functional impairment associated with DOMS (Shimomura et al., 2010).

Acute exercise also diminishes DOMS (Tufano et al., 2012). Indeed, exercise is the most effective technique for decreasing the pain of DOMS. Unfortunately, this analgesic effect ends quickly when the activity is stopped. Compression of the sore area may be of some value. Two meta-analyses with an overlap of only one research study have both shown that the wearing of compression garments during recovery from exercise (but not necessarily during exercise) is beneficial for reducing the severity of DOMS. The effects of compression garments during recovery probably results from an improvement in venous return and waste clearance from blood (Beliard et al., 2015; Hill et al., 2014). Massage has shown varying results that warrant further research. Research is needed to determine whether manual manipulation of injured tissue enhances healing or delays it. Static stretching is thought to initiate the inverse myotatic reflex (by stimulation of

the Golgi tendon organs, GTO), which results in relaxation of the stretched muscle (DeVries and Housh, 1994). (Reflexes are discussed in detail in Chapter 20.) However, static stretching does not help reduce or prevent muscle soreness (Herbert et al., 2011). Icing, ultrasound, and electrical current modalities have not been shown to consistently eliminate DOMS symptoms (Cheung et al., 2003) although there is evidence that cold water immersion or contrast water therapy may be of benefit (Vaile et al., 2008). There is no apparent relationship between ratings of muscle soreness and protein ingestion whether consumed prior to, during, or after an acute bout of endurance or resistance exercise. However, reduced muscle soreness does appear to be a benefit when supplemental protein is consumed after daily training sessions (Pasiakos et al., 2014).

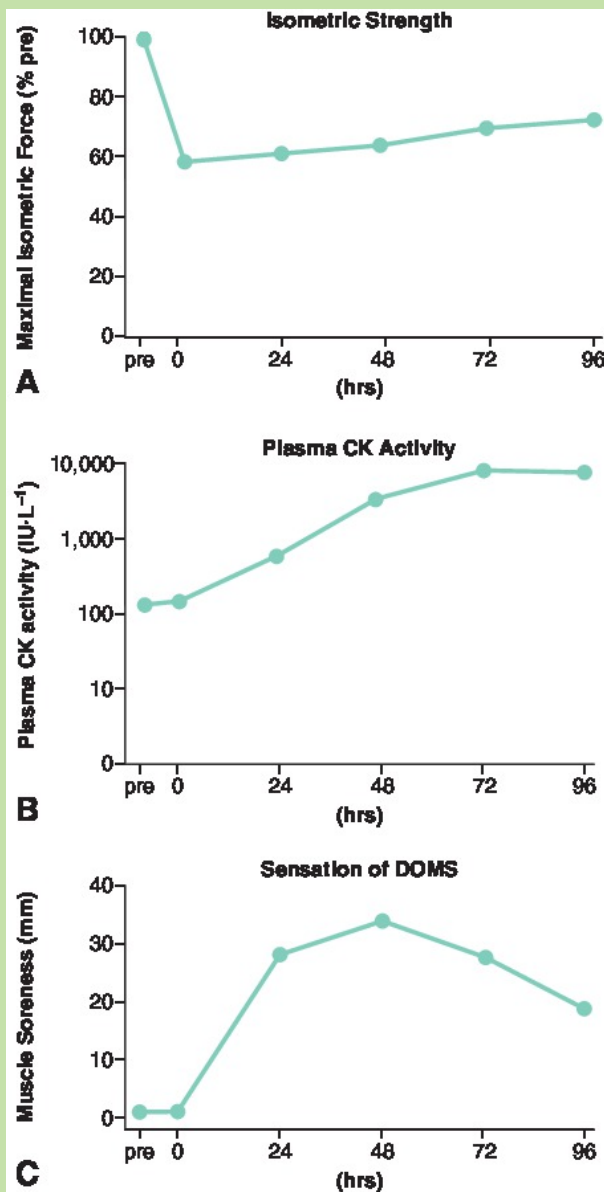
FOCUS ON APPLICATION | *Clinically Relevant*

Muscle Damage and DOMS

While it seems clear that exercise-induced muscle damage is related to the sensation of DOMS, the timelines of recovery from muscle damage and DOMS may be very different. Exercise-induced muscle damage (within the contractile elements, the sarcolemma, and the cytoskeleton) leads to inflammation that is believed responsible for the sensation of pain, tenderness, and stiffness that characterize DOMS and result in impairment of muscle function. In many studies, the sensation of DOMS is used as the primary marker of muscle damage. However, several studies have shown that measures of muscle function do not correspond with the sensation of DOMS. The accompanying figures report changes in maximal isometric force production, plasma levels of CK (representing muscle damage), and sensation of DOMS for 96 hours following 12 maximal eccentric contractions of the elbow flexors.

It can be seen clearly in these graphs that muscle function (strength) was impaired immediately after exercise without

an immediate change in plasma CK level or an immediate sensation of pain (measured as a visual analog scale from 0 to 50 mm). On the other hand, by 96 hours after exercise, the sensation of pain was beginning to subside, while the measure of muscle damage remained at its highest level. The lack of a temporal association among DOMS, muscle damage, and muscle function has important implications for athletes and fitness participants alike. Eccentric exercise can cause muscle damage and impairment of muscle function that last far longer than does the sensation of pain or discomfort associated with DOMS. The fact that muscular pain recedes does not guarantee that muscle repair is complete or that function is restored. Thus, it seems prudent to avoid another bout of strenuous eccentric work as soon as pain is no longer a limiting factor. In fact, a hasty return to strenuous work—without adequate recovery time—may be an important factor in overtraining.



Sources: Reprinted with permission from Nosaka, K., M. Newton, & P. Sacco: Muscle damage and soreness after endurance exercise of the elbow flexors. *Medicine & Science in Sports & Exercise*. 34(6):920–927 (2002). Copyright ©2002 The American College of Sports Medicine; Byrne et al.

(2004); Warren et al. (1999).

Complete the [Check Your Comprehension 1—Case Study 1](#) to apply your knowledge of muscle soreness.

CHECK YOUR COMPREHENSION 1-CASE STUDY 1

Claire and Louise are relatively active middle-aged women who were doing a 1.6-mile hike (3.2-mile round trip) that they had done several times. After a little friendly banter about their fitness levels, the women decided to see how fast they could get up and then back down the mountain. They made it up the mountain trail in record time by running whenever it was safe to do so. They had some water at the top and then descended as quickly as possible, running most of the way. They were more tired than usual that evening but generally felt very good. However, the next afternoon, they both noticed that they were very sore. By the evening of the next day, and all during the following day, their quadriceps were incredibly sore to the touch, and it was painful to sit from a standing position. What explains this level of soreness following the “hike”? What could they do to help relieve the pain?

Check your answers in Appendix C.

Repeated Bout Effect

As discussed previously, a single bout of unaccustomed, predominately eccentric exercise causes DOMS, symptoms of muscle damage, and a loss of muscle function. However, it is well established that a repeated bout of similar eccentric exercise results in markedly reduced symptoms of damage compared to the initial bout of exercise. This protective adaptation to a single bout of exercise is referred to as the *repeated bout effect* ([McHugh, 2003](#); [McHugh et al., 1999](#); [Nosaka and Clarkson, 1995](#)). The protective adaptation has been shown to last for several weeks and possibly up to 6 months when the initial bout of eccentric exercise was performed at near maximal intensity. While the

existence of the repeated bout effect is well known, and the conditions necessary to induce it are fairly well characterized, the mechanisms of its protective effect are not well understood (McHugh, 2003). Research suggests that decreased inflammation signaling and increased infiltration of immune cells into muscle may influence the extent of the repeated bout effect (Deyhle et al., 2016; Xin et al., 2014).

Measurement of Muscular Function

Accurate measurement of the strength, endurance, and power a muscle can generate is important for five reasons:

1. Measurement aids screening to determine the extent of muscle weaknesses and/or imbalances. Both weakness and imbalance can make an individual more prone to injury. Measurement also establishes baseline values for an individual.
2. Measurement can be used as a guide to rehabilitation. Measurement of the loss of function after an injury or accident is needed to determine rehabilitation workloads and to monitor progress.
3. Measurement is necessary for exercise prescription. Both athletes and fitness participants need realistic programs based on their own performance capabilities.
4. Measurement aids selection of the best exercises for working on specific problems.
5. Measurement is a research tool for studying which types of training programs produce the greatest changes in muscle function.

Strength is the ability of a muscle or muscle group to exert force against a resistance. It is usually measured as one maximal effort. For dynamic resistance exercise, this is often called a one-repetition maximum (1-RM), whereas for static exercise, it is referred to as a maximal voluntary contraction (MVC). Torque is the more correct term when movement is made through a range of motion, but strength is the more commonly used term.

Muscular endurance is the ability of a muscle or muscle group to repeatedly exert force against a resistance; the activity is typically performed at a given percentage of the 1-RM (for resistance exercise) or MVC (for static exercise). For example, an exerciser wishing to improve muscle endurance might lift a weight equal to 60% of their 1-RM for 12 repetitions. Similarly, a researcher studying static muscle endurance may ask an individual to hold a handgrip dynamometer at 50% of MVC for 90 seconds. **Power** is the amount of work done per unit of time and is the product of force and velocity ($P = F \times V$). Thus, power is the expression of strength exerted quickly.

Strength The ability of a muscle or muscle group to exert maximal force against a resistance in a single repetition.

Muscular Endurance The ability of a muscle or muscle group to repeatedly exert force against a resistance.

Power The amount of work done per unit of time; the product of force and velocity; the ability to exert force quickly.

Strength and muscular endurance can be expressed as either absolute or relative values. Absolute values refer to the actual external load commonly measured in pounds (lb), kilograms (kg), newtons (N), or newton-meters (N·m). Relative values are expressed in relation to body weight. Both values are useful. For example, if two individuals are doing a two-arm curl and individual A can lift 70 kg and individual B can lift only 60 kg, it might seem that individual A is stronger. However, if individual A weighs 55 kg and individual B weighs 47 kg, then both are lifting 1.27 kg per kg of body weight—and on a relative basis, neither is considered stronger. When a comparison is made between individuals, a relative expression of strength is generally preferred. In contrast, absolute values are preferred for

comparisons made of the same person under different conditions or at different times, for example, before and after a training program.

In terms of muscular endurance, if individual A has a handgrip strength of 70 kg and individual B has a handgrip strength of 60 kg, and both are asked to hold a handgrip dynamometer at 40 kg, you would assume that individual A could do so for a longer period of time, because that load represents 57% MVC ($40/70$) for individual A and 67% MVC ($40/60$) for individual B. An absolute load thus puts the weaker individual at a disadvantage. However, if both are asked to hold a load representing a 50% MVC (35 kg for individual A, 30 kg for individual B), you would expect that both individuals could hold that load for the same length of time, assuming equal motivation. To confirm your understanding of the influence of body weight on the expression of strength, complete the [Check Your Comprehension 2](#) box.

CHECK YOUR COMPREHENSION 2

Listed below are the body weight (BW) and MVC for a handgrip task.

| Name | Gender | BW (kg) | Absolute Strength MVC (kg) | Relative Strength $\text{kg}\cdot\text{kg}^{-1}$ BW | 50% MVC |
|-------|--------|---------|----------------------------|---|---------|
| Jody | F | 60.0 | 40.0 | | |
| Jill | F | 68.0 | 60.0 | | |
| Pat | F | 70.0 | 36.0 | | |
| Scott | M | 63.0 | 50.0 | | |
| Tom | M | 82.0 | 72.0 | | |
| Mike | M | 70.0 | 71.0 | | |

1. Calculate the relative strength for each individual (absolute strength \div BW).

2. Calculate the load that would need to be held for each of the subjects to be working at 50% MVC (absolute strength $\times 0.5$).
3. Who is the strongest person on an absolute basis? On a relative basis?
4. For whom would 50% MVC represent the most absolute force?

Check your answers in Appendix C.

Laboratory Methods

To quantify the several aspects of muscular function, exercise physiologists use various laboratory and field methods. Laboratory methods are generally more accurate and precise for measuring different aspects of muscular function but are more expensive and inaccessible to many people.

Electromyography

Electromyography (EMG) is the measurement of the electrical activity, known as muscle action potentials, that brings about muscle contraction. Motor unit activity can be recorded using needle electrodes, or whole-muscle activity can be recorded using surface electrodes. The EMG voltage is proportional to the force of a static contraction, to the tension developed in constant-velocity contractions ([Lippold, 1952](#)), and to velocity in dynamic contractions ([Bigland and Lippold, 1954](#)). Therefore, one can obtain a reasonably valid estimation of tension development in a muscle by EMG.

Electromyography (EMG) The measurement of the neural or electrical activity that brings about muscle contraction.

EMG provides not an absolute value of force or torque but a direct functional indication of muscle activity. If it is plotted against incremental loads or values resulting from a submaximal

hold, it can be used to predict strength or endurance, respectively. Such submaximal testing using EMG is particularly important in situations where an individual either cannot or is not motivated to perform maximally. In addition, because weak individuals produce a greater EMG signal than do strong individuals for any absolute load (Fischer and Merhautova, 1961), EMG activity can be used to monitor rehabilitation or training progress.

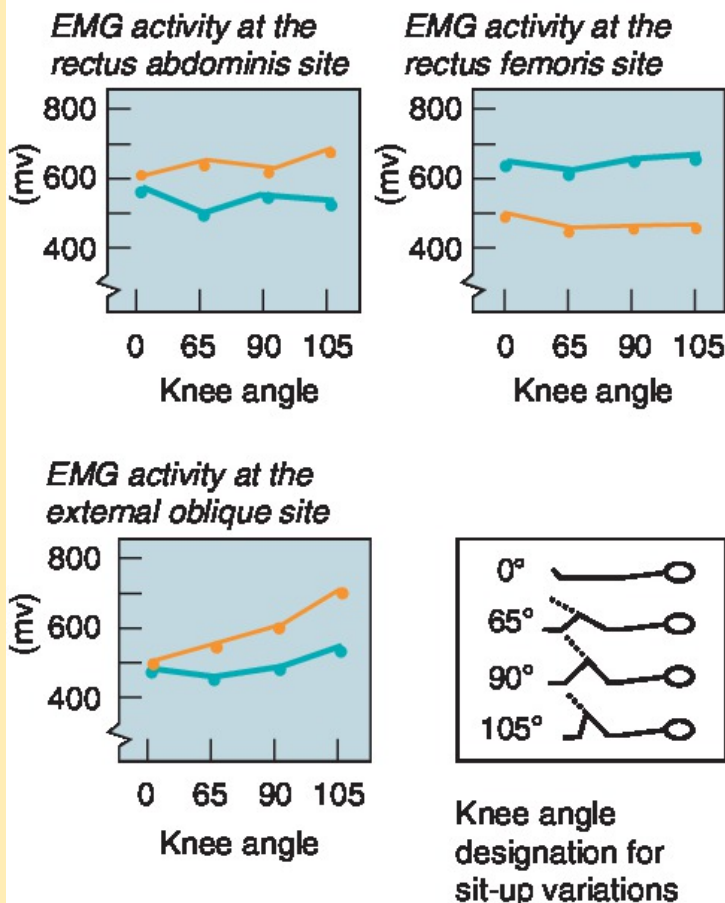
Perhaps the most valuable role of EMG in muscle function assessment is showing which muscles are primarily responsible for specific actions. For example, when training or testing individuals for abdominal strength, an exercise should be selected that maximizes the involvement of the abdominal muscles (the rectus abdominis and the external obliques) and that minimizes the involvement of the hip flexors and thigh muscles (rectus femoris). EMG helps in this selection.

The Check Your Comprehension 3 box provides an example of the EMG activity of these muscles during a sit-up with the feet supported and unsupported and the knees at various angles (Hall et al., 1990; Halpern and Bleck, 1979). Work through the questions in the box to confirm your understanding.

CHECK YOUR COMPREHENSION 3

Study the EMG data for each of the muscle groups with the feet supported (•) and with the feet unsupported (◦), and answer the following questions:

● Feet supported ● Feet unsupported



1. Does bending the knees eliminate the involvement of thigh muscles (rectus femoris) if the feet are held down? Which muscles are more active if the feet are held—the abdominal or thigh muscles?
2. Which muscle group does the angle of knee bend affect most?
3. Should the feet be held or not held to maximize the involvement of the abdominal muscles?
4. On the basis of your answers in 1–3, which sit-up form (feet held or not held) and which knee angle would you

recommend?

Check your answers in Appendix C.

Isokinetic Machines

Isokinetic machines, such as a HUMAC/Cybex or Biodex (**Figure 18.13**), allow the velocity of limb movement to be kept nearly constant throughout a contraction. These devices provide accurate and reliable measurements of muscular strength, muscular endurance, and power while the speed of the limb is kept constant at a predetermined velocity. Any increase in muscular force results in increased resistance rather than increased acceleration of the limb (Heyward, 1991). Measurements obtained on isokinetic machines serve as a reference against which other methods of measurement can be compared. These devices can be configured to test the limbs of the upper or the lower body.



Figure 18.13 Isokinetic Exercise Equipment.

The measurement of isokinetic strength of the quadriceps is recorded throughout the normal range of motion.

Force Transducers

Force transducers measure static strength and endurance. The deformation of the transducer sends an electrical signal to a computer, which displays the force output. When the subject exerts as much force as possible in a single trial, this device measures maximal strength. It can also measure static muscular endurance when the subject holds a given percentage of the predetermined maximal value (%MVC). Static muscular endurance is then calculated by determining either how long the individual can maintain the predetermined value or how long it takes the individual to drop to a specified percentage of the maximal contraction.

Laboratory and Field Methods

Several other methods of measuring muscle function require only relatively simple testing devices. They may be used in a laboratory or field test.

Dynamometers

Dynamometers measure static strength and static muscular endurance. Two commercially available dynamometers are the handgrip dynamometer and the back and leg dynamometer. As force is applied to the dynamometer, its spring is compressed and moves the needle to indicate force produced. Like the force transducer, these devices can be used to measure maximal strength when the subject exerts as much force as possible in a single MVC. Because of its availability and ease of administration, the handgrip dynamometer has been extensively used to measure grip strength for all ages. However, it is currently of particular interest as a fitness battery test item for children and adolescents.

Handgrip strength has been shown to be a valid test to assess muscular strength in youth whether evaluated against isometric strength ([Castro-Piñero et al., 2010a](#)), 1-RM dynamic strength ([Milliken et al., 2008](#)), or isokinetic strength ([Artero et al., 2012](#)).

Handgrip is currently an item in the ALPHA (Assessing Levels of Physical Activity) (Ruiz et al., 2011) and EUROFIT (Eurofit, 1993) health-related fitness test batteries for children and adolescents in Europe, and the CSEP-PATH (Canadian Society for Exercise Physiology–Physical Activity Training for Health, 2019) fitness assessment in Canada. It has been recommended for inclusion in any future surveys of physical fitness in the United States (Institute of Medicine, 2012).

Dynamometers can also be used to measure static muscular endurance when the subject performs at a given percentage of the predetermined maximal value. Muscular endurance is then calculated by determining how long the individual can maintain the predetermined (submaximal) value.

Constant Resistance Equipment

The most common method of measuring dynamic strength is to determine the maximal amount an individual can lift in a single repetition against constant resistance using free weight or weight machines. Known as a one-repetition max (1-RM), this is a trial-and-error method of determining how much an individual can lift. If the selected weight is too heavy to be lifted, a lower weight is used. If the weight is successfully lifted, then additional weight is added. Care must be taken because too many trials will cause fatigue, thereby decreasing the true maximal strength value.

Constant resistance equipment can also be used to measure dynamic muscular endurance by determining how many times an individual can lift a submaximal load. The submaximal load is usually a predetermined load (such as the 80-lb bench press test for males or the 35-lb test for females used in the YMCA assessment battery [Golding, 2000](#)), or it may be expressed as a percentage of body weight or as a percentage of 1-RM.

Field Tests

Several easily administered tests are commonly used in the field to measure muscle function. Common field tests include calisthenic and jumping activities.

Calisthenic Activities

Calisthenic activities are often used in field settings to measure muscular endurance and, to a lesser extent, muscular strength. The most common tests include some version of sit-ups or curl-ups, push-ups, and pull-ups or flexed-arm hang. Although these tests are often reported to measure strength, they are in fact usually endurance tests; that is, they measure the maximum number of times an individual can perform a given test (often within a specified time period, such as the number of curl-ups per minute). If the participant can complete more than one repetition, the result represents muscular endurance. If the subject can complete only one repetition (or sometimes no repetitions), the result represents strength (or lack thereof). Note that these tests indicate relative strength or endurance, because the amount of resistance depends on the individual's body size and mass.

Vertical Jump/Standing Broad Jump

Jump tests are used to measure the explosive muscular power of the legs. Although they are easy to administer, they are influenced by the individual's weight. Furthermore, these tests may have a strong neural component and have a definite alactic anaerobic metabolic component (see [Chapter 3](#)). As with handgrip, there is currently considerable interest in these lower body tests of explosive power in children and adolescence as health-related physical fitness test items.

Several studies have attempted to validate these two tests. [Milliken et al. \(2008\)](#) found that both jumps related well to 1-RM strength in children. [Castro-Piñero et al. \(2010b\)](#) determined that the standing long jump was strongly enough related to the vertical jump and other lower- and upper-body explosive muscular tests to be considered a general index of muscular fitness in youth. [Artero et al. \(2012\)](#) concluded that the standing long jump was valid when compared to isokinetic strength. The standing long jump is currently an item in most fitness batteries for children and youth ([Marques et al., 2021](#)) including both the ALPHA ([Ruiz et al., 2011](#)) and EUROFIT ([Eurofit, 1993](#)) health-related fitness test batteries in Europe. The CSEP-PATH battery

(Canadian Society for Exercise Physiology–Physical Activity Training for Health, 2019) uses the vertical jump. The standing long jump has been recommended for inclusion in any future surveys of physical fitness in the United States (Institute of Medicine, 2012).

The Influence of Age and Sex on Muscle Function

Male-Female Differences

Adult females on average have about 56% of the static strength values of adult males in upper-body locations, about 64% in trunk strength, and about 72% in lower-body locations (Lauback, 1976). As shown in **Figure 18.14**, the variation between upper- and lower-body strength occurs also in the bench press and the leg press (Wells and Plowman, 1983). Note the considerable overlap in the distribution of leg strength even in untrained males and females (**Figure 18.14A**), whereas virtually no overlap exists in arm strength (bench press) distributions (**Figure 18.14B**).

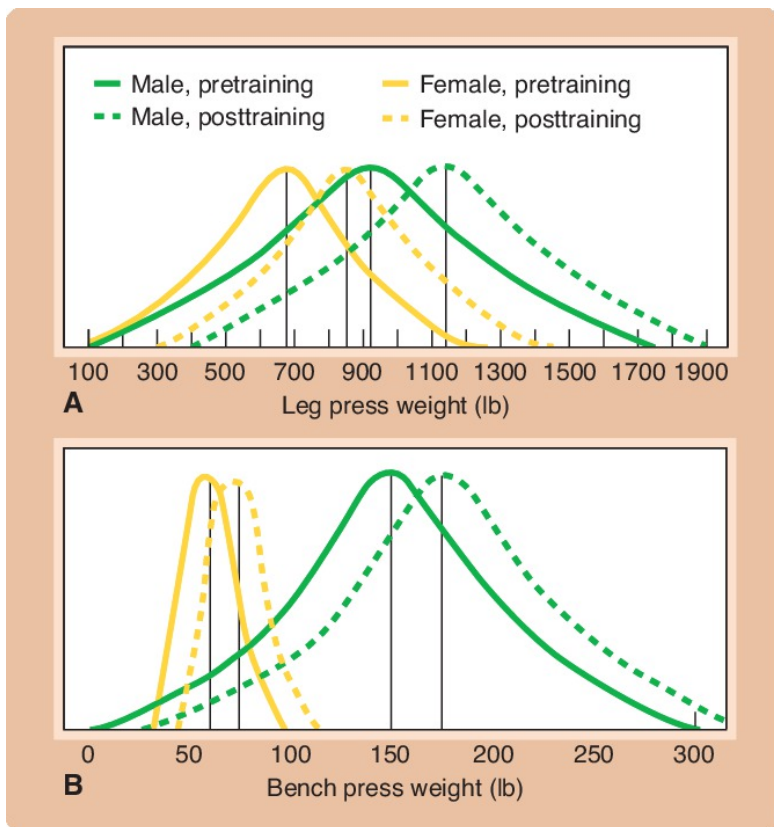
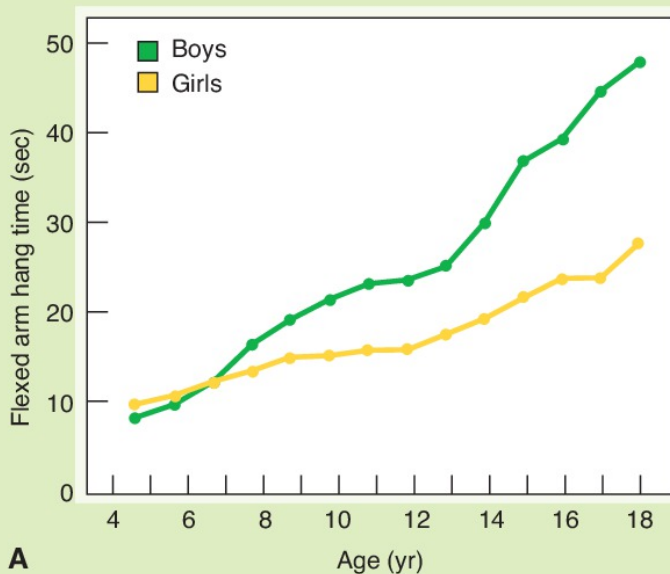


Figure 18.14.

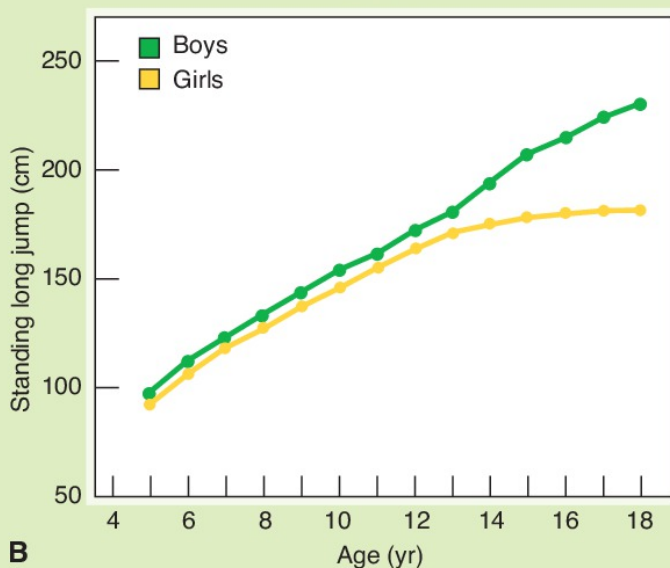
Frequency distribution for leg press (A) and bench press (B) values for males and females before and after a training program. **Source:** From Wells, C. L., & S. A. Plowman: Sexual differences in athletic performance: Biological or behavioral? *Physician and Sports Medicine*. 11(8):52–63 (1983). Reprinted by permission of Taylor & Francis Ltd., <https://www.tandfonline.com>.

Although field tests are not pure tests of muscle function, differences in these tests parallel those seen in strength with growth and maturation and between boys and girls. **Figure 18.15A and B** illustrates these relationships for the flexed-arm hang and the standing long jump, respectively. Adult performances also appear to parallel adolescent male-female

strength differences. **Figure 18.16A** shows that throughout the adult age span, both inactive and active males can perform more push-ups than active or inactive females. With a leg-lift exercise (**Figure 18.16B**), however, the performance gap is smaller, with considerable overlap between inactive males and active females.



A



B

Figure 18.15 Performance on Field Tests of Muscular Function in Children.

A. Flexed-arm hang (muscular endurance). **B.** Standing long jump (muscular power). **Source:** Reprinted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth*,

Maturation and Physical Activity (2nd ed.). Champaign, IL: Human Kinetics (2004); Data from Michigan State University Motor Performance Study.

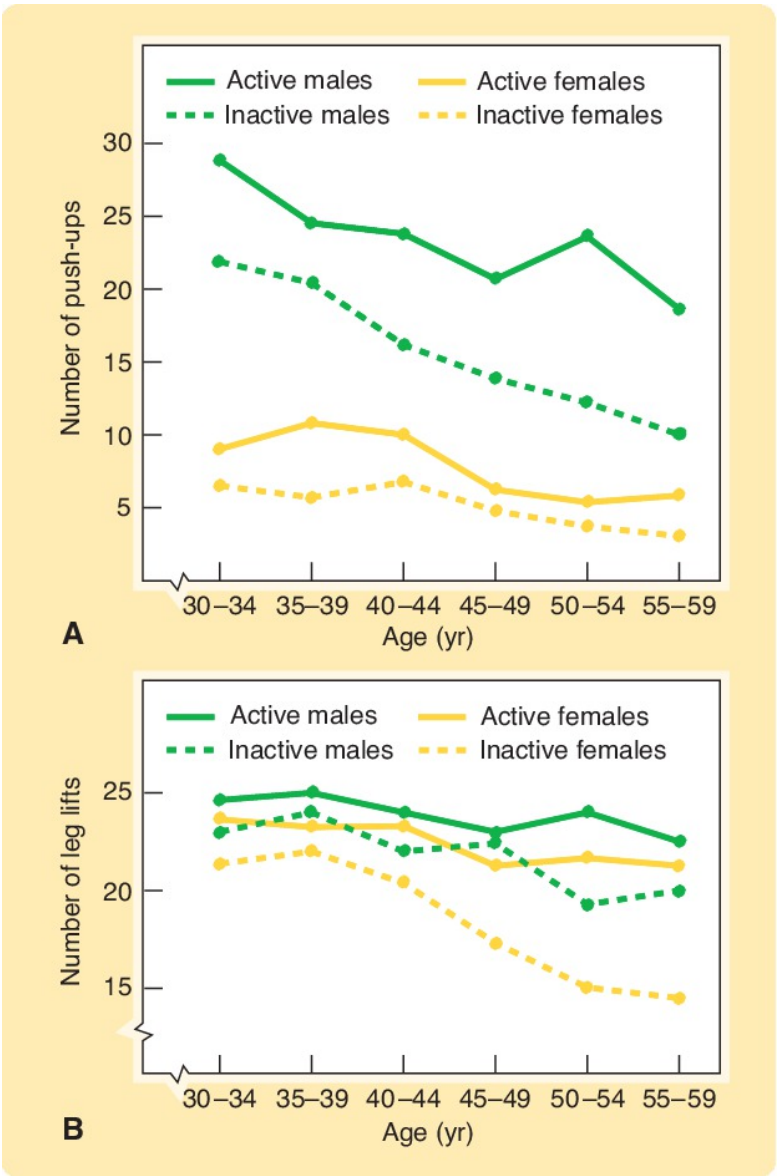


Figure 18.16 Performance on Field Tests of Muscular

Function in Adults.

A. Push-up (muscular endurance). **B.** Leg lifts. **Source:** Republished with permission of John Wiley & Sons from Israel, S.: Age-related changes in strength and special groups. In Komi, P. V. (ed.): *Strength and Power in Sport*. Champaign, IL: Human Kinetics, 323 (1992).

Why do these differences occur? Strength and other muscle functions increase as children grow because muscle mass increases in parallel with increases in body mass. Pubertal hormonal changes—particularly in testosterone, which is involved with the anabolic process of muscle growth—favor males. While young males are adding muscle mass under the influence of testosterone, young females are adding fat under the influence of estrogen (**Figure 18.17**) ([Malina and Bouchard, 2004](#)). The similarities among the graphic representations of fat-free mass (**Figure 18.17A**), strength (**Figure 18.18**), and muscle performance (**Figure 18.15**) are striking, leading to the conclusion that the quantity of muscle mass is what accounts for the difference in the expression of strength.

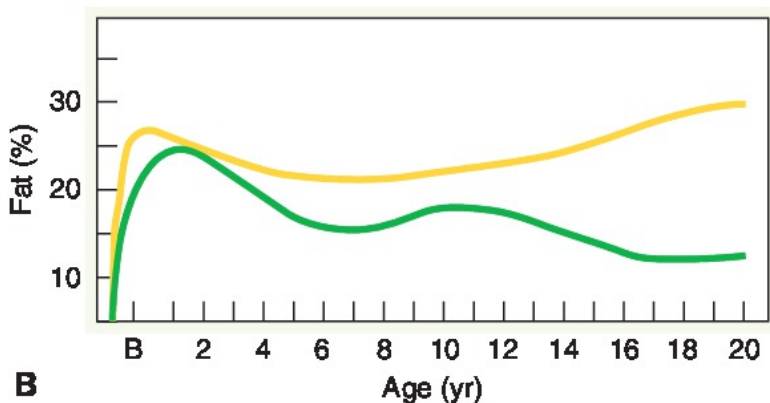
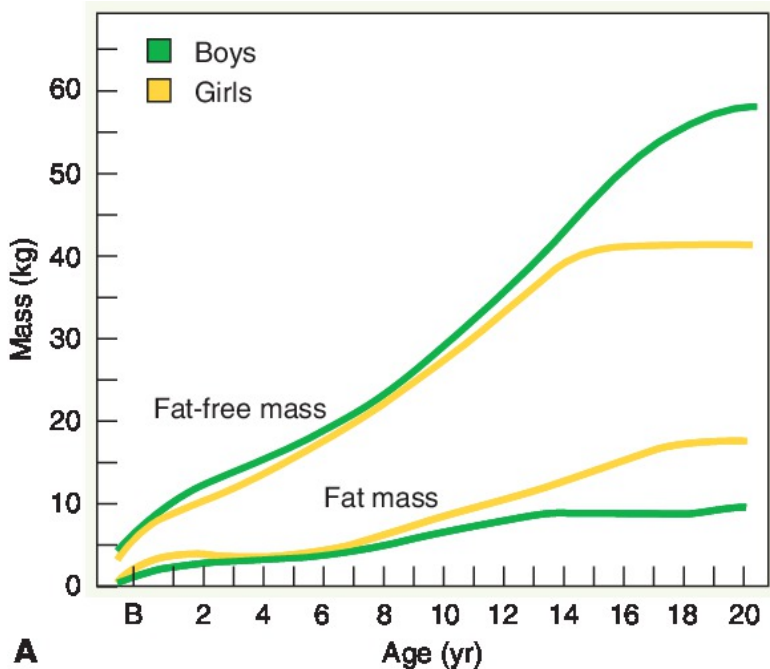


Figure 18.17 Growth Curves for Mass and Percent Fat in Children.

A. Mass. B. Fat. Source: Reprinted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth, Maturation and Physical Activity* (2nd ed.). Champaign, IL: Human Kinetics (2004); Growth curves for Fat (B): Data from Malina (1989) and Malina et al. (1988).

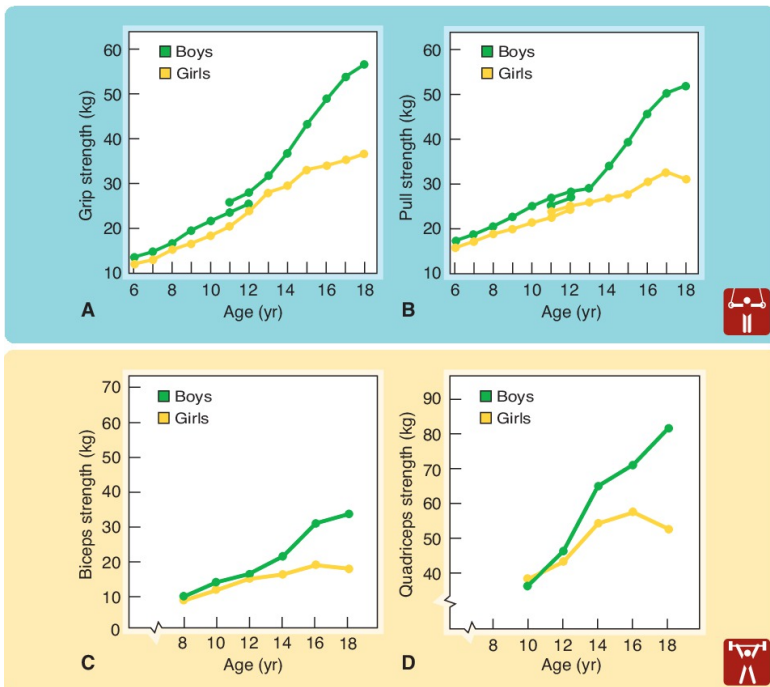


Figure 18.18 Strength Development in Boys and Girls.

A. Grip strength in young children. **B.** Pull strength in children 6–18 years old. **C.** Biceps strength. **D.** Quadriceps strength. **Source:** Reprinted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth, Maturation and Physical Activity* (2nd ed.). Champaign, IL: Human Kinetics (2004); Growth curves for Fat (B): Data from Malina (1989) and Malina et al. (1988).

That it is muscle quantity rather than quality that is responsible for these male-female inequities is emphasized by calculating relative strength values (Wells and Plowman, 1983; Wilmore, 1974). In one study, the percentage of handgrip strength exhibited by the females in relation to the males increased from 57% in absolute strength terms to relative values of 73% (absolute strength divided by total body weight) and 83% (absolute strength divided by lean body mass) (Wilmore, 1974). For the bench press, the corresponding figures were 37%, 46%, and 53%, and for the leg press, they were 73%, 92%, and 106%.

Thus, in leg strength relative to lean body mass, female performance actually exceeded that of males.

In addition, studies relating strength to cross-sectional area of muscle show no inequities between the sexes. Overall then, all these studies show that the larger size of males in general, their greater muscle mass, and their larger fiber size are physiologically responsible for their greater strength, rather than any inherent difference in the potential or function of the muscle fibers per se.

Another important factor is cultural expectations. Many adolescent girls become less active as they grow up, detraining to some extent. Some detraining may be anatomically selective. Although males and females of all ages experience gravity equally when walking, climbing stairs, sitting down, and standing up, individuals can selectively avoid upper-body activities, such as lifting heavy loads, opening jars, hammering, and weight lifting. Thus, differences in upper-body versus lower-body activities in males and females may in part be cultural. Future studies may note a change in these relationships as more girls have the opportunity to enter sports at an early age and continue to be active into middle age and old age.

Children and Adolescents

Strength generally develops in humans from infancy through maturity. **Figure 18.18** shows a common pattern of development. Strength increases rectilinearly from early childhood (3–7 years) through early adolescence (13–14 years) for both sexes. A marked increase in strength then occurs during the rest of adolescence and into early adulthood (15–20 years) for boys. Girls, however, do not have an accelerated increase in strength in late adolescence. They either maintain a slow rectilinear rise, as shown for grip strength (**Figure 18.18B**), or decline after age 16, as shown for both elbow flexion and knee extension (**Figure 18.18C and D**) (Malina and Bouchard, 2004). The increase in strength during childhood and adolescence, even without training, is more than can be accounted for just from growth in size, although other causative factors have not been determined. In males, the average muscle mass increases from 42% at age 5 to

53% at age 17 years of total body mass. In females, comparable values are 41% at age 5 and 42% at age 17 (Rowland, 2005). This growth occurs by increasing protein content in the existing muscle fibers, not by increasing the number of muscle fibers.

In early childhood, there is virtually no difference in strength measurements between boys and girls. As puberty begins and progresses though, a gap appears and progressively widens. On the average, at 11–12 years of age, girls have approximately 90% of boys' strength; at 13–14 years, this percentage has decreased to 80% and at 15–16 years, to 75%. These percentages vary not only with age but also with the muscle group being measured. Because most research documents static strength measures in children and adolescents, there is still a clear need to measure and interpret isokinetic muscle strength and muscle power during growth and maturation (De Ste Croix et al., 2003; Van Praagh and Doré, 2002).

During physical activity, muscular contractions place force on bone leading to structural adaptations in bone (see Chapter 16). Researchers have found that muscle power, assessed by a vertical jump, has strong and consistent positive associations with bone strength in adolescent boys and girls (Janz et al., 2015). These findings support the use of the vertical jump in a comprehensive assessment of health-related physical fitness.

There is also a place for handgrip analysis in children and adolescents in the assessment of health-related physical fitness. Although (as noted above) skeletal mass typically increases throughout childhood and adolescence, there are exceptions to this pattern. Skeletal muscle mass and function less than expected for any given age are considered pathological and termed **sarcopenia**. Recent research has shown that children and adolescents may develop sarcopenia. In youth, when sarcopenia occurs, it is often associated with osteoporosis (termed sarcosteoporosis), obesity (labelled sarcopenic obesity), or both conditions (termed osteosarcopenic obesity). Sarcopenic obesity is present when there is a disproportion between the amounts of lean mass and fat mass, while osteosarcopenic obesity is present when bone mineral density and muscle mass is low and body fat is high (Ormsbee et al., 2014). Given the increasing levels of physical inactivity and obesity, there is growing concern about

these conditions. Normative data for skeletal muscle mass per se are lacking. However, decreased muscle mass is only one component of sarcopenia; impaired muscle function is also needed to establish the presence of sarcopenia. Therefore, a fitness test is useful. The ratio of handgrip strength to body mass index (BMI) has been shown to predict the presence of sarcopenic obesity in children (Gilligan et al., 2020; Ormsbee et al., 2014; Steffl et al., 2017).

Sarcopenia Skeletal muscle mass and function less than expected for any given age.

Older Adults

Aging is inevitably associated with a loss of contractile muscle mass resulting in impaired contractile function and performance. As with children and adolescent, such changes are called sarcopenia. Although aging is always associated with loss of muscle mass and function, there is ample evidence that lifelong activity has the potential to mitigate the amount of loss of muscle mass and function (Power et al., 2013). It is becoming increasingly clear that changes in muscle function with aging are multidimensional and include qualitative features about the structure and function of contractile tissue in addition to quantitative measures of the amount of muscle mass (Fragala et al., 2015).

Significant declines in muscular strength in both males and females are well documented from middle age to old age, but the rate of loss varies substantially among muscle groups (Rogers and Evans, 1993). **Figure 18.19** shows a generalized change in strength (grip strength) over the normal lifespan for males and females (Komi, 1992). From the peak values in late adolescence or early adulthood, strength is maintained until approximately 45–50 years, followed by a fairly gradual decline into and beyond the 70s. The decline in muscle strength generally amounts to about 15% per decade in the sixth and seventh decades of life and 30% per decade after that (Rogers and Evans, 1993). Relative static endurance (at ~40–50% MVC) is similar between older and

younger individuals. **Figures 18.16 and 18.19** show parallel declines in performance and show that different muscle groups decline at different rates, even between the sexes.

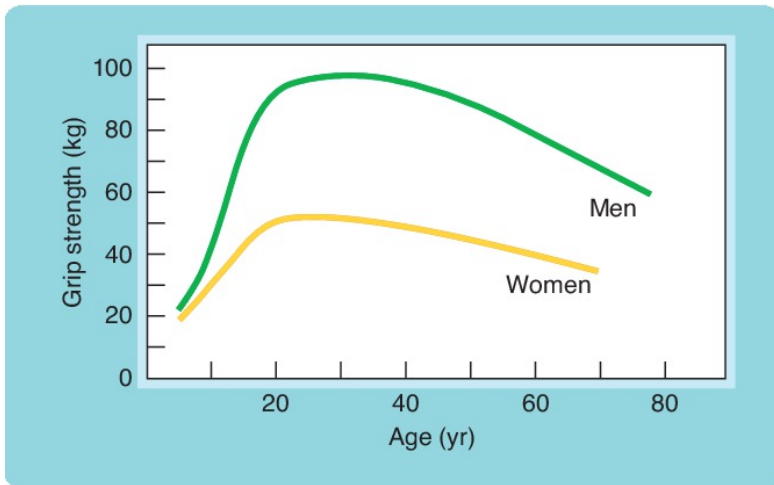


Figure 18.19 Theoretical Maximal Handgrip Strength.

What causes the age-related decline in strength? Three possibilities are (1) a loss of muscle mass, (2) a loss of mechanical or contractile properties (fiber type changes, fiber size changes, fiber number changes), and/or (3) reduced activation of motor units or denervation.

Muscle mass is clearly lost with age. Between the ages of 30 and 70 years, almost 25% of muscle mass is lost in both males and females (Rogers and Evans, 1993). This reduces force production. Some evidence, however, supports the contention that the decline in strength with aging is greater than can be accounted for just by the loss of muscle mass. The capacity to exert force per unit of cross-sectional area also declines (Rogers and Evans, 1993).

Early studies using traditional biopsy techniques seemed to show a preferential loss of FT and, especially, FG fibers. Since these are the high-force fibers, this result was intuitively logical. More definitive studies using whole-muscle cross-sectional techniques have shown, however, that both ST and FT fibers are lost equally with aging (Deschenes, 2004).

As shown in **Figure 18.20**, the loss of muscle fibers begins at about age 30, and by age 80, a reduction of between 25% and 40% has occurred in females and males (Rogers and Evans, 1993). The genetically predetermined percentages of ST and FT fibers remain constant. The SO fibers appear to maintain their size longer than do FT fibers. Thus, although FT fibers are not lost at a faster rate than SO fibers, they do atrophy faster, and FG fibers atrophy faster than do FOG fibers. Since FT fibers are generally the larger fibers, the preferential loss in size of these fibers largely accounts for the overall decrease in muscle size and strength with aging.

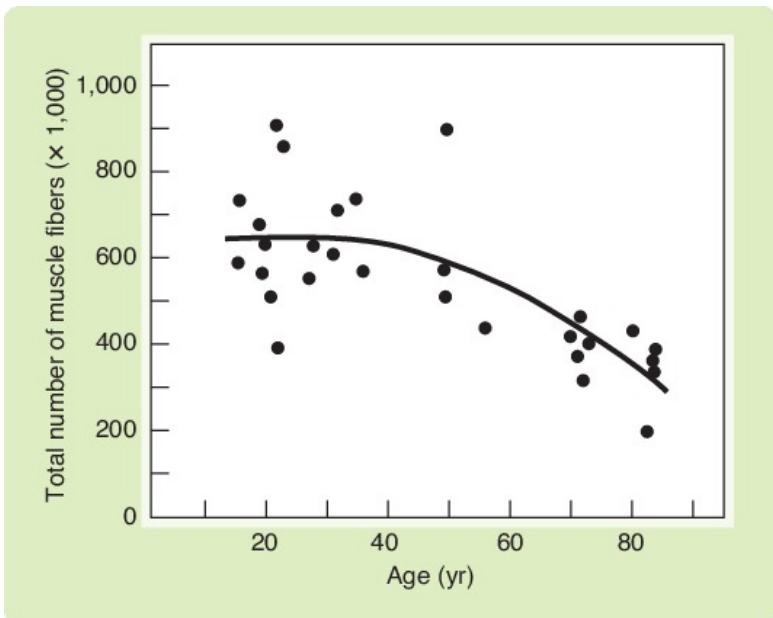


Figure 18.20 Loss of Muscle Fibers with Age.

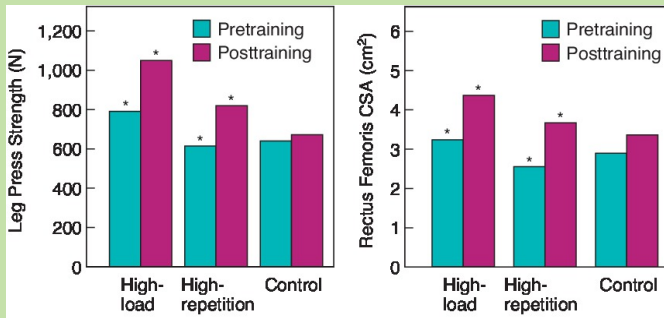
Source: Reprinted with permission from Rogers, M. A., & W. J. Evans: Changes in skeletal muscle with aging: Effects of exercise training. In Holloszy, J. O. (ed.): *Exercise and Sport Sciences Reviews* (Vol. 21). Baltimore, MD: Williams & Wilkins, 65–102 (1993).

FOCUS ON APPLICATION

Effect of Different Training Programs on Muscle Size and Strength

The cross-sectional area of muscle is related to the force it can generate. This implies that if a training program causes muscle hypertrophy (increase in size), it will also result in increased strength. However, there is considerable uncertainty about the optimal training program for improving muscle strength, and it has been speculated that not all populations may experience muscle hypertrophy. Postmenopausal women who are estrogen deficient, for example, may benefit tremendously from increased strength because independent living is related to strength. Yet it has not been established how this group would respond to different resistance training programs. [Bemben et al. \(2000\)](#) investigated the effects of high-load (80% of 1-RM; 8 reps) and high-repetition (40% of 1-RM; 16 reps) resistance training protocols on the musculoskeletal system of early postmenopausal, estrogen-deficient females. The accompanying graphs display changes in only two of the many variables investigated: rectus femoris muscle cross-sectional area (CSA) (measured by ultrasound) and leg press strength.

These graphs show that both training protocols effectively increased muscle strength (measured by the leg press) and increased muscle cross-sectional area (CSA). Based on these and other data, these authors concluded that both high-load and high-repetition resistance training programs are effective for improving muscular strength and size in postmenopausal women. This finding is important for exercise professionals because it indicates that postmenopausal women do adapt positively to exercise training and that a low-intensity, high-repetition training program can be beneficial for developing muscular fitness in women for whom a high-intensity program may not be appropriate.



Source: Adapted with permission from Bemben, D. A., N. L. Fethers, M. G. Bemben, N. Nabavi, & E. T. Koh: Musculoskeletal responses to high- and low-intensity resistance in early menopausal women. *Medicine & Science in Sports & Exercise*. 32(11):1949–1957 (2000). Copyright ©2000 The American College of Sports Medicine.

Of great concern is the practical meaning of these changes. Weakened respiratory muscles restrict aerobic activity. Weakened muscles around joints lead to instability, difficulty in restoring balance, and a greater potential for falls ([Aoyagi and Shephard, 1992](#)). Insufficient strength to get in and out of chairs, carry groceries, or take caps off medicine or food jars can lead to a loss of independent living. The most effective way to prevent these difficulties is systematic exercise training. Muscle aging cannot be prevented but can be delayed. Thus, maintaining and/or increasing muscular strength, endurance, and power is important for different reasons throughout the life span. Nonetheless, muscles respond to exercise training in basically the same fashion at all ages; that is, trained muscles produce greater force.

Summary

1. Muscle tension is the force developed when a contracting muscle acts on an object. Load is the force exerted on the muscle by the object. For a muscle to move a load, muscle

tension must exceed the force of the load.

2. In isotonic contractions of muscle fibers, force production is constant as the muscle fiber contracts. In the intact human system, such a contraction is practically impossible. Instead, the load is constant but the force needed to move it through the range of motion is not. Thus, the term dynamic more accurately describes contraction within the intact human.
3. If movement results from a contraction in which muscle shortening occurs, it is a concentric dynamic contraction. If movement results from a contraction in which muscle lengthening occurs, the contraction is referred to as an eccentric dynamic contraction.
4. In isokinetic contractions of muscle fibers, the velocity of contraction is constant. In the intact human, the velocity of movement varies with joint angle. Specialized equipment can hold the rate of limb displacement constant, resulting in isokinematic contraction.
5. A muscle fiber contraction that does not result in a meaningful length change in the muscle fiber is termed isometric. An intact fiber has an elastic element, such that some fiber shortening actually occurs with contraction even though no limb displacement occurs. Thus, the term static is preferable to isometric to describe this type of contraction in humans.
6. The amount of force produced by muscles depends on neural and mechanical factors. Important mechanical factors include length-tension-angle relationships, force-velocity relationships, elasticity-force relationships, and architectural design.
7. Muscular fatigue may be caused by a variety of factors, which can be described as central or peripheral, or as electrophysiological or biochemical in nature. The cause is determined largely by the muscle fiber type and, therefore, varies with different types of activity. Muscular fatigue results in a loss of muscle function.
8. Muscular soreness probably results from a combination of mechanical trauma, muscle damage, and inflammation.
9. Differences in strength between the sexes are largely due to the greater muscle mass of males. The magnitude of the

difference in strength between males and females depends on the absolute or relative expressions of strength, the region of the body where strength is measured, and the training status of the individuals.

10. Strength development occurs from infancy through maturity. The increase in strength during childhood and adolescence is more than can be accounted for just from growth in size.
11. Muscular strength declines in both males and females from middle age to old age, due in part to loss in both ST and FT fibers. Aging of muscles cannot be prevented, but loss of strength can be delayed with appropriate exercise training.

Review Questions

1. Define isotonic, isokinetic, and isometric contractions. Discuss how they relate to dynamic and static contractions.
2. Diagram the force-length relationship in a muscle fiber. Diagram a strength curve for biceps flexion, knee flexion, and knee extension. Discuss the relationship between force and length in the muscle fiber and in the whole muscle.
3. Graph the force-velocity relationship in (a) a muscle fiber and in (b) a whole muscle. Identify the eccentric contraction on graph (a) and identify a static contraction on graph (b).
4. Create a schematic representation of the possible sites of muscular fatigue.
5. Indicate the most probable cause of muscle fatigue for the following categories of exercise: long-term, moderate to heavy, submaximal aerobic, incremental aerobic exercise to maximum, static, dynamic resistance, and very short-term, high-intensity anaerobic exercise.
6. Discuss the integrated model of delayed-onset muscle soreness (DOMS). How can DOMS be prevented or treated?
7. What are the primary laboratory methods for measuring muscular function? What are the primary field tests to measure muscular function? What are the limitations of these methods? What determines the appropriate test to administer?

8. Compare male and female strength development during childhood and adolescence.
9. Discuss differences in strength between adult males and females. How is the difference in strength affected by absolute or relative units used to express strength? How do differences vary among different regions of the body? What are the most likely causes of sex-related differences in muscular function?
10. What factors account for the age-related decline in muscular strength? Can this loss be minimized or slowed? If so, how?
11. What is sarcopenic obesity and how does it differ from osteosarcopenic obesity?

For further review and study tools, visit Lippincott Connect.

Literature Search

In this chapter, we discussed skeletal muscle contraction for movement. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search skeletal muscle contraction, this will yield a huge selection of articles.
 - b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Skeletal muscle contraction mechanisms.
 - ii. Skeletal muscle contraction and muscle damage.
 - iii. Skeletal muscle contraction and aging.
 - iv. Skeletal muscle contraction and nutrition.
 - v. Continue your search for aspects of this topic that are of particular interest to you.
-

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19 Muscular Training

Principles and Adaptations



CHAPTER OUTLINE

Introduction

Overview of Resistance Training

Application of the Training Principles

Specificity

Overload

Rest/Recovery/Adaptation

Progression

Individualization

Maintenance

Retrogression/Plateau/Reversibility

Warm-Up and Cooldown

The Application of Training Principles to Bodybuilding

Neuromuscular Adaptations to Resistance Training

Muscle Function

Muscle Size and Structure

Neural Adaptations

Metabolic Adaptations

Hormonal Adaptations

**Male-Female Resistance Training Adaptation
Comparisons**

**Resistance Training Adaptations in Children and
Adolescents**

Resistance Training Adaptations in Older Adults

**Muscular Adaptations to Aerobic Endurance Training
Programs**

Muscular Adaptations to Concurrent Training

**Special Application: Muscular Strength/Endurance and
Health**

**Special Application: Muscular Strength and Endurance
and Low Back Health**

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Apply each of the training principles to the development of a resistance training program.
- Describe muscular adaptations to resistance training.
- Identify similarities and differences in training adaptations between males and females, children and adults, and young and older fitness participants.
- Discuss the relationship between muscle function and low-back

pain.

Introduction

The previous chapters have discussed how isolated muscle fibers and intact muscles contract to produce coordinated movement. This chapter discusses specific applications of the training principles for the development of muscular fitness and the training adaptations that result from an exercise training program. The final section applies this information to the problems of low-back pain.

Although a training program can be developed using static contractions, in reality such programs are rare. Therefore, this chapter focuses on dynamic resistance and isokinetic training programs. The term *resistance training program* is used inclusively to encompass dynamic resistance and isokinetic training unless otherwise specified.

Overview of Resistance Training

Resistance training is a systematic program of exercises involving the exertion of force against a load, with the goal of developing strength, endurance, and/or hypertrophy of the muscular system (Davies and Barnes, 1972). It is commonly called *weight training*. Resistance training is a recommended component of a well-rounded fitness program for healthy children, adolescents, young and middle-aged adults, and older adults. A resistance training program should be individualized, be progressive, and involve all the major muscle groups (American College of Sports Medicine [ACSM], 2009; Haskell et al., 2007; Nelson et al., 2007).

Resistance Training A systematic program of exercises involving the exertion of force against a load, used to develop strength, endurance, and/or hypertrophy of the muscular system.

The scientific and medical community accepts that muscular strength is a necessary trait for health, functional ability, athletic performance, and an enhanced quality of life. Resistance training is acknowledged as an effective way to develop musculoskeletal health and is now routinely recommended by health and fitness professionals. Especially when incorporated as part of a comprehensive fitness program, resistance training helps reduce the risk factors associated with coronary artery disease, type 2 diabetes, and colon cancer; maintains muscle mass in weight loss; and improves dynamic stability and preserves functional capacity (ACSM, 2009; Bird et al., 2005). The American College of Sports Medicine (ACSM) position stand titled “Progression Models in Resistance Training for Healthy Adults” provides guidelines for progression models of resistance training that can be applied to novice, intermediate, and advanced training levels (ACSM, 2009).

When done under skilled supervision with proper instruction in form, breathing, body mechanics, and prescription of loads, resistance training can be enjoyed by all individuals. It carries a relatively low risk of harm for prepubescent children (Behm et al., 2008; Faigenbaum and Myer, 2010a; Faigenbaum et al., 2009, 2015). Resistance training is also appropriate and effective in older adults as long as training principles are properly applied (Borde et al., 2015; Nelson et al., 2007; Ribeiro et al., 2014; Steib et al., 2010).

Application of the Training Principles

The training principles for a safe and effective resistance training program are the same as for other types of exercise programs.

Specificity

As in any training program, a plan for muscular fitness must be specific to the individual's goals. Individuals may pursue resistance training as part of a comprehensive fitness program, to improve overall health, to enhance functional capacity, to change personal appearance, or to improve athletic performance. For muscular fitness, the goals of a resistance training program may

include the development of muscular strength, hypertrophy, power, muscular endurance, or any combination of the above. Of course, these goals overlap. A person who gains muscle mass (hypertrophy), for example, is certainly stronger. However, the resistance training program should emphasize the goal that is most important to the individual. Many individuals focus on different components of muscular fitness at different times as part of a well-designed periodization plan.

Muscles respond specifically to different types of contraction and loads imposed. Athletes in many sports, including basketball, football, hockey, volleyball, etc., use dynamic resistance programs to increase muscle strength, mass, and power. Athletes in other sports, including swimming, are more likely to use isokinetic training programs to develop strength through a specified range of motion.

The specificity training principle applies to the muscle groups being trained, the type of contraction performed, the selection of single- or multiple-joint exercises, and the velocity of contraction. Because resistance training is specific to the muscle groups being trained, a resistance training program should include at least one exercise for all the major muscle groups of the body. It is recommended that all resistance training programs include a concentric and eccentric component. Although most resistance training programs should include single-joint and multiple-joint exercises, programs designed to develop muscular power should emphasize multiple-joint exercises ([ACSM, 2009](#)).

Specificity also applies to the velocity of contraction in isokinetic exercises ([ACSM, 2009](#); [Kawamori and Haff, 2004](#); [Perrin, 1993](#)). Exercise performed at slow velocities generally increases torque specific to the training velocity. Training at high velocities generally increases strength at and below the exercise velocity and is thus not as specific as slow-velocity training. Furthermore, it is a misconception that the velocity of isokinetic exercise should be specific to athletic events ([Perrin, 1993](#)). In reality, the angular velocities of joint movements in many athletic events (such as throwing) far exceed what can be performed during isokinetic exercise.

Overload

Successfully applying the overload principle in resistance training requires the manipulation of intensity (load), volume, frequency, and rest intervals. The *intensity* of the workout can be expressed as a relative load (a percentage of an individual's 1-RM) or an absolute load (a specific amount of weight). *Volume* is a measure of total amount of work done in an exercise session and can be expressed in several ways, for example, total number of repetitions, number of repetitions \times number of sets, or number of repetitions \times number of sets \times intensity (Wernbom et al., 2007). **Table 19.1** provides recommendations for the manipulation of these variables depending on the individual's goal and fitness level. The amount of stress, or load, applied to the muscle largely determines the response of the muscle. A muscle exposed to near-maximal load will develop greater strength than a muscle experiencing many repetitions of a lighter load. In contrast, a muscle that performs many repetitions of a lighter load will develop relatively more muscular endurance than one exposed to a small number of near-maximal repetitions. There is an inverse relationship between the load (weight) that can be lifted and the number of repetitions that can be performed. By definition, the most weight that can be lifted one time is the **one-repetition maximum (1 rep max; 1-RM)**. **Figure 19.1** provides guidelines for estimating the number of repetitions that are possible at various loads, expressed as a percentage of 1-RM (Baechle et al., 2000). When using **Figure 19.1**, keep in mind that the number of repetitions that can be performed at any given load is only an estimate. The actual number of repetitions that can be performed at any given load varies among individuals (resistance-trained athletes often can exceed the predicted repetitions) and among muscle groups.

TABLE 19.1 Application of Overload Principle to Resistance Training Program

| Goal/Level | Order | Loading | Volume | Rest intervals |
|--------------------|---|---|--|---|
| Strength | | | | |
| Novice | | 60–70% of 1-RM | 1–3 sets, 8–12 reps | 2–3 min for core, 1–2 min for others |
| Intermediate | Large → small | 70–80% of 1-RM | Multiple sets, 6–12 reps | 2–3 min for core, 1–2 min for others |
| Advanced | Multiple joint → single joint High intensity → low intensity | 70–100% of 1-RM—periodized | Multiple sets, 1–12 reps—periodized | 2–3 min for core, 1–2 min for others |
| Hypertrophy | | | | |
| Novice | | 60–70% of 1-RM | 1–3 sets, 8–12 reps | 1–2 min |
| Intermediate | Large → small | 70–80% of 1-RM | Multiple sets, 6–12 reps | 1–2 min |
| Advanced | Multiple joint → single joint High intensity → low intensity | 70–80% of 1-RM with emphasis on 70–85%—periodized | Multiple sets, 1–12 reps with emphasis on 6–12 reps— periodized | 2–3 min—very heavy, 1–2 min—light to medium heavy |
| Power | | | | |
| Novice | | Heavy loads (>80% of 1-RM)—strength | Train for strength | 2–3 min for core, 1–2 min for others |
| Intermediate | Large → small | Light (30–60% of 1-RM) velocity—periodized | 1–3 sets, 3–6 reps | 2–3 min for core, 1–2 min for others |
| Advanced | High intensity → low intensity | | 3–6 sets, 1–6 reps— periodized | 2–3 min for core, 1–2 min for others |
| Endurance | | | | |
| Novice | | 50–70% of 1-RM | 1–3 sets, 10–15 reps | 1–2 min for high-rep sets |
| Intermediate | Variety recommended | 50–70% of 1-RM | Multiple sets, 10–15 reps or more | 1–2 min for high-rep sets |
| Advanced | | 30–80% of 1-RM— periodized | Multiple sets, 10–25 reps or more— periodized | >1 min for 10–15 reps |

Source: ACSM (2009).

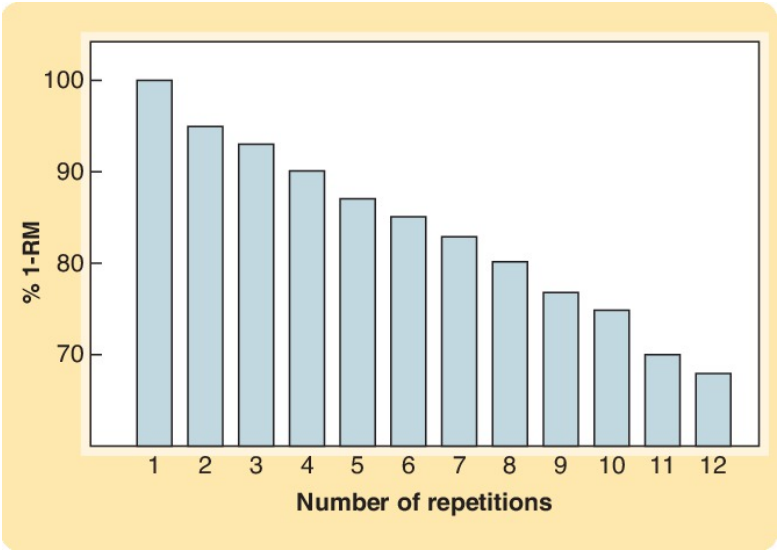


Figure 19.1 Estimated Repetitions that Can Be Performed at a Particular % of 1-RM.

One-Repetition Maximum (1 rep max; 1-RM) The most weight that can be lifted one time.

Complete the [Check Your Comprehension box](#) to use the information presented in this section.

CHECK YOUR COMPREHENSION 1

1. A college basketball player is in the specific preparation phase of her periodization plan, preparing for her fourth season as a forward. What volume should she emphasize to increase her strength? What rest interval is appropriate?

Check your answer in Appendix C.

Historically, programs based on the overload principle began in 1948 when DeLorme and Watkins introduced *progressive resistance exercise*. The DeLorme and Watkins (1948) program uses 30 repetitions per training session for each muscle group exercised. The 30 repetitions are broken down into 3 sets of 10 repetitions (reps) each, as follows:

Set 1 = 10 repetitions at 50% of 10-RM

Set 2 = 10 repetitions at 75% of 10-RM

Set 3 = 10 repetitions at 100% of 10-RM

Since the 1950s, considerable research has been done to determine the optimal number of repetitions and sets, the workload, and the frequency for developing muscular strength, endurance, and power. This research has led to the development of many training systems. No single combination of repetitions and sets produces the best results; however, the ideal number of sets depends on the individual's goals and differences.

Modern progressive resistance training programs often address goals along a “strength-endurance” continuum. The proper

manipulation of repetitions, intensity, sets, and rest periods is important in achieving desired goals. [Campos et al. \(2002\)](#) designed a study that investigated three different training regimes:

1. Low repetitions (3- to 5-RM); high intensity; 4 sets; 3-minute rests
2. Medium repetitions (9- to 11-RM); intermediate intensity; 3 sets; 2-minute rests
3. High repetitions (20- to 28-RM); low intensity; 2 sets; 1-minute rest

The authors found that maximal strength improved significantly more with the low-repetition group, but the maximal number of repetitions that could be performed at 60% of 1-RM improved the most in the high-repetition group. Maximal aerobic capacity and time to exhaustion on a graded exercise test increased only in the high-repetition group. In contrast, all three major groups of muscle fibers increased in the low- and intermediate-repetition group but not in the high-repetition group ([Campos et al., 2002](#)). These findings confirm that physiological adaptations to resistance training are dependent upon the design of the training program and the manipulation of the variables that determine overload, highlighting the importance of identifying an individual's goal and designing a specific resistance training program to meet that goal.

To elicit improvements in both muscular strength and endurance, the ACSM recommends that a minimum of one set of 8–12 repetitions be performed with each of the major muscle groups 2–3 d·wk⁻¹ ([ACSM, 2009](#)). It may be more appropriate for older or frailer individuals to perform 10–15 repetitions per muscle group ([Nelson et al., 2007](#)). Although greater gains in strength may result from performing more than one set of exercise, for the general public, the incremental strength gains from additional sets are offset by the longer period needed to complete the exercise and the increased risk of orthopedic injury. A meta-analysis compiled evidence that increasing training volume leads to improved muscle hypertrophy. This dose-response curve was characterized by an increase in the rate of

hypertrophy in the initial part of the curve, followed by peak rate of hypertrophy and, in turn, followed by a plateau ([Wernbom et al., 2007](#)). Thus, athletes and individuals wishing to optimize muscular fitness (and hypertrophy in particular) may benefit from performing more than one set; this is reflected in current recommendations (see **Table 19.1**).

Resistance training programs need to address multiple considerations. Core exercises develop the strength and endurance of the muscles of the trunk and pelvis, which are responsible for body stability and essential for most human movement. Core strengthening has received greater emphasis in recent years, and it is currently recommended that all resistance training programs include core strengthening exercises.

To avoid fatigue, it is recommended that exercises be arranged in a specific order, alternating lower-body with upper-body exercises. This tactic allows the muscles to recover between exercises or exercise sessions. Generally, large muscle groups should be exercised first, followed by smaller muscle groups. For example, if an exerciser wants to work the latissimus dorsi (lats) and the biceps, the lats should be worked first (pulldowns), because they involve a larger muscle group. This approach helps ensure that the fatigue of the smaller muscle group (the biceps) does not limit the work that can be performed by the larger muscle group (the lats). Complete the [Check Your Comprehension 2 box](#) to determine your understanding of sequencing strength training exercises.

CHECK YOUR COMPREHENSION 2

1. A 22-year-old collegiate football player is writing out his next resistance training workout. If he wants to avoid fatiguing his muscles too early and allow himself to recover as much as he can between exercises, in what order should he put the following exercises?
2. Bench press, calf raises, shrugs, sit-ups, shoulder press, power cleans, leg extensions, calf raises, forearm curls, back squats, and lat pulldowns.

Check your answer in Appendix C.

The length of rest periods between exercise sets is also related to the overload placed on the muscles. If the goal is maximal strength gains, relatively long (several minutes) rest periods should be used between sets. If endurance is the primary goal, shorter rest periods should be used (ACSM, 2009; Baechle et al., 2000).

The optimal frequency of resistance training also depends on the individual's goals and training status. Additionally, frequency varies depending on training stage (periodization). Obviously, a competitive bodybuilder trains more frequently than an adult fitness participant hoping to derive the health-related benefits of resistance training. The ACSM recommends strengthening exercises at least 2 days a week to achieve the health-related benefits of such exercises (ACSM, 2009; Haskell et al., 2007). Additional training leads to additional benefits. Training from 2 to 4 days a week appears to be most popular with weight lifters. Twice a week is considered the minimum necessary to improve muscular strength; training less than twice a week may predispose the individual to muscle soreness and injury. Athletes who train 4 days a week often follow a program that alternates an upper-body workout day with a lower-body workout day, so that 2 days a week are devoted to each anatomical area.

Competitive resistance-trained athletes often follow a training program in which they train 3 or 4 days consecutively and then take a day off. With such a program, they follow a split routine: each muscle group is exercised only twice a week. A split routine emphasizes a single muscle group in a workout. This muscle group is then rested for 48–72 hours. A variation of this program is the double-split routine in which two exercise sessions are performed on each workout day.

The frequency of training also depends on the individual's periodization plan. An athlete may engage in resistance training 4–6 d·wk⁻¹ in the general preparation stage (off-season), 3 or 4 d·wk⁻¹ in the specific preparation stage (preseason), 1 or 2 d·wk⁻¹ in the competition season (in-season), and 1–3 d·wk⁻¹ in the transition (active rest) stage of periodization.

The duration—the amount of time spent in the weight room—

depends largely on the number of repetitions, the number of sets, and the number of exercises performed. In itself, time spent on resistance training is not a critical component of the exercise prescription.

Overloading for prepubescent children should involve the same factors as outlined for adults, with some modification. A beginning program should stress learning proper form, techniques, and safety considerations, such as spotting (Behm et al., 2008; Faigenbaum and Myer, 2010b; Faigenbaum et al., 2015). The equipment should fit the child, which may mean that some exercise machines (designed primarily for adult males) should not be used. Exercises should include major muscle groups and work both agonist and antagonist muscles at each joint.

Figure 19.2 outlines guidelines from the International Consensus Statement for the progression of a resistance training program based on each athlete's resistance training skill competency (RTSC) (Lloyd et al., 2014). Children should begin with a program consisting of 1–2 sets early in the training program while skill development is emphasized. In the early phase, it is recommended that intensity be less than or equal to 50% of 1-RM. As children become more skilled, it is reasonable to increase to 2–4 sets of 6–12 reps at up to 80% of 1-RM. A frequency of 2 or 3 d·wk⁻¹ is recommended at this point. Highly skilled young athletes may undertake a resistance training program that involves multiple sets using weights of more than 85% of 1-RM. Historically, there has been concern about having children perform maximal lifts. However, studies (see Focus on Application: Clinically Relevant box) have shown that children can safely perform a 1-RM test to guide a lifting program. Please review the **Check Your Comprehension 3—Case Study box** to ensure your understanding of the use of resistance training to improve health.

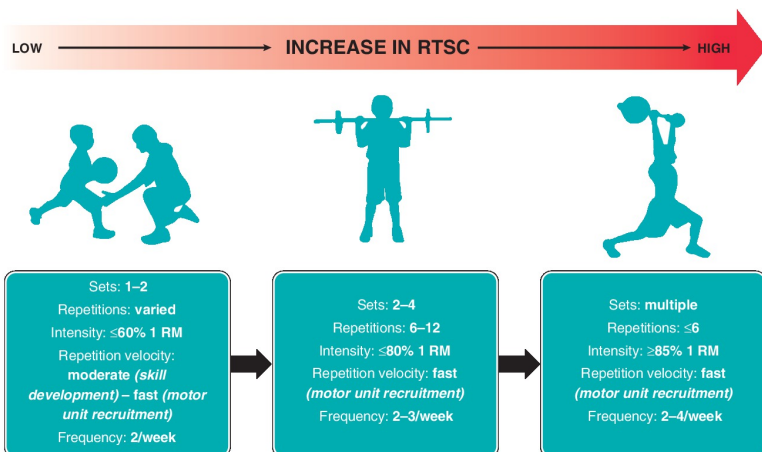


Figure 19.2 Resistance Training Guidelines for Youth with Progression Based on Resistance Training Skill Competency (RTSC).

Source: Lloyd et al. (2014).

CHECK YOUR COMPREHENSION 3-CASE STUDY 1

Nikesha, a 48-year-old woman, is committed to improving her health. Her youngest child just enrolled in college, and approximately 2 months ago, she began walking 3 d·wk⁻¹ for 30 minutes. She recently read an article in a health magazine that extolled the benefits of resistance training. Hoping to improve her strength, she joins the local YMCA and now is seeking advice on how to design and implement her program. Based on the overload principle and her individual goals, what would you recommend?

Check your answer in Appendix C.

Older adults also benefit from resistance exercise in many ways (including improved overall health, strength, and functional ability). Many professional organizations, including the American College of Sports Medicine and the American Heart Association, recommend resistance training for older individuals. Current

recommendations for older individuals include 1 set of 10–15 repetitions of exercises involving all the major muscle groups at least 2 days a week (Nelson et al., 2007). However, research evidence suggests that older individuals seeking more strength gain have superior adaptations to 3-set resistance training than with 1-set training in leg exercises (Paulsen et al., 2003; Steib et al., 2010).

Rest/Recovery/Adaptation

Muscles adapt to the stress placed on them. The most obvious changes that result from a resistance training program are increased muscle strength and size. However, the extent to which muscles adapt to training by becoming stronger and bigger depends on the training program that is followed. For example, at some point during a resistance training program, individuals will realize that their initial 10-repetition maximal weight can now be lifted more than 10 times. This indicates that adaptation has occurred. The rate of adaptation depends on several factors, including rest periods and adequate diet, and the rate may not be the same for all muscle groups trained. The importance of rest (recovery) between exercise sessions to allow for the positive adaptations of exercise training cannot be overemphasized. At least 1 day of rest should follow a day of training for a particular muscle group. Adequate rest periods and alternating heavy and light days are important to allow training adaptations to occur and to prevent injury and soreness. As mentioned earlier, many competitive athletes who lift high volumes allow 72-hour rest periods before training the same muscle group again.

FOCUS ON APPLICATION | *Clinically Relevant*

Is It Safe to Determine 1-RM in Children?

Establishing a 1-RM load is necessary to develop a resistance

training program based on a percentage of maximal capacity. The safety of maximal testing has been explored for many populations, including older adults, and patients with cardiac and pulmonary disease. However, many clinicians and researchers have been cautious about maximal testing in children for fear of injury.

Faigenbaum and colleagues addressed the safety and efficacy of 1-RM testing in healthy children. The participants were healthy boys ($n = 64$) and girls ($n = 32$) between the ages of 6.2 and 12.3 years (mean age = 9.3 years). The children had no previous history of resistance training. The children participated in an introductory training session and were taught proper lifting technique for each exercise. Each participant's 1-RM was then determined for an upper-body exercise (either standing chest press or seated chest press) and a lower-body exercise (either leg extension or leg press). All testing was performed on a weight machine. Before attempting a 1-RM, participants performed 6 reps with a relatively light load, then 3 reps with a heavier load, and finally a series of single reps with increasing loads. If the weight was lifted with proper form, the load was increased by 0.5–2.3 kg. On average, the upper-body 1-RM was determined within 7 trials and the lower-body 1-RM was determined within 11 trials. An NSCA-certified strength and conditioning specialist supervised all testing.

No injuries occurred during the study period, and the participants tolerated all testing well. No complaints of severe muscle soreness were reported. This study supports other smaller studies that found that children can perform 1-RM with no apparent adverse consequences when properly supervised. Since resistance training is becoming more popular and helps increase health and overall fitness in this population, a 1-RM strength test would be beneficial for practitioners to evaluate the effectiveness of a resistance program, assess the participant's strengths and weaknesses, base the training program on, and motivate progress. Of course, the authors rightly note that any testing with children must be properly supervised and that great care must be taken to teach proper form and ensure that it is used—both during testing and during lifting exercises.



Source: [Faigenbaum et al. \(2003\)](#).

[McLester et al. \(2003\)](#) have investigated the time course of muscle endurance recovery in a group of young (18–30 years) and older (50–65 years) men using the number of repetitions that can be performed after a given time as a measure of recovery. The authors report that after 24 hours of recovery, neither of the groups could perform as many repetitions as they did on the initial day of testing. By 48 hours, both groups could perform the same number of repetitions, and by 72 hours, the younger subjects could perform more repetitions. This study reported large individual variability and suggests that individual recovery testing is practical.

Adaptation occurs in children as it does in adults. Careful monitoring of recovery between sessions is probably even more important for children to ensure they rest adequately (at least 48 hours).

Progression

Once the body has adapted to the current training level, exercise stress should be increased following the overload principle if

further adaptations are desired. This principle is the basis of progressive resistance exercise.

Progression should be done gradually. Progression can be accomplished by increasing the load, the repetitions, the number of sets, or the frequency of the workout or by decreasing the rest period between sets. Load and the number of repetitions are the variables most often manipulated. Again, the choice depends largely on the individual's goals. If strength is the primary goal, then a heavier weight should be used. If endurance is the goal, then the same weight should be lifted more times. Often a combination of these two variables is used. For instance, many people begin with a weight they can lift for 6 repetitions. As they adapt to this stress, they progress to 7 repetitions, then 8 repetitions, and so on. Once they can perform 8–10 repetitions, they increase the weight to something they can again only lift for 6 repetitions. As recommended earlier, a novice lifter should begin by doing more repetitions with a lighter load. Once the body has adapted, the individual can lift heavier weights. Although it has been recommended that acute increases in training volume should be small (2.5–5%), advanced athletes often exceed this recommendation ([ACSM, 2009](#); [Kraemer and Ratamess, 2004](#)).

FOCUS ON APPLICATION

Does Supervision of a Strength Training Program Make a Difference?

Many individuals seek the assistance of an exercise professional when implementing their training programs. A personal trainer may provide important information and advice and serve as a motivator. But does the supervision of a training program lead to improved performance? [Mazzetti et al. \(2000\)](#) investigated the influence of direct supervision of resistance training on strength performance. Their results suggest that supervision does make a difference. These

researchers randomly assigned volunteers with 1–2 years of lifting experience to a supervised group or an unsupervised group for a 12-week training period. The supervised group was trained one-on-one by a personal trainer. The unsupervised group attended one private fitness consultation at the beginning of the training and performed subsequent training without direct supervision, although the personal trainer was present at all training sessions to answer questions concerning the program and to confirm the participants' adherence to the program. Both groups followed identical periodized resistance training programs consisting of preparatory (10- to 12-RM), hypertrophy (8- to 10-RM), strength (5- to 8-RM), and peaking (3- to 6-RM) phases using free weights and variable resistance equipment. At the end of 12 weeks, there was no difference between the number of training sessions, sets, or repetitions performed per week for the squat and bench exercises. However, the supervised group had lifted more weight per set than the unsupervised group. Both training groups experienced increases in strength, but the supervised group had greater increases than the unsupervised group. These results clearly show that personal training (one-on-one supervision) can affect the strength gains achieved by participants, even if they have been lifting on their own for 1–2 years.



Source: Mazzetti et al. (2000).

As in adults, progression in prepubescent children should be done slowly, especially in terms of intensity. Light to moderate loads of 8- to 15-RM are recommended for the first 2–6 months; after that, heavier loads can be introduced.

Individualization

The first step in individualizing a resistance training program is to determine the participant's personal goals, followed by evaluating the individual's current strength level. This assessment is usually done by determining the individual's one-repetition maximum (1-RM). A 1-RM should be established for each muscle group exercised; it is then used to determine the work intensity. For example, a program may call for an individual to do 6 repetitions per set at 80% of the 1-RM.

The final step is determining the training cycle to be used. This technique is often referred to as periodization (see [Chapter 1](#)), and it is a common step among athletes who use resistance training as an integral part of their training programs for athletic competition. For instance, a player who is conditioning for basketball would follow a different weight training program during the transition phase, general preparation phase, and specific preparation phase than during the competition season. Periodization is also important to individuals who use weight training to maintain health-related muscular fitness; it prevents boredom by regularly changing the training program. Competitive resistance-trained athletes also use periodization techniques to vary their training and to prepare for a peak season. **Table 19.2** provides an outline for developing a training program based on the individualization of the training principles.

TABLE 19.2 Developing a Resistance Training Program

| Step | Special Considerations | Applicable Training Principles |
|--|---|---|
| 1. Goal identification | Desired outcome Component of muscular fitness to be stressed Mode of contraction most appropriate Muscle groups to be stressed | Specificity Individualization |
| 2. Evaluation of initial strength or muscular endurance levels | Each muscle group to be used Proper lifting techniques | Specificity |
| 3. Determination of the training cycle (periodization) | Prevention of boredom Peaking | Adaptation Progression Retrogression/ plateau Individualization |
| 4. Determination of the training system (design of a single session) | Exercises to be included Load Number of sets Rest periods Order in which exercises are to be done Warm-up and cool down | Specificity Overload Individualization Warm-up and cool down |

Even if different individuals follow the same program, training adaptations should be expected at different rates. The principle of individualization states that responses to exercise vary among individuals because of factors unique to the individual. In resistance training, these factors include age, body size and type,

initial strength, and, perhaps most importantly, genetic makeup (including fiber-type distribution).

A coach or exercise leader must be sensitive to differences in the rate of individual adaptation because it directly affects the progression of training. Unfortunately, it is a common mistake for a coach to design a program for the entire team and expect everyone's adaptations to occur at the same rate. The result is often frustration for both the coach and athletes and sometimes even overtraining and/or injuries.

Individually prescribing a resistance training program is probably even more important for prepubescent children than for adults. Children are growing both physiologically and psychologically, and their rate of growth varies greatly. Periodization should be used with children as well as adults. Competition between children should be discouraged.

Maintenance

Once the desired level of muscular strength and endurance is achieved, it can be maintained by reduced amounts of work as long as the intensity (workload) is maintained; that is, as long as the same weight is lifted, the individual can maintain strength with only one session per week. A reasonable program to maintain muscular strength and endurance would allow individuals to train at a similar workload but with fewer days per week.

Retrogression/Plateau/Reversibility

Despite the best plans of coaches, training improvements do not occur in a linear fashion. Even with progressively increasing workloads, at times performance will stay at the same level (plateau) or show a decrease (retrogression). The causes may be overtraining or individual differences. If overtraining is suspected, it is wise to include more rest days or include light days in the training regimen. It may also be beneficial to alter the training program using the periodization technique discussed earlier. Once training stops, achieved gains will be reversed.

Warm-Up and Cooldown

A proper warm-up raises body temperature and is often recommended to prevent injury and muscle soreness. Although a warm-up has not been conclusively proven to decrease the incidence of injury, some evidence is consistent with this theory. A higher temperature decreases the viscosity of the joint capsule and increases the speed of muscle contraction and relaxation and enzymatic reactions (Enoka, 1988).

General and specific warm-ups for resistance training are recommended for weight lifting and isokinetic exercises (ACSM, 2009; Perrin, 1993). A general warm-up involves the major muscles of the body; it is similar to the warm-up used for aerobic exercise and includes activities such as jumping rope or jogging. Specific warm-up activities for weight training involve performing the same lifts that are part of the normal program but at a weight well below the training level. The duration and the intensity of the warm-up should be suited to the individual and the task to be performed. A proper warm-up should cause a rise in core body temperature of 0.5–1.0°C but should not be so strenuous that it causes fatigue. Generally, a warm-up is considered adequate when the individual begins to sweat.

A cooldown period, followed by stretching, is recommended after a training session. Cooling down may prevent muscle soreness and lead to an increase in flexibility, an aspect of muscular fitness often overlooked in resistance training programs. Importantly, cooling down helps prevent venous pooling of blood in the lower extremities.

A warm-up and cooldown are just as important for children as for adults. The same pattern of activities should be followed to increase body temperature and to stretch the muscles.

The Application of Training Principles to Bodybuilding

The sport of competitive bodybuilding has gained great popularity in the past several decades. Furthermore, many individuals engage in resistance training programs to enhance their physique. For these athletes, weight lifting is not only about

gaining strength but also about “sculpting” the body. The goals of bodybuilding are to develop superior muscularity and mass, to develop symmetry and harmony between different body parts, and to enhance muscle density and visual separation of muscles. The effect of such programs is shown in **Figure 19.3**. To achieve their goals, bodybuilders follow specific training strategies and a strict diet.



Figure 19.3 Bodybuilders.

A goal of bodybuilding is to “sculpt” the body.

The training strategies for bodybuilding are designed to increase muscle hypertrophy. As discussed earlier, this means that bodybuilders use a very high volume (load times repetitions) of

training. The frequency of training varies, but split routines or double splits are most common among competitive bodybuilders. These programs often allow for training on 3 or 4 consecutive days, followed by a day of rest. This schedule allows for more than 48 hours of recovery for each muscle group. The overall cycle of training (periodization) is important to bodybuilders. Bodybuilders commonly do more strength training in the general preparation phase of their periodization plan to build muscle mass. As the season approaches, the load is reduced and more repetitions are performed in an attempt to gain muscle symmetry.

Diet is perhaps as important in bodybuilding as an appropriate resistance training program. To enhance muscle definition and promote visual separation of the various muscle groups, bodybuilders maintain a low percentage of body fat through a combination of training and diet. Bodybuilders follow a strict low-fat diet, despite the large number of calories. The practice of **cutting** or *ripping* refers to the bodybuilder's attempt to decrease body fat and body water to very low levels before a competition in order to increase muscle definition.

Cutting Decreasing body fat and body water content to very low levels in order to increase muscle definition.

Neuromuscular Adaptations to Resistance Training

Resistance training programs are important for health, fitness, and performance and are widely recommended by leading health organizations. This section addresses neuromuscular training adaptations from resistance training, with an emphasis on changes that occur within muscle tissue. Human skeletal muscle readily adapts to changes in the loading state. The hallmark adaptations to resistance training are increases in muscle strength and size (hypertrophy). **Table 19.3** summarizes neuromuscular adaptations to resistance training.

TABLE 19.3 Neuromuscular Adaptations to Resistance Training

1. Muscle Function
 - a. ↑ Strength
 - b. ↑ Endurance
 - c. ↑ Power
2. Muscle Size and Structure
 - a. Muscle fibers
 - i. ↑ Whole muscle cross-sectional area (CSA)
 - ii. ↑ Muscle fiber CSA
 - iii. ↑ Myofibril protein content
 - iv. Conversion of FOG to FG fibers
 - b. Connective tissue
 - i. ↑ Collagen synthesis
 - ii. = Portion of connective tissue to skeletal muscle
 - iii. ↑ Collagen stiffness
3. Neural Adaptations
 - a. ↑ Motor unit recruitment
 - b. ↑ Synchronization
 - c. ↓ Golgi tendon organ reflex
4. Metabolic Adaptations
 - a. ↑ Glycogen
 - b. ↑ Phosphocreatine (PC)
 - c. ↑ Creatine phosphokinase (CPK)
5. Hormonal Adaptations
 - a. Inconsistent findings for testosterone
 - b. No change in growth hormone (GH)
 - c. Inconsistent findings for cortisol
 - d. ↑ Insulin-like growth factor (IGF-1)

Muscle Function

Resistance exercise leads to increased muscular strength, endurance, and power. Increased strength is the most obvious result of a resistance training program and the reason many individuals participate in resistance training. Strength gains

following a resistance training program vary widely, owing largely to differences in initial strength and the training program. Resistance training at least twice a week improves muscle strength and endurance by approximately 25–100% (Haskell et al., 2007). Furthermore, several meta-analyses have demonstrated that different resistance training protocols, including high versus low loads and high versus low volume, elicit muscular and strength gains in young and old populations (Csapo & Alegre, 2016; Lixandrão et al., 2018; Schoenfeld et al., 2017a, 2017b).

The increase in muscle strength from a resistance training program depends on the particular resistance training program and the muscle group(s) trained. **Figure 19.4** shows the percent change in 1-RM during 11 weeks of training using two different training programs—one using 1 set of lower-body exercises and 3 sets of upper-body exercise (1L-3UB) and one using 3 sets of lower-body exercises and 1 set of upper-body exercise (3L-1UB). Notice that the percent changes were greater for the leg exercises (shown in panel A) when a greater number of sets using the lower body was used. However, for the upper-body exercises (panel B), there was no difference between groups, with both training groups increasing arm strength approximately 25% (Rønnestad et al., 2007).

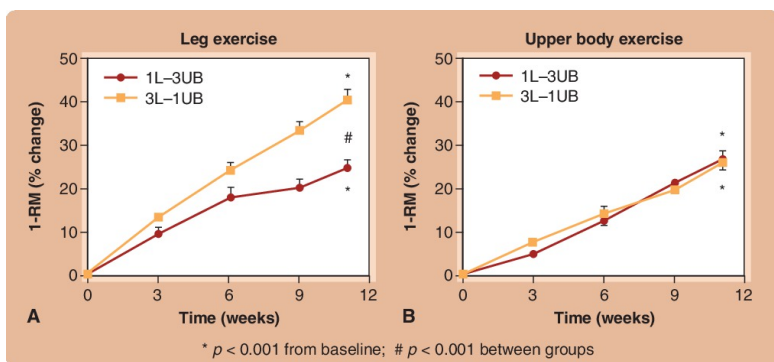


Figure 19.4 Relative Changes in 1-RM in Leg Exercise (A) and Upper-Body exercise (B) During 11-Week Training Program.

Note: 1L-3UB, 1 set of leg exercises and 3 sets of upper body exercises; 3L-1UB, 3 sets of leg exercises and 1 set of upper

body exercises.

Source: Reprinted with permission from Rønnestad, B. R., W. Egeland, N. H. Kvamme, P. E. Refsnes, F. Kadi, & T. Raastad: Dissimilar effects of one- and three-set strength training on strength and muscle mass gains in upper and lower body in untrained subjects. *Journal of Strength and Conditioning Research*. 21(1):157–163 (2007). Copyright © 2007 National Strength and Conditioning Association.

The intensity of training also affects the magnitude of adaptation. A 12-week training program that compared low-intensity (15% of 1-RM) and high-intensity (70% 1-RM) contractions while carefully equalizing the total volume of training found that 1-RM quadriceps strength increased by 36% following high-intensity contractions and by 19% with low-intensity contractions ([Holm et al., 2008](#)).

Muscle Size and Structure

Resistance training increases muscle size and strength. The hypertrophic response to resistance training is affected by the individual factors, such as genetic background, age, and gender, and by the training protocol ([Schoenfeld, 2010, 2021](#)). Although changes in muscle cross-sectional area (CSA) have been reported as early as 3 weeks after resistance training, it is likely that these early changes largely reflect muscle swelling due to muscle damage and inflammation ([Damas et al., 2015](#)). Increases in muscle cross-sectional area (CSA) of 7–15% have been reported with 10–14 weeks of resistance training when intensity greater than 60% of 1-RM was used ([Holm et al., 2008](#); [Rønnestad et al., 2007](#)). The increase in cross-sectional area of the whole muscle reflects an increase in individual muscle fiber cross-sectional area. The increased cross-sectional area of muscle results from hypertrophy of all three muscle fiber types, although the fast glycolytic (FG) fibers exhibit the greatest increase ([Bird et al., 2005](#)). The hypertrophy that occurs is due to an increase in the total contractile protein (actin and myosin), the size and number of the myofibrils per fiber, and the amount of connective tissue

surrounding the muscle fibers (Folland and Williams, 2007; Schoenfeld, 2010). Although the exact mechanisms are still unknown, there is evidence to suggest that titin and titin-related mechanosensing proteins play an important role in resistance training-induced adaptations (Ibata and Terentjev, 2021). Resistance training results in increased collagen synthesis and a strengthening of the connective tissue around the muscle. However, the ratio of connective tissue to skeletal muscle appears relatively consistent between trained and untrained individuals. There is also evidence of increased tendon stiffness with resistance training, which could enhance the rapid application of force (Folland and Williams, 2007).

Figure 19.5 presents data from a study in which muscle biopsies were taken from a group of resistance-trained (RT) and untrained (UT) men. Each major subdivision of muscle fiber type clearly had a greater cross-sectional area in the RT group than the untrained group. Furthermore, each of the muscle fiber types could produce greater force in the trained group (Shoeppe et al., 2003).

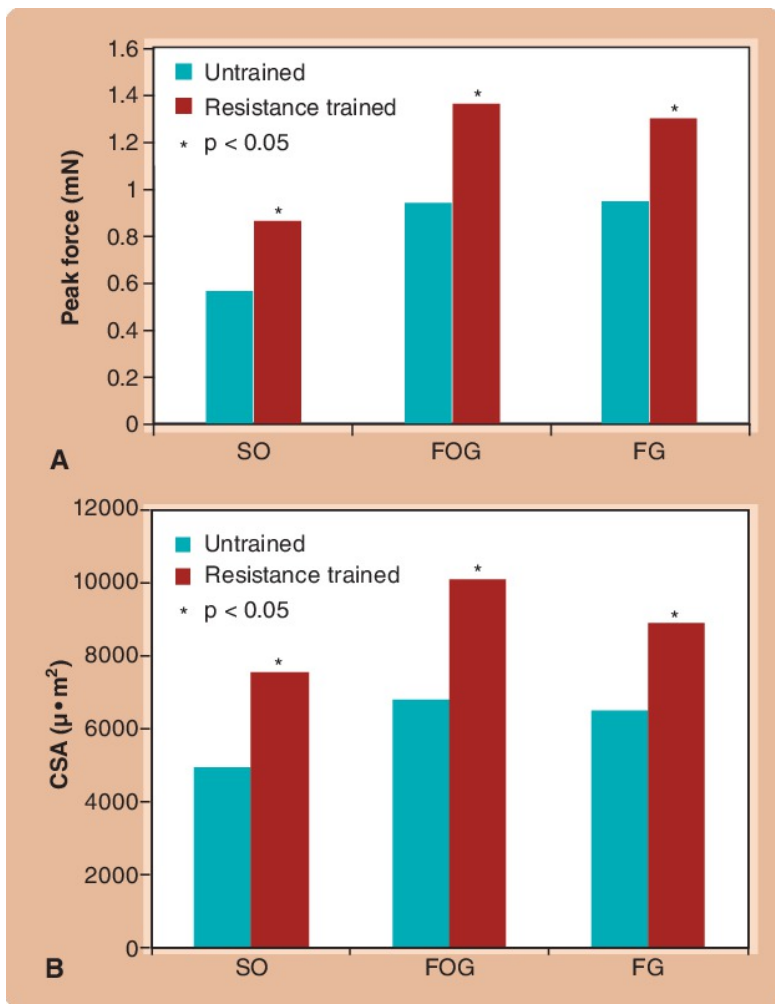


Figure 19.5 Peak Force (A) and Cross-Sectional Area (CSA) (B) of Muscle Fibers in Untrained (UT) and Resistance-Trained (RT) Individuals.

Source: [Shoepe et al. \(2003\)](#).

Resistance training causes a conversion of muscle fiber subtypes in humans. High-intensity resistance training induces a change from fast oxidative glycolytic (FOG) to fast glycolytic (FG) in the early phases of training, with transitions being complete by approximately 12 weeks of training ([Kraemer et al., 1995](#)). It

appears that intensity of training is an important stimulus for this adaptation (Holm et al., 2008).

Resistance training can theoretically lead to *hyperplasia*—an increase in number of muscle fibers. Hyperplasia could occur as a result of muscle fiber splitting or branching with subsequent hypertrophy or myogenesis or a combination of the two, but that may only be possible when fibers are extremely large, such as in anabolic steroid users (Eriksson et al., 2006). The contribution of hyperplasia to increased muscle cross-sectional area (and strength) remains controversial. However, given the magnitude of changes in muscle size attributable to hypertrophy, it seems that the contribution of hyperplasia to increased muscle cross-sectional area is minimal at best (Folland and Williams, 2007; Jorgenson et al., 2020).

Neural Adaptations

It is generally accepted that neural adaptations have a critical role in the increased force production resulting from resistance training. Resistance training programs typically result in strength gains within the first few weeks despite modest changes in muscle mass, suggesting that neural factors are largely responsible for early strength gains (Gabriel et al., 2006; Sale, 1988). **Figure 19.6** illustrates the relative contributions of neural and muscular adaptations to strength gains.

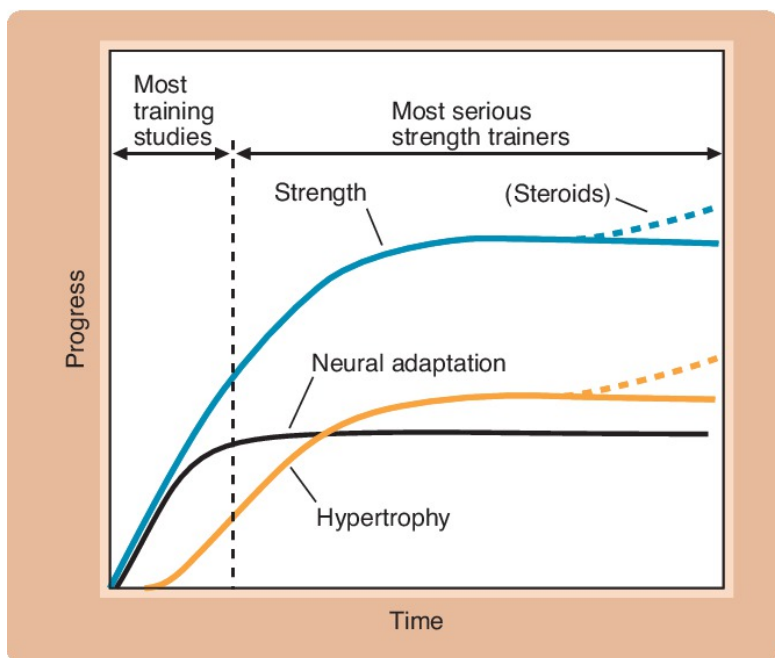


Figure 19.6 Contribution of Neural and Muscular Adaptations to Strength Gains.

Source: Reprinted with permission from Sale, D. G.: Neural adaptation to resistance training. *Medicine and Science in Sports and Exercise*. 20(5 Suppl):S135–S145 (1988).

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Neural adaptations to resistance training include increased neural drive to the muscle, increased synchronization of the motor units, and an inhibition of the protective mechanism of the Golgi tendon organs (Fleck and Kraemer, 1987; Gabriel et al., 2006). Increased neural drive indicates that greater muscle activation can occur because more motor units can be recruited. Evidence suggests that resistance-trained individuals exhibit greater synchronization—a higher correlation between timing of action potentials of concurrently active motor units that allows for greater force production. Indirect evidence also suggests that resistance training leads to changes in intermuscular coordination of agonists, antagonists, and synergists, which dampens inhibitory reflexes and allows for greater force production.

Activation of Golgi tendon organs results in inhibition of the agonist via inhibitory motor neurons, thus providing an important protective reflex that limits excessive force generation within muscle (see [Chapter 20](#)). Resistance training dampens the reflex action of the Golgi tendon organ, although it is not clear whether this occurs by changing the receptor or the neural pathways ([Gabriel et al., 2006](#)).

The effects of resistance training on neural adaptations seem to be, at least in part, influenced by the load being used for training. Jenkins et al. demonstrated that while training at both 30% and 80% of one-repetition maximum elicited similar hypertrophic growth, the training program with the heavier load exhibited greater neural adaptations. Furthermore, training with heavier loads reduced neural cost meaning they required less neural activity to produce similar torque than lighter loads ([Jenkins et al., 2017](#)).

Recent research has also sought to determine where in the nervous system these neural adaptations occur. Evidence suggests that resistance training elicits neural adaptation in several areas, including the motor cortex in the brain, the spinal cord, and the end motor units that stimulate muscle contractions. It has been speculated that neural adaptations following resistance training mainly occur at the motor neuron level, since those neurons actively stimulate muscle contraction. However, a 2020 meta-analysis concluded that neural adaptations seem to occur evenly at both the cortical and subcortical levels ([Siddique et al., 2020](#)).

Metabolic Adaptations

In addition to greater strength and hypertrophy, metabolic adaptations occur within muscle fibers that increase the ability of the muscle to generate ATP. These changes are characterized by an increased ability to generate ATP from anaerobic metabolism; thus, there is an increase in phosphocreatine (PC) and glycogen stores and an increase in the enzyme creatine phosphokinase that breaks down PC ([MacDougall et al., 1977](#)). Refer to Chapters 2 and 3 for a review of anaerobic metabolism.

Hormonal Adaptations

An acute bout of resistance exercise results in a catabolic state in which muscle proteins are broken down. During recovery, anabolism predominates, leading to muscle repair and growth. This coupled process of catabolism and anabolism is responsible for the remodeling of muscle tissue in response to resistance training. Many anabolic and catabolic processes are controlled by the neuroendocrine system, with numerous acute hormonal responses to resistance exercise. It is now generally agreed upon that acute changes in circulating anabolic hormones do not impact long-term muscle adaptations ([Morton et al., 2016](#); [West et al., 2010a](#)). Similarly, resistance training causes relatively few hormonal adaptations over time.

Published reports are not consistent about the chronic effect of resistance training on resting testosterone levels. Several studies have reported elevated resting testosterone levels, whereas many others have reported no change in resting levels of testosterone following resistance training. Similarly, findings are inconsistent regarding the effect of resistance training on resting cortisol levels. It appears that the majority of resistance training programs do not change resting growth hormone concentration, but limited amounts of data from high volume-low repetition resistance training program studies show that these types of programs may cause a more dramatic growth hormone response. Some evidence suggests that resistance training leads to an increased resting level of insulin-like growth factor (IGF-1) ([Kraemer and Ratamess, 2005](#); [Mangine et al., 2015](#)). It may be that IGF-1 secreted from muscle itself is particularly important as it functions in an autocrine manner to stimulate growth over time ([West et al., 2010b](#)).

Male-Female Resistance Training Adaptation Comparisons

Although men are typically stronger than women, both sexes respond to resistance training in a similar manner ([Bird et al., 2005](#); [Borde et al., 2015](#); [Cureton et al., 1988](#); [Ribeiro et al.,](#)

2014; Tesch, 1992). Typically, sedentary males and females can attain strength gains of 25–100% in a training program, although the actual increase in strength varies among muscle groups and is affected by the individual's initial strength (Haskell et al., 2007). **Figure 19.7** shows the percentage change in muscle strength for men and women for the elbow flexors, elbow extensors, knee flexors, and knee extensors after 16 weeks of participation in a weight training program (Cureton et al., 1988). In addition to having similar patterns of strength gains, the men and women in this study both had similar changes in muscle cross-sectional area (as determined from CT scans). The cross-sectional area of the upper-arm muscles for the men and women increased 15 and 23%, respectively, after the 16-week training program. Although the men had a greater cross-sectional area than did the women, both before and after training, in both sexes, the muscle strength and muscle cross-sectional area of the upper arm increased with training.

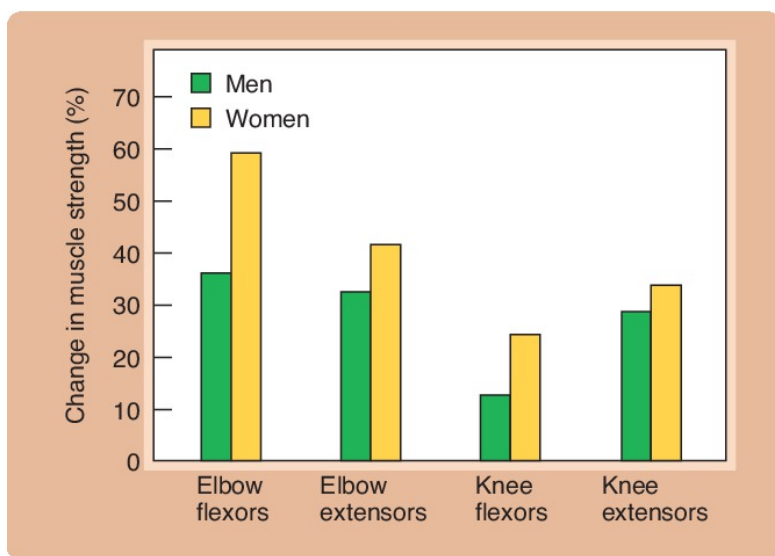


Figure 19.7 Improvement in Muscular Strength after 16 Weeks of Weight Training.

Source: Cureton et al. (1988).

A review article analyzing the results of studies in which

groups of men and women followed the same training program also found that men and women respond to resistance training with similar increases in muscle cross-sectional area. This review found that on average, muscle cross-sectional area increases an average 0.13% per day in males and 0.14% per day in females (Wernbom et al., 2007). Another study comparing the increase in training load in men and women after a 16-week resistance training program suggested that both experience similar increases in strength and body mass in response to resistance training, but women actually experienced a more significant increase in training load (Ribeiro et al., 2014).

Although men and women respond similarly to resistance training, there is evidence to suggest that there are sex-based differences in postexercise anabolic signaling. West et al., found that following a bout of resistance training, men and women experienced similar myofibrillar protein synthetic (MPS) responses. However, there were significantly greater increases in postexercise testosterone, as well as signaling molecules involved in the MPS response, in men compared to women (West et al., 2012). These results suggest that resistance training-induced increases in MPS may not be directly influenced by postexercise testosterone concentrations and that MPS is effectively stimulated in low testosterone environments of women.

Resistance Training Adaptations in Children and Adolescents

Resistance training produces strength gains in prepubescents and adolescents and is recognized as an important component of youth fitness programs (Faigenbaum et al., 2009, 2015). Unfortunately, there is mounting evidence that children are less active than in previous generations. This lack of activity can result in muscular weakness and lack of power that undermine sports participation and increase the risk for injuries. Resistance training programs have been shown to be an effective means to increase strength and improve motor performance skills in children and adolescents (Faigenbaum et al., 2015).

Some parents may deter their children from participating in

resistance training due to a fear of injury or “stunted growth.” However, evidence suggests that under supervised conditions, children who perform resistance training exercises have a lower injury risk compared to other traditional sports (Eisenberg et al., 2012). Furthermore, data suggest that the use of resistance training as a prehabilitation measure in adolescent athletes can reduce the risk of injury from other sports (Stricker et al., 2020). Finally, evidence suggests that under supervised conditions, resistance training does not negatively impact growth and maturation of pre- and early-pubertal children (Malina, 2006).

Research suggests that strength gains of approximately 30% (range 13–40%) are typical following short-term resistance training programs (up to 20 weeks) in children. Increases in strength seem fairly consistent between prepubescents and adolescents. There is no apparent difference in the relative strength (percentage) increases between boys and girls (Faigenbaum et al., 2009) nor between children/adolescents and adults (Rowland, 2005).

The strength improvement in children and adolescents is consistent with improvements documented in adults; however, the underlying physiological adaptations that account for increased strength appear to differ slightly. There seems to be general agreement that changes occur in the ability of the nervous system to activate motor units (increased muscle activation, increased activation rates, increased twitch torque, and a reduction in electromechanical delay) as well as improved coordination, which contribute to the increased strength in both adults and children. However, it is generally agreed that such neural adaptations are the predominant mechanism responsible for exercise-induced strength and power increases (rather than muscle morphological changes) in children (Faigenbaum et al., 2009; Legerlotz et al., 2016; Rowland, 2005).

Resistance training does not appear to induce muscle fiber hypertrophy in preadolescents, possibly because of the lack of testosterone. Studies investigating prepubertal children have shown increases in muscle size in both experimental and control groups, suggesting that hypertrophy experienced during the prepubertal stage of childhood and adolescence is attributable primarily to biological maturation (Cunha et al., 2015). However,

there is also newer evidence that morphological adaptation of muscles and tendons occurs, specifically increased cross sectional areas and fascicle length. Cross-sectional area is proportional to strength and muscle fascicle length is proportional to contraction velocity. Together the product is power. These changes undoubtedly contribute, along with the more predominant neural changes, to training-induced improvements in muscular activities in children and adolescents ([Legerlotz et al., 2016](#)).

Resistance Training Adaptations in Older Adults

Resistance exercise is widely acknowledged as beneficial for older adults, and many agencies actively promote resistance exercise for this age group (ACSM, 2022; [Nelson et al., 2007](#)). Resistance training is also safe and effective for increasing muscular strength and cross-sectional area in older individuals ([Aagaard et al., 2007](#); [Charette et al., 1991](#); [Csapo and Alegre, 2016](#); [Frontera et al., 1991](#); [Nelson et al., 2007](#)). Because of the ability of resistance training to increase muscle mass, strength, and function, it is recognized as an appropriate way to compensate for age-related decline in muscle mass (sarcopenia) and combat other degenerative age-related disorders ([Ciolac and Rodrigues-da-Silva, 2016](#); [Power et al., 2013](#); [Watanabe et al., 2014](#)). In fact, older men and women have similar or even greater strength gains than young individuals from resistance training. Resistance training is a recommended component of fitness programs for older adults and is considered important for minimizing or reversing physical frailty, which is prevalent among the elderly (ACSM, 2022; [Kraemer and Ratamess, 2004](#)). Chronic resistance training over a long period results in greater strength, rate of force development, and muscle fiber cross-sectional area than is evidenced in untrained individuals ([Aagaard et al., 2007](#)). Short-term resistance training programs are also successful in leading to muscular adaptations in the elderly. **Figure 19.8** shows the improvements in dynamic strength of the knee extensors (quadriceps) and the knee flexors (hamstrings) following 12 weeks of training in older men ([Rogers and Evans, 1993](#)). By the

end of the training period, strength in both muscle groups had increased over 100%. Additionally, these men demonstrated an 11% increase in muscle cross-sectional area, accompanied by a 34% increase in ST fiber area and a 28% increase in FT fiber area. Similar improvements have been reported in women. Bacelar et al. had sedentary women (aged 64 ± 3 years) perform 20 sessions of resistance training program over 10 weeks to examine the effects on the participants' muscle strength and bone mineral density. They found a significant increase in 10-repetition maximal bench press and leg press and no significant negative changes in lumbar spine and femoral neck bone mineral density. The results suggest that resistance training in elderly females is an effective method to increase muscle strength and maintain bone mineral density (Bacelar et al., 2015). A meta-analysis investigating the dose-response relationships of resistance training in healthy older adults found that resistance training improved muscle strength substantially but had small effects on measures of muscle size. The study also found that the length of training period had the greatest effect on muscle strength (Borde et al., 2015).

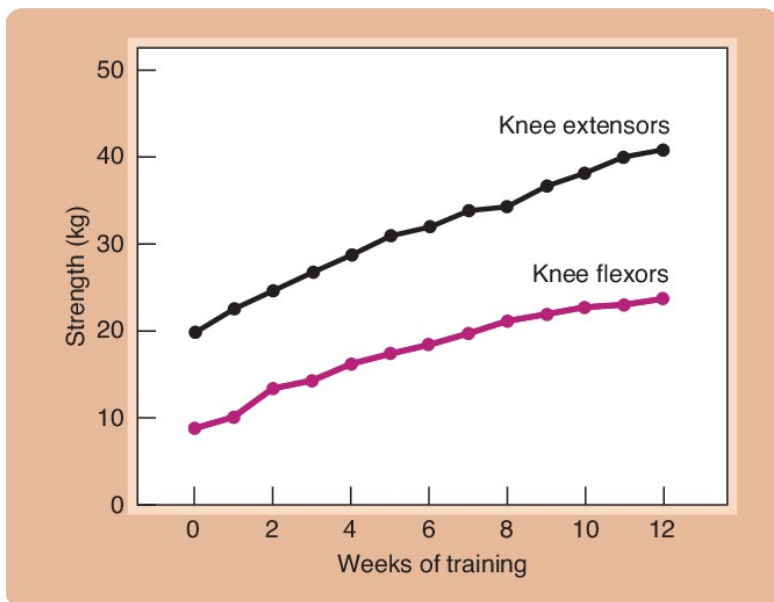


Figure 19.8 Effects of Dynamic Resistance Exercise on

Muscular Strength in Older Men.

Source: Reprinted with permission from Rogers, M. A., & W. J. Evans: Changes in skeletal muscle with aging: Effects of exercise training. In Holloszy, J. O. (ed.): *Exercise and Sport Sciences Reviews* (Vol. 21). Baltimore, MD: Williams & Wilkins, 65–102 (1993).

Resistance training is also beneficial for frail, institutionalized men and women ([Fiatarone et al., 1990](#)). A group of 90- to 100-year-old men and women who engaged in a resistance training program for 8 weeks increased their strength 174% (from an average initial value of 8–21 kg). Their muscle cross-sectional area increased 15%. These improvements demonstrate rather remarkably the capacity of the muscular system to adapt to progressive resistance exercise as long as a person is willing to participate in a program at an appropriate intensity.

Most studies that have investigated the effects of resistance training in older adults have relied on participants who were not obese. Recently, researchers extended these findings by investigating the effects of resistance training in obese individuals. This is particularly important as many individuals enter old age with excess adiposity and obesity may limit the ability to perform muscular work. The authors studied 126 older adults (age 65–79 years) who were overweight or obese and found that resistance training improved body composition, muscle strength, and physical function in obese elderly, but those with higher levels of initial adiposity experienced less improvement ([Nicklas et al., 2015](#)). These findings highlight the important interaction among systems (metabolic and neuromuscular) and reinforce the need to find ways to help individuals maintain a healthy weight.

Figure 19.9 summarizes the results of several studies that investigated changes in muscle strength and cross-sectional area that occur in the quadriceps of males and females of various ages after a resistance training program. These values indicate a percentage change from the initial values; they do not imply that females are stronger than males as adults nor that the elderly are stronger than the younger subjects. In fact, those who are

weakest initially may be in the best position to show a high percentage improvement.

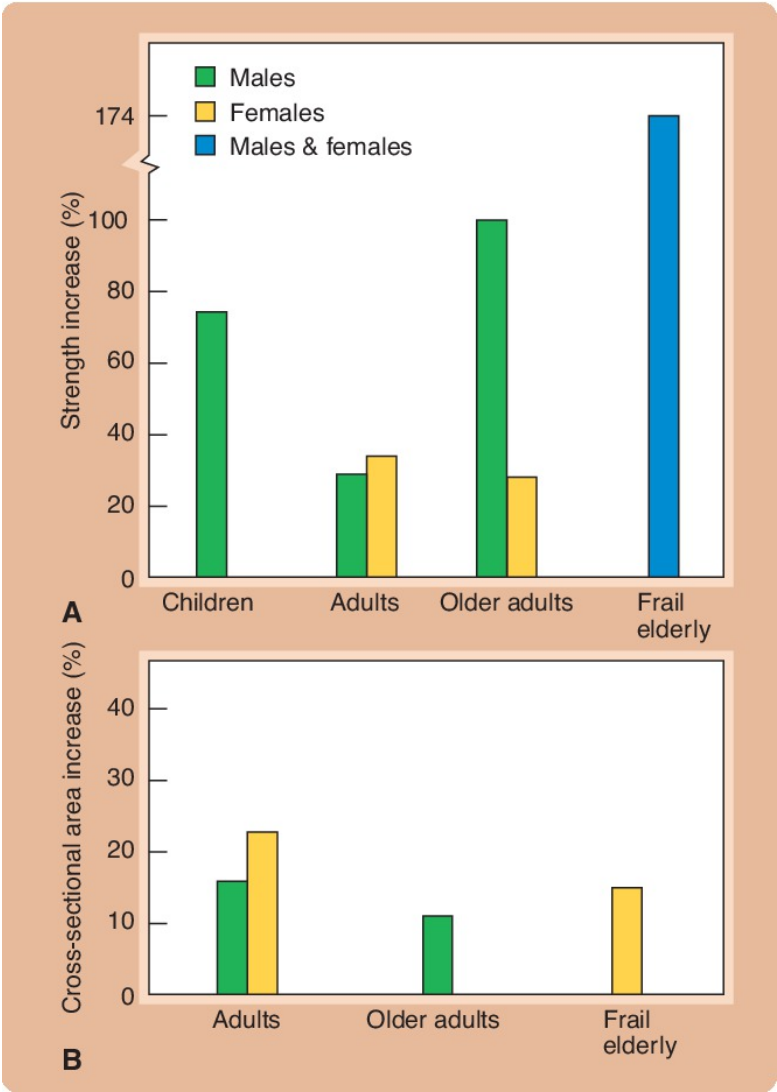


Figure 19.9 Strength and Cross-Sectional Area Increases in Quadriceps Following Resistance Training.

A. Strength increase. B. Cross-sectional area increase.

Sources: Charette et al. (1991); Cureton et al. (1988); Faigenbaum et al. (1993); Fiatarone et al. (1990); Frontera

et al. (1991).

Muscular Adaptations to Aerobic Endurance Training Programs

Muscle fibers respond differently to aerobic training than to resistance training. Aerobic training is characterized by increased aerobic power ($\dot{V}O_2 \text{ max}$) with little or no change in muscle strength or power. Similarly, the structural and metabolic changes in muscle fibers facilitate the production of large quantities of ATP, primarily by aerobic means, following an aerobic training program, thus enhancing muscular endurance.

Aerobic endurance training results in an increase in ST fiber size in adult males and no change in FT fiber size (Gollnick et al., 1972). There is also evidence that aerobic endurance training can result in the transformation of FG muscle fibers to FOG muscle fibers (Fleck and Kraemer, 1987). Aerobic endurance training in older men and women also results in an increase in the cross-sectional area of the ST fibers (averaging 12%) and an increase in the percentage of FOG fibers (Coggan et al., 1990). Data have also shown that a 12-week cycle ergometer aerobic training protocol significantly increased ST fiber size in the vastus lateralis by 16% and 20% in old and young women, respectively (Harber et al., 2009, 2012).

Muscular Adaptations to Concurrent Training

Many sports require a combination of muscular strength and aerobic endurance, and thus, a combination of resistance and aerobic endurance training is required to improve performance. The integration of endurance- and resistance-based training into a training program is called **concurrent training**. The most consistent finding from studies of concurrent training is that increases in strength and power are lower than with strength

training alone but changes in aerobic fitness are only slightly lower than those found with aerobic training alone. However, research findings are not consistent. Other studies have shown that concurrent training has no inhibitory effect on the development of strength or aerobic endurance. Finally, at least one study has shown that the development of aerobic fitness but not strength is compromised by concurrent training (Leveritt et al., 1999; Nader, 2006). The contradictory findings from concurrent training studies are likely due to methodological differences such as the training status of participants, as well as the training protocol itself. For example, the order in which exercises are performed may influence concurrent training adaptations. Murlasits et al., reported that when resistance training was performed prior to endurance training, lower body 1-RM was significantly higher (~4 kg), compared to when endurance training was performed prior to resistance training. However, exercise order did not influence aerobic adaptations suggesting that perhaps, athletes performing concurrent training should begin with resistance training that is followed by aerobic training rather than vice versa (Murlasits et al., 2018). Furthermore, some data show that performing resistance training prior to aerobic training results in a more favorable postexercise hormonal profile. Those who performed aerobic exercise prior to resistance training experienced a significantly higher increase in cortisol and lower testosterone postexercise compared to the opposite exercise order (Jones et al., 2017). Although postexercise hormone fluctuations have not been strongly correlated to exercise-induced adaptations, this is yet another potential reason to perform resistance training prior to aerobic training in most concurrent training protocols. Aside from exercise order, another major consideration to keep in mind when considering adaptations to concurrent training is the type of athlete performing concurrent training. For example, an endurance athlete will benefit from including some resistance training. However, a power athlete may not benefit as much by including endurance exercise.

Concurrent Training The integration of endurance- and resistance-based training into a training program.

Several mechanisms have been suggested to explain the more prevalent finding of inhibition of strength during concurrent training. If individuals do high-intensity training in both modalities, overtraining may be responsible. The chronic hypothesis contends that skeletal muscles cannot adapt to both types of training at the same time because many of the adaptations shown in **Table 19.3** are different. The acute hypothesis contends that residual fatigue from the aerobic endurance component compromises the ability to work as hard as necessary during the resistance training. While both of these hypotheses are intuitively reasonable, only limited evidence exists for either one (Leveritt et al., 1999). Newer data highlight potential molecular mechanisms (Hawley, 2009). It is now clear that different modalities of exercise cause different intracellular signaling mechanisms; endurance training elicits increases in mitochondrial content and respiratory capacity of muscle fibers and resistance training initiates a cascade of events leading to increased synthesis of muscle contractile protein. The stimulation of the different pathways may result in inhibition or interference with the other pathway (Hawley, 2009; Nader, 2006).

Periodization programs that emphasize one or the other type of training throughout the training year rather than concurrent high-intensity training in both modalities may be very important for optimizing both muscular and aerobic endurance adaptations for athletes in sports requiring high levels of both. Training for fitness typically involves exercise sessions on different days for the different modalities, as opposed to true concurrent training. Also peak levels of performance are not generally the goal. Therefore, inhibition of one type of training by the other is not typically a major concern.

Special Application: Muscular Strength/Endurance and Health

Athletes have long used resistance training programs to increase muscular function and to improve performance. There is now increasing evidence for both children/adolescents and adults of all ages that enhanced neuromuscular fitness, especially muscular

strength/endurance, is also associated with an improvement in overall health status and, conversely, a reduction of risk for chronic disease, disability, and, in adults and adolescents, mortality ([Institute of Medicine, 2012](#); [Liu and Lee, 2019](#); [Ortega et al., 2012](#); [Plowman, 2013](#); [Smith et al., 2014](#); [Warburton et al., 2006](#)).

Mortality rates have been found to be lower in adult males and females when individuals with moderate/high muscular fitness (primarily measured by handgrip strength, sit-ups, leg and bench press) were compared with individuals with low muscular fitness, even after adjusting for cardiorespiratory fitness, body composition, and other potentially confounding variables ([FitzGerald et al., 2004](#); [Katzmarzyk and Craig, 2002](#)). In one large-scale study, researchers assessed muscular fitness in over 9,100 men and women between the ages of 20 and 82 years over an 8-year period ([FitzGerald et al., 2004](#)). Muscular fitness was based on a composite score from a maximal bench press, maximal leg press, and the number of sit-ups completed in 1 minute. For each individual test, participants were divided into tertiles, and a combined score from the three tests was used as an indicator of muscular fitness—defined as low, medium, or high. A mortality follow-up was conducted 7 years after the completion of the muscular fitness evaluation, revealing that 194 participants had died since their evaluation. A statistical analysis was performed comparing the relative risk of all-cause mortality across all three groups in which the relative risk was set at 1.0 for the low muscular fitness group. Participants in the moderate and high muscular fitness groups had a relative risk of 0.64 (95% CI = 0.44–0.93) and 0.80 (95% CI = 0.49–1.31) compared with the low muscular fitness group after statistically adjusting for differences in age, health status, body mass index, cigarette smoking, and cardiorespiratory fitness. In a related study, high muscular strength in male adolescents (females were not studied as the participants were Swedish military conscripts), as assessed by knee extension and handgrip tests, was associated with a 20–25% lower risk of premature mortality due to any cause or cardiovascular disease ([Ortega et al., 2012](#)). More details about this important study can be found in the accompanying Focus on Research box.

FOCUS ON RESEARCH

Relationship between Muscular Strength and Cardiovascular Disease Mortality

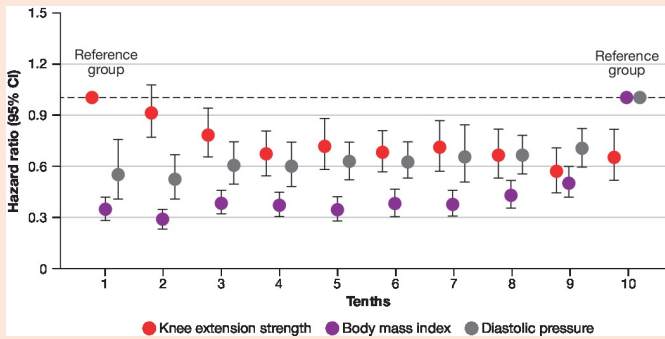
Understanding which factors contribute to premature mortality, defined as death before the age of 55 years, may help early treatment of disease or even prevent disease from developing. For example, as described in [Chapter 15](#), there are several risk factors associated with the development of cardiovascular disease (CVD), including cigarette smoking, high cholesterol fractions, and physical inactivity. By decreasing the number of CVD risk factors an individual possesses, his or her likelihood of death from CVD is diminished. You are likely aware that research has found that obesity, hypertension, and low cardiorespiratory fitness are powerful predictors of premature mortality due to CVD. The influence of muscular strength on cardiovascular disease mortality is less obvious and less well studied. However, one large-scale study provides compelling data.

In this study, the authors examined the influence that muscular strength during adolescence has on all-cause mortality and mortality due to CVD (coronary heart disease and stroke). Just over 1 million 16- to 19-year-old adolescent males were included in the study. Baseline measures of muscle strength (knee extension and handgrip strength), body mass index (BMI), and systolic and diastolic blood pressure (SBP and DBP, respectively) were recorded. Participants were followed until death, date of emigration, or the end of the follow-up period, whichever came first. The median follow-up period was 24.2 years.

Approximately 2.3% of the participants died throughout the follow-up period, 7.8% of which were due to CVD (coronary heart disease or stroke). Suicide, cancer, accidents, and other causes of mortality accounted for the remainder of deaths during follow-up.

For analysis, participants were divided into tenths to assess how incremental increases in muscular strength influenced the likelihood of all-cause mortality and CVD mortality. Compared with the weakest group (first tenth), greater muscular strength in the second, third, and fourth tenth groups resulted in a reduction in all-cause mortality (not shown). Mortality risk plateaued around a 20% reduction from the fourth tenth to the last tenth (strongest). Muscular strength alone was not associated with premature cardiovascular mortality; however, when adjusted for BMI and DBP, high muscular strength groups reduced their risk of cardiovascular mortality by 35% (see accompanying figure). These findings suggest that low muscular strength during adolescence may be a valid predictor of premature death from all-cause mortality and CVD mortality, suggesting that interventions target those with strength levels lower than the population average. Greater levels of muscular strength do not appear to provide additional protection. Furthermore, the study indicates that risk of premature death is a complex issue, affected by multiple body systems (muscular, metabolic, and cardiovascular) working in concert.

The importance of muscular strength on CVD mortality has also been demonstrated in an occupationally active cohort of firefighters. Data from 1104 firefighters showed that the total number of pushups that an individual can perform is negatively correlated with cardiovascular disease risk within a 10-year follow-up. Those firefighters who were able to perform more than 40 pushups had a significantly lower risk of CVD incidence compared to individuals who were able to perform less than 10 pushups.



Sources: Reproduced from Ortega, F. B., K. Silverton, P. Tynelius, & F. Rasmussen: Muscular strength in male adolescents and premature death: Cohort study of one million participants. *British Medical Journal (Online)*. 345:e7279 (2012), with permission from BMJ Publishing Group Ltd.; Yang J., C. A. Christophi, A. Farioli, D. M. Baur, S. Moffatt, T. W. Zollinger, & S. N. Kales: Association between push-up exercise capacity and future cardiovascular events among active adult men. *Journal of the American Medical Association Network Open*. 2(2):e188341 (2019).

Muscular strength/endurance seems to be of particular importance in older populations and has been shown to be an independent risk factor of quality of life, function, and risk of all-cause mortality. A 2011 study, which included NHANES data from more than 1,200 adults over the age of 55 years, found that leg strength is an independent predictor of physical function (Bouchard et al., 2011). Furthermore, another study included NHANES data from more 4,000 adults over the age of 50 years and found that lower-body strength was an independent predictor of all-cause mortality among this population (Li et al., 2018). These data suggest that improving muscle strength is not only important during early- and mid-life but in aging populations in order to maintain physical function and potentially extend lifespan.

Muscular fitness has also been shown to have a positive influence on cardiovascular risk factors (Garcia-Hermoso et al., 2019; Magnussen et al., 2012; Ortega et al., 2008; Smith et al.,

2014; Warburton et al., 2001a). Specifically, resistance training has been shown to lower LDL-C, raise HDL-C, and reduce both SBP and DBP (Phillips and Winett, 2010; Strasser et al., 2010).

Several lines of research indicate the positive effects of resistance training on metabolic health. Substantial improvements in blood glucose, glycosylated hemoglobin (an index of glucose control), insulin sensitivity, and insulin stabilization have been reported in individuals with diabetes who have weight trained (Phillips and Winett, 2010; Strasser et al., 2010). Evidence is also emerging that indicates a positive impact of neuromuscular fitness on metabolic syndrome/metabolic health risk factors in both adults (Churilla et al., 2012; Strasser et al., 2010) and youths (Artero et al., 2011; Benson et al., 2008; Castro-Piñero et al., 2019; Smith et al., 2014; Steene-Johannessen et al., 2009). This relationship appears to operate independently of, or in addition to, cardiorespiratory fitness and/or body mass/body composition. A large, multicenter European study examined the independent associations of muscular and cardiorespiratory fitness with clustered metabolic risk in over 700 adolescents (aged 12.7–17.5) (Artero et al., 2011). In this study, adolescents in the lowest muscular fitness quartile (based on handgrip score) had a significantly elevated metabolic risk compared to all other fitness quartiles. Additional analysis revealed that the participants who were in the lowest quartile for muscular fitness and the lowest quartile for cardiorespiratory fitness had a significantly higher metabolic risk score than all other groups.

High levels of muscular strength and muscular endurance and/or resistance training improvements have also been shown to positively impact or predict long-term changes in body composition (Institute of Medicine, 2012; Smith et al., 2014; Strasser et al., 2010; Warburton et al., 2001b, 2006). In general, resistance training programs are associated with a decrease in fat mass and visceral adipose tissue (a component of the metabolic syndrome). Because resistance training decreases fat mass and increases lean mass, it results in positive health benefits even in the absence of overall changes in body weight.

The evidence for a positive association between muscular fitness and bone health is strong in children and adolescents (Smith et al., 2014). The optimal prevention strategy for

maintenance of bone health in adults is the attainment of a strong, dense skeleton during the growth years. Despite a large genetic contribution to bone mass, resistance and high-impact exercise can contribute an additional 5–15% to bone formation (Boreham and Riddoch, 2001; Faigenbaum et al., 2009).

In addition to the evidence of a role of resistance training in improving cardiometabolic health and body composition, there is also extensive literature indicating that muscular fitness is an important determinant of quality of life, including measures of functional status and psychological well-being.

Because of the numerous benefits of muscular fitness, many organizations are promoting resistance training as a part of every complete fitness program. In fact, as described in the preceding paragraphs, there are many well-documented health benefits of resistance training programs, some of which are summarized in **Table 19.4**.

TABLE 19.4 Comparison of Adaptations to Aerobic and Resistance Training

| Variable | Aerobic Training | Resistance Training |
|---------------------------------------|------------------|---------------------|
| Functional and Structural | | |
| Physical endurance | ↑↑↑ | ↑↑ |
| Strength | ↔ | ↑↑↑ |
| Bone mineral density | ↑ | ↑↑↑ |
| Metabolic | | |
| Body composition | | |
| Fat mass | ↓↓ | ↓ |
| Muscle mass | ↔ | ↑↑ |
| Basal metabolism | ↑ | ↑↑ |
| Glucose metabolism | | |
| Insulin response to glucose challenge | ↓↓ | ↓↓ |
| Basal insulin levels | ↓ | ↓ |
| Insulin sensitivity | ↑↑ | ↑↑ |
| Serum lipids | | |
| High-density lipoprotein | ↑↔ | ↑↔ |
| Low-density lipoprotein | ↓↔ | ↓↔ |
| Cardiovascular | | |
| Resting heart rate | ↓↓ | ↔ |
| Blood pressure at rest | | |
| Systolic | ↓↓ | ↓↓ |
| Diastolic | ↓↓ | ↓ |

Source: Reprinted with permission from Braith, R. W., & K. J. Stewart: Resistance exercise training: Its role in the prevention of cardiovascular disease. *Circulation*. 113(22):2642-2650 (2006).

Special Application: Muscular Strength and Endurance and Low Back Health

To have a healthy, well-functioning back, an individual must have flexible low-back (lumbar) muscles, hamstrings, and hip flexors and strong, fatigue-resistant abdominal and back extensor muscles. The goal is to keep the vertebrae aligned properly without excessive disk pressure, allowing a full range of motion in all directions. In addition, the pelvis must freely rotate both posteriorly and anteriorly without straining the muscle or fascia (**Table 19.5**).

TABLE 19.5 Theoretical Relationship between Physical Fitness Components and Healthy or Unhealthy Low Back or Spinal Function

| Physical Fitness Component (Neuromuscular) | Normal Anatomical Function in Low Back: Healthy | Dysfunction | Results of Dysfunction: Unhealthy |
|--|---|-----------------------|---|
| Lumbar flexibility | Allows the lumbar curve to almost be reversed in forward flexion | Inflexible | Disrupts forward and lateral movement; places excessive stretch on hamstrings, leading to low back and hamstring pain |
| Hamstring flexibility | Allows anterior rotation (tilt) of the pelvis in forward flexion and posterior rotation in the sitting position | Inflexible | Restricts anterior pelvic rotation and exaggerates posterior tilt; both cause increased disk compression; excessive stretching causes strain and pain |
| Hip flexor flexibility | Allows achievement of neutral pelvic position | Inflexible | Exaggerates anterior pelvic tilt if not counteracted by strong abdominal muscles, thereby increasing disk compression |
| Abdominal strength or endurance | Maintains pelvic position; reinforces back extensor fascia and pulls it laterally on forward flexion, providing support | Weak, easily fatigued | Allows abnormal pelvic tilt; increases strain on back extensor muscles |
| Back extensor strength or endurance | Provides stability for the spine; maintains erect posture; controls forward flexion | Weak, easily fatigued | Increases loading on the spine; causes increased disk compression |

Unfortunately, as illustrated in **Figure 19.10** at some point in their lives, 60–80% of all people experience low-back pain (LBP). The condition is disabling in 1–5% of the population. In 2018 (the latest year for which statistics were available), 28% of males and approximately 32% of females ≥ 18 years of age reported LBP within the last 3 months of taking the National Health Interview Survey ([NHIS, 2018](#)). For males, the percentage increased with age through 74 years and then decreased. For females, the percentage increased throughout the age span. The percentage of females experience LBP exceeded that of males in all age categories except 65–74 years.



Figure 19.10 Approximately 60–80% of the Population Experience Low Back Pain (LBP) at Some Point.

The exact causes and risk factors for LBP have not been identified. However, there has been great interest in the link between muscular fitness and the absence or occurrence of LBP. Some tests of health-related physical fitness have included sit-and-reach, sit-ups or curl-ups, and trunk extension tests as means of testing low back function. The theoretical link between physical fitness and LBP is largely based on functional anatomy, and at this time, the anatomical logic is stronger than the research evidence.

Research evidence shows that individuals suffering from LBP have less strength in both abdominal and back extensors. EMG fatigue indices are also increased in the back muscles of individuals with LBP. These differences, however, are more likely to be the result of LBP rather than the cause. Studies that have attempted to predict who might get LBP, either for a first time or in recurrent episodes, based on strength and muscular endurance measures have identified back extension endurance as the critical variable. That is, individuals with low levels of back extension endurance are more likely to develop LBP than individuals with high levels of back extensor endurance (Steele et al., 2014). Although high levels of back extensor strength and abdominal

flexion strength or endurance have not been shown to have this same predictive value, they have never been shown to be detrimental. Thus, a total body workout for strength and muscular endurance should include exercises for the back and abdominals, even though such a program does not absolutely protect individuals from LBP (Plowman, 1992, 2013).

FOCUS ON RESEARCH *Clinically Relevant*

Inspiratory Muscle Training in Rehabilitation of Low Back Pain

Although linking inspiratory muscle training (IMT) and low back pain may seem counterintuitive initially, there is strong anatomical logic for this. Respiratory muscle activities, including the diaphragm, erector spinae (ES), multifundus (MF), rectus abdominis (RA), and transverse abdominis (TVA), play an important role in spinal stability. Of all the core muscles, the TVA has the widest and closest connection to the thoracolumbar fascia and is essential for spinal stability. Respiratory muscle training and equipment is described in [Chapter 10](#). The purpose of the current study was to determine the effectiveness of 8 weeks of IMT on core muscle activity, pulmonary function, and pain intensity in athletes with chronic low back pain (CLBP).

All participants were weightlifting and powerlifting athletes (18–25 years old) with CLBP. All had been lifting for at least 3 years, three times per week, 75 minutes per session. This training continued throughout the study. Their CLBP was classified as not severe on the visual analog scale (VAS) but was persistent for approximately 6 months. None were using pain medication or receiving other physical treatment for their CLBP for the duration of the study. A total of 48 athletes participated with 12 M and 12 F each randomized into either the experimental or control group. One experimental subject did not complete the study.

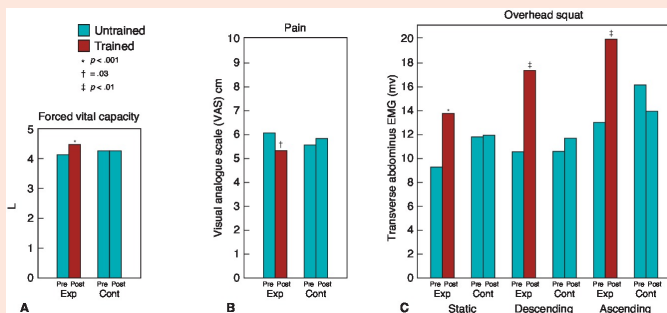
The experimental group performed IMT for 8 weeks, twice

daily, 7 d·wk⁻¹ using specific respiratory resistance equipment. Training began at 50% of maximum inspiratory pressure and increased progressively by 5% per week to a maximum of 90%. Each session consisted of 30 breaths with a breathing frequency of 15 per minute. The control group did no respiratory training.

Electromyograph (EMG) data were collected for the ES, MF, TVA, and RA during static and dynamic performance of the overhead squat. The overhead squat is a functional movement screening method used in rehabilitation that requires strength of the trunk and shoulder muscles, balance, and coordination in the entire kinetic chain.

The effect of the IMT (between groups) was analyzed using an analysis of covariance where the pretest scores were used as the covariates. Respiratory function was significantly better in the experimental group than the control group after training for all respiratory variables. As an example, forced vital capacity (FVC) is shown in panel A. However, the primary outcome was the increase in the activity of the core muscles in both the static and dynamic (ascending and descending phases) squat tests. Eleven of the 12 results (four muscles, 3 conditions) were statistically and clinically significantly better in the experimental than the control group. Only the values for TVA are presented in panel C. Pain intensity (panel B) decreased only in the experimental group and was statistically different from the control group after inspiratory muscle training.

These IMT exercises are easy and accessible and can be recommended to improve not just respiratory function but also increase core stability and reduce pain intensity in individual with moderate CLBP.



Source: Ahmadnezhad, L., A. Yalfani, & B. G. Borujeni: Inspiratory muscle training in rehabilitation of low back pain: A randomized controlled trial. *Journal of Sport Rehabilitation*. 28:1151–1158 (2020).

The strongest evidence for the importance of anatomically specific exercises for low back health comes from the rehabilitation of individuals suffering from LBP. Acute back pain is pain that remains for less than 6 weeks. After 6 weeks, the pain is considered to be chronic. LBP is classified as specific when the cause can be determined (as in an acute injury like a fracture) and as nonspecific when the cause of the back pain is unknown. Nonspecific LBP accounts for approximately 85% of all back pain cases. Most individuals with nonspecific LBP recover in 4–6 weeks with or without physical treatment of any kind, but, of course, no one knows at the outset of LBP whether it will be an acute or chronic condition. Four systemic review articles on exercise and LBP have described the value of exercise for pain reduction. First, research data reviewed by [Carpenter and Nelson \(1999\)](#) supported the following exercise prescription for individuals who suffer from CLBP: resistance exercise of lumbar extensors with the pelvis stabilized, 1 set of 8–15 repetitions to fatigue, 1 d·wk⁻¹. Patients who demonstrated the greatest gains in muscle strength also experienced the greatest decrease in pain. Second, [Gordon and Bloxham \(2016\)](#) found that a general exercise program that combined core muscular strength (to assist in supporting the lumbar spine), flexibility of the muscle-tendons and ligaments in the back (to increase the range of motion and

assist with functional movement), and aerobic exercise (to increase blood flow and nutrient to the soft tissues in the back thus reducing stiffness) was beneficial in the rehabilitation of nonspecific LBP. Third, Müller et al. (2020) concluded that targeted exercises of the spine-stabilizing musculature at moderate to high intensities were effective and financially efficient in reducing chronic LBP and the cost of treatment. Finally, Tataryn et al. (2021) revealed that posterior-chain resistance training (focused on the thoracic, lumbar, and hip extensor musculature) was more effective than general exercise for chronic sufferers in increasing strength, reducing pain and reducing the level of disability.

The Focus on Research: Clinically Relevant box presents a study on the use of specific inspiratory muscles exercise training that can be used alone or with specific back muscle and/or aerobic training in LBP rehabilitation.

Summary

1. Resistance training is used to improve overall health, improve athletic performance, rehabilitate injuries, and change physical appearance. Resistance training is also the primary activity in the sports of power lifting and bodybuilding.
2. A plan for muscular fitness should be specific to the individual's goals, which may include the development of muscular strength, hypertrophy, power or endurance, or any combination of these properties.
3. Overload of the muscular system is achieved by manipulating the load, volume, rest intervals, and frequency of training. Volume is determined by load (intensity) and repetitions ($\text{volume} = \text{load} \times \text{reps} \times \text{sets}$).
4. If different individuals use the same training program, adaptation will occur at different rates because of individual differences in age, body size and type, initial strength, and genetic makeup.
5. A coach or exercise leader must be sensitive to the

differences in rates of individual adaptation, which directly affect the progression of training. A common mistake is for coaches to design a program for the entire team and expect adaptations to occur at the same rate.

6. Adaptation to resistance training in children and older adults is very similar to adaptations that occur in young and middle-aged adults.
7. Periodization within a training program can help prevent the inhibitions of strength gains during concurrent training.
8. Strength gains are preserved for longer periods of time compared to other training adaptations. The impact of detraining on muscle strength varies by muscle group and exercise type.
9. Resistance training provides multiple health benefits, including decreased mortality, improved cardiometabolic health, and improved functional measures.
10. Functional back health requires flexibility in the lumbar muscles, hamstrings, and hip flexors and strength of the abdominals and back extensor muscles.

Review Questions

1. Give several reasons why an individual may engage in a resistance training program and specify different goals of such programs.
2. Discuss how each of the training principles can be applied in the development of a resistance training program. How do these applications vary if the exerciser is a child?
3. Is there an ideal number of repetitions and sets that should be performed by everyone? Defend your answer.
4. Discuss the importance of adequate recovery time for training adaptations to a resistance training program.
5. Do all individuals respond to a training program with the same adaptation (or magnitude of adaptation)? Why or why not?
6. What is the importance of a warm-up period before

resistance training?

7. Compare and contrast the training adaptations that occur in skeletal muscle as a result of resistance training and endurance training.
8. Describe the health benefits of muscular strength and endurance training.
9. What is the relationship between muscle function and low back health? What role does exercise play in LBP rehabilitation?
10. Identify three research questions about resistance training for which scientists have yet to provide answers.
11. Discuss the major differences in strength adaptations between children and adolescents compared to adults.
12. Discuss how the order of training (e.g., resistance training first vs. aerobic training first) in concurrent training programs may affect training-induced adaptations.
13. Explain the similarities and differences in training adaptations from low- versus high-load resistance training protocols.

Literature Search

In this chapter, we discussed muscular training principles and adaptations. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search muscular training, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Muscular training and hypertrophy.
 - ii. Muscular training and power.
 - iii. Muscular training and children.
 - iv. Muscular training and disease.
 - v. Continue your search for aspects of this topic that are of

particular interest to you.

For further review and study tools, visit Lippincott Connect.

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20 Neuromuscular Aspects of Movement



CHAPTER OUTLINE

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The Nervous System

The Basic Structure of the Nervous System

Activation of the Nervous System

The Nerve Cell

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Specificity

Overload

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Individualization

Maintenance

Retrogression/Plateau/Reversibility

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Progression

Individualization

Maintenance

Retrogression/Plateau/Reversibility

Warm-Up and Cooldown

Adaptation to Balance Training

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Describe the nerve supply to muscle.
- Describe the sequence of events at the neuromuscular junction.
- Identify the components of a reflex arc.
- Describe the structure and innervation of the muscle spindle, and explain how the muscle spindle functions in the myotatic reflex.
- Describe the structure and innervation of the Golgi tendon

organ, and explain how it functions in the inverse myotatic reflex.

- Provide research and clinical evidence that individual motor units can be volitionally controlled.
- Diagram the sequence of events involved in volitional control of movement.
- Differentiate between dynamic and static flexibility.
- Identify the anatomical factors that influence flexibility.
- Describe the basic methods of measuring flexibility.
- Discuss the relationship between flexibility and low back pain.
- Differentiate among the different types of flexibility training.
- Describe the acute responses to static stretching.
- Identify the benefits of a balance training program.
- Apply the training principles to the development of a flexibility program and a balance program.

Introduction

As spectators watching the Olympic Games, we marvel at the grace and skill of figure skaters and stare in amazement at the incredible feats of gymnasts. As adults, we look on with wonder as a child learns a new task—rolling over, walking, tying a shoe—or an injured individual relearns basic skills. As coaches or fitness leaders, we experience the satisfaction of seeing individuals incorporate our suggestions to improve their skill. Understanding the awe-inspiring accomplishments of athletes and the simple movements that are often taken for granted requires knowledge of the nervous system.

The nervous system is made up of the brain, spinal cord, and nerves. It is the primary control and communication center for the entire body. The nervous system functions with the endocrine system to control and regulate the body's internal environment; that is, it helps maintain homeostasis. All human movement depends on the nervous system; skeletal muscles will not contract unless they receive a signal from the nervous system.

This chapter introduces some basic neuroanatomy and examines the role of the somatic nervous system in controlling human movement. It also discusses the influence of the nervous

system on flexibility and flexibility training and on balance and balance training.

The Nervous System

The nervous system is a fast-acting control system that regulates a virtually endless list of bodily functions. The nervous system has three primary functions:

1. Monitoring the internal and external environment through sensory receptors
2. Integrating the information it has received
3. Initiating and coordinating a response by activating muscles (skeletal, smooth, and cardiac) and glands (including endocrine glands)

These functions are accomplished by the cells of the nervous system (neurons) that communicate with each other and with effector organs (muscle and glands). Communication within a neuron occurs by electrical signals (action potentials). Communication between neurons or between neurons and an effector organ (e.g., skeletal muscle) occurs by chemical signals (neurotransmitters).

The Basic Structure of the Nervous System

As shown in **Figure 20.1**, the nervous system can be structurally divided into the central nervous system (CNS), which consists of the brain and spinal cord, and the peripheral nervous system (PNS), which consists of all neural tissue outside the CNS. The PNS contains *afferent* and *efferent neurons*. *Afferent neurons* relay information about the internal and external environment (from sensory receptors in the periphery) to the CNS. The CNS integrates information it receives from afferent neurons and responds by activating the efferent division. *Efferent neurons* relay signals from the CNS to effector organs in the periphery.

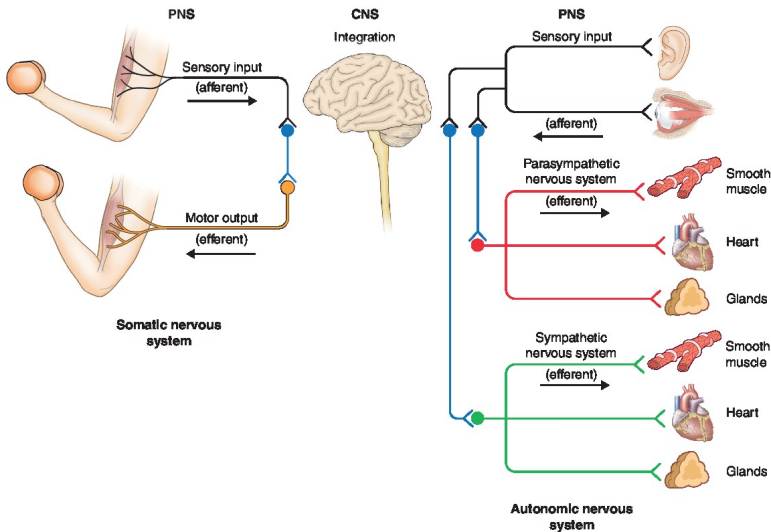


Figure 20.1 Major Divisions of the Nervous System.

The nervous system can be divided anatomically into the central (brain and spinal cord) nervous system (CNS) and the peripheral (sensory organs and nerves) nervous system (PNS). The nervous system can be functionally divided into the somatic nervous system and the autonomic nervous system (ANS). The somatic nervous system leads to contraction of muscle fibers through efferent (motor) neurons. The autonomic nervous system controls the heart, glands, and hollow organs and is essential in maintaining homeostasis.

The somatic (or motor) system, shown on the left side of **Figure 20.1**, sends signals from the CNS to skeletal muscle to initiate muscle contraction and thus movement. This is the focus of this chapter. Although we may not always achieve the desired result from such movement (think about your last golf outing), the somatic system is under voluntary control. The autonomic nervous system (ANS) is involuntary, meaning we do not consciously control its activity. The autonomic nervous system, depicted on the right side of **Figure 20.1**, carries information from the CNS to cardiac muscle, smooth muscle, and endocrine glands, thereby providing subconscious neural regulation of the

internal environment of the body. The autonomic nervous system is discussed more fully in the following chapter.

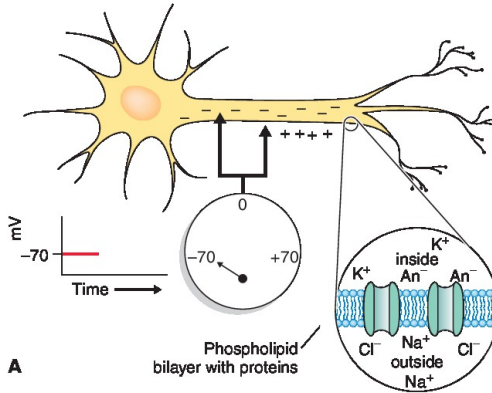
Activation of the Nervous System

The somatic nervous system may be activated by conscious thought or by afferent input from the periphery. Afferent signals involved in regulating nervous control of muscle contraction rely on different types of sensory receptors: mechanoreceptors (pressure, stretch, or contraction) and proprioceptors (spatial orientation), located primarily in skeletal muscle, tendons, and joints. Activation of these receptors often results in a reflex movement. A *reflex* is a rapid, involuntary movement in response to a stimulus (discussed in more detail later in this chapter).

The Nerve Cell

The neuron, or nerve cell, is the functional unit of the nervous system. In addition to the afferent and efferent neurons described above, the central nervous system also has connection or association neurons. Neurons vary considerably in size and shape, depending on their function and location in the body. However, the neurons described in this text generally contain three distinct regions (**Figure 20.2**). The dendrites, highly branched extensions of the neuron, are receptor sites that receive information and convey it to the cell body. The cell body is the control center. It integrates information that it receives from the dendrites and, if the signal is strong enough (at or above threshold level), passes it along to the axon. A cluster of cell bodies in the CNS is called a nucleus; a cluster of cell bodies in the PNS is called a ganglion.

Resting (Polarized) State



Action Potential

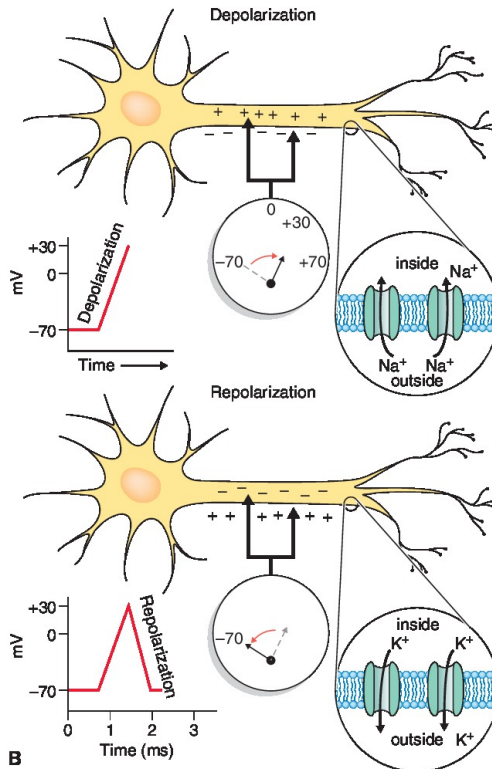


Figure 20.2 Generation of Action Potential.

A. Resting (polarized) state. **B.** Action potential. In the resting state, the neuron cell membrane is polarized (the

inside is negative relative to the outside). During an action potential, the membrane polarity is reversed and the inside of the cell becomes positive.

The axon of a motor neuron is a single extension of the neuron. The axon has two important functions: conducting the action potential and secreting the neurotransmitter. An action potential causes the axon to release a neurotransmitter (a chemical signal) from its axon terminal. The release of neurotransmitters allows neurons to communicate with one another. The length of axons varies according to the body location that is being innervated, but they may be very long. Axons that extend from the spinal cord to the feet may be over a meter long. A long axon is called a nerve fiber, and a bundle of axons is called a nerve. The axon may be myelinated (wrapped with Schwann cells) or unmyelinated.

Nerve cells have the functional characteristic of irritability (the ability to respond to a stimulus). That characteristic is evident in the dendrites and cell body. Nerve cells also have the characteristic of conductivity (the transmission of an electrical impulse from one location to another). The axon conducts an electrical impulse. In general, large-diameter nerves conduct an impulse faster than small-diameter nerves. A myelin sheath protects and electrically insulates axons and increases the rate at which electrical impulses can be conducted. For example, the nerve conduction velocity in an unmyelinated or lightly myelinated neuron such as in the autonomic nervous system or nerves serving sensory functions is $2\text{--}30\text{ mi}\cdot\text{hr}^{-1}$; in a myelinated skeletal muscle neuron, the speed is over $300\text{ mi}\cdot\text{hr}^{-1}$ ([Marieb & Hoehn, 2016](#))!

The Neural Impulse

An **impulse** is a charge transmitted through certain tissue that results in the stimulation or inhibition of physiological activity. A neuron carries an electrical impulse, called the action potential. Neurons, and all cells, possess an electrical resting membrane potential. The resting membrane potential is the difference between the electrical charge on the inside of the cell and the

charge on the outside of the cell (**Figure 20.2A**). This resting potential results from the unequal distribution of positively and negatively charged ions. Negatively charged ions (anions [An^-]) predominate along the inside of the cell membrane and attract positively charged ions (cations) along the outside of the cell membrane. In a typical neuron at rest, sodium (Na^+) and chloride (Cl^-) ions predominate extracellularly. Potassium ions (K^+) and negatively charged protein anions predominate intracellularly.

Impulse An electrical charge transmitted through certain tissue that results in the stimulation or inhibition of physiological activity.

The cell membrane itself is composed of proteins floating in a bilayer of lipids. The membrane is permeable, or capable of allowing ions to pass through it, some by diffusion and some through specific protein channels. Channels may be passive (always open so that they allow a leakage) or active (requiring a chemical or electrical change to open their gates).

At rest, potassium (K^+) “leaks” out through passive channels, and a sodium-potassium pump, which actively transports sodium out across the membrane and potassium back into the cell, removes three sodium ions from the cell for each two potassium ions it brings into the cell. Thus, at rest, there is a net loss of positive ions from the interior of the cell, making the interior negatively charged (-65 to -85 μV) relative to the exterior (**Figure 20.2A**). Thus, the resting neuron is said to be polarized.

When a sufficient stimulus (usually a chemical stimulus from other neurons) is applied to the cell, sodium (Na^+) channels open, and positive ions flow into the neuron. Polarity in this region of the neuron is reversed; that is, the cell membrane is depolarized, meaning that the inside of the cell is now positive relative to the outside (**Figure 20.2B**). This process takes about 1 msec, at which time the sodium gates close and potassium gates open. Potassium exits the cell, bringing about repolarization, or a return to a net negative charge inside. Sodium is returned to the outside of the cell and potassium to the inside by the sodium-

potassium pump, which restores the ionic balance and resting membrane potential. This sequence of events is repeated down the length of the axon. The reversal of polarity or change in electrical potential across a nerve membrane that generates an electrical current is an **action potential**. The propagation of an action potential along the axon of a neuron is the mechanism by which electrical signals are sent within a neuron. Once generated in the axon, the action potential moves along the entire length of the axon and causes a neurotransmitter to be released from the terminal end of the axon. The terminal end of the axon communicates with other neurons, muscle cells, or glands across junctions known as **synapses**. If the synapse is between a neuron and a muscle cell, it is known as a neuromuscular junction.

Action Potential Reversal of polarity or change in electrical potential.

Synapses The gap, or junction, between terminal ends of the axon and other neurons, muscle cells, or glands.

Neural Control of Muscle Contraction

Both the somatic nervous system and the autonomic nervous system play important roles in controlling and regulating the body's response to exercise. However, this chapter focuses on the role of the somatic nervous system in initiating and controlling skeletal muscle contraction. The role of the somatic nervous system in exercise is straightforward. Skeletal muscle will not contract unless it receives a signal from a motor neuron. The action potential in the neuron causes the neuron to release its neurotransmitter, which acts as the signal to initiate contraction. Thus, the somatic nervous system directly regulates exercise. ([Chapter 18](#) explains the events involved in muscle contraction in detail.)

Nerve Supply

All skeletal muscles require nervous stimulation to produce the electrical excitation in the muscle cells that leads to contraction. *Efferent neurons* carry information from the central nervous system to the muscle. *Motor neurons* are efferent neurons that innervate skeletal muscle and are classified as alpha motor neurons or gamma motor neurons. Alpha (α) motor neurons are relatively large motor neurons that innervate skeletal muscle fibers and result in contraction of muscles. Recall from [Chapter 17](#) that $\alpha 1$ motor neurons innervate fast twitch (FT) muscle fibers, whereas $\alpha 2$ motor neurons innervate slow twitch (ST) muscle fibers. *Gamma (g) motor neurons* innervate proprioceptors.

As a nerve enters the connective tissue of the muscle, it divides into branches that all end near the surface of a muscle fiber. Because the axon of the motor neuron branches, each neuron is connected to several muscle fibers. As defined in [Chapter 17](#), a motor neuron and the muscle fibers it innervates are called a *motor unit* (**Figure 20.3**). The motor unit is the basic unit of contraction. Because each muscle fiber in a motor unit is innervated by the same neuron, the electrical activity in that neuron controls the contractile activity of all the muscle fibers in that motor unit. The number of muscle fibers controlled by a single neuron (i.e., the number of muscle cells in a motor unit) varies tremendously, depending on the size and function of the muscle. Although a single neuron may innervate many muscle fibers, each muscle fiber is only innervated by a single motor neuron.

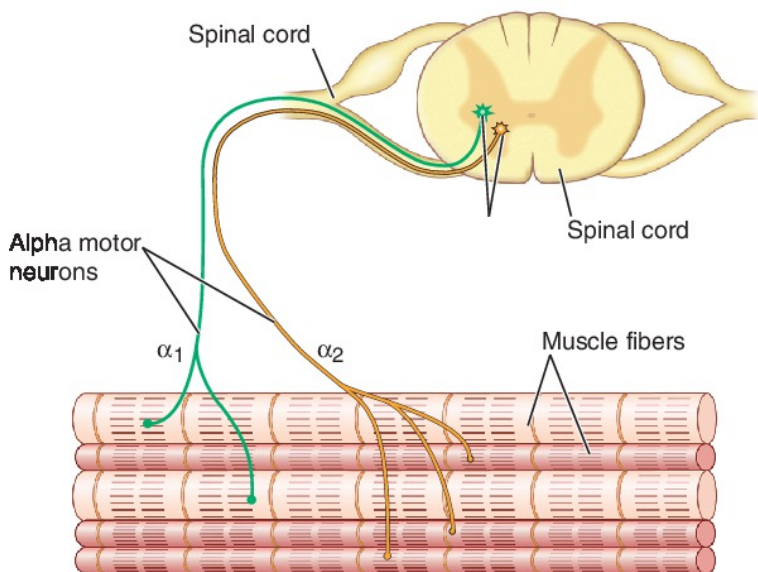


Figure 20.3 Motor Units.

Two motor units are depicted. Notice that the muscle fibers of the two motor units are intermingled.

Figure 20.4 illustrates the relationship between the motor neuron (originating in the central nervous system labeled 1 in **Figure 20.4**) and muscle fibers. The cell body of the motor neuron is located within the gray matter of the spinal cord (labeled 2 in **Figure 20.4**), and the axon extends out through the ventral root of the spinal nerve (labeled 3 in **Figure 20.4**) to carry the electrical signal to the muscle fiber (labeled 4 in **Figure 20.4**). Each branch of the motor neuron terminates in a slight bulge called the axon terminal, which lies very close to but does not touch the underlying muscle fiber.

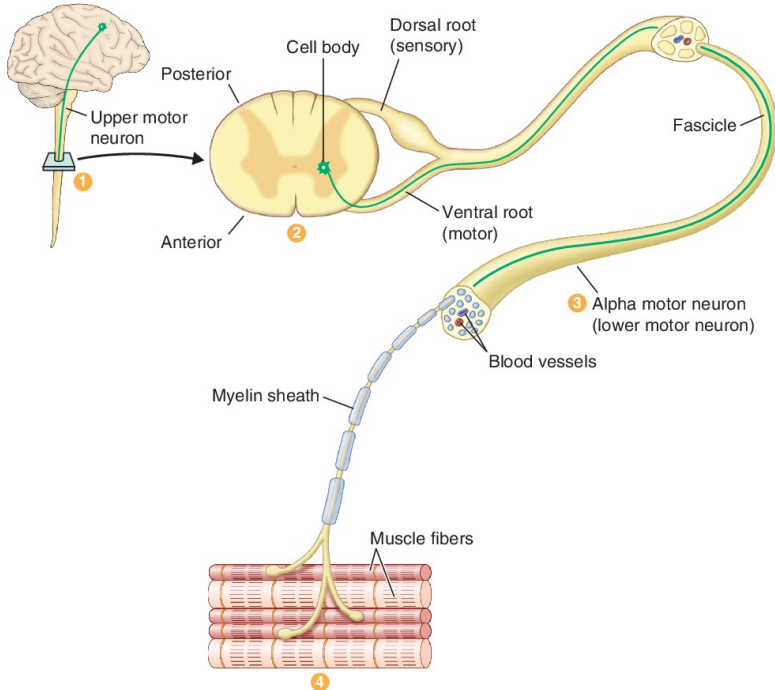


Figure 20.4 Functional Relationship between Motor (Efferent) Neurons and Muscle Cells.

A motor neuron and the muscle cells it innervates are called a motor unit. (1) Schematic of central nervous system, (2) cross-sectional view of the spinal cord, (3) cross-sectional view of peripheral nerve emphasizing axon of motor neuron, and (4) the motor neuron branching near its terminal end where it forms the neuromuscular junction with the muscle fibers it innervates.

The space between the membrane of the neuron and the muscle cell membrane at the *motor end plate* is called the neuromuscular (or synaptic) cleft. The entire region is referred to as the *neuromuscular junction*. The neuromuscular junction is important because the electrical signal from the motor neuron is transmitted here, via a neurotransmitter, to the surface of the muscle cell that is to contract.

Muscle cells also have afferent (sensory) nerve endings, which

are sensitive to mechanical and chemical changes in the muscle tissue and which relay this information back to the central nervous system. The information carried by afferent neurons is used by the central nervous system to make adjustments in muscular contractions.

The Neuromuscular Junction

The neuromuscular junction is a specialized synapse formed between a terminal end of a motor neuron and a muscle fiber. **Figure 20.5** summarizes the events that occur at the neuromuscular junction. When a neuronal action potential reaches the axon terminal, the membrane of the neuron increases its permeability to calcium, and calcium is taken up into the cell (**Figure 20.5A**). The increased level of calcium causes the synaptic vesicles to migrate to the cell membrane and release the neurotransmitter (acetylcholine, ACh) into the synaptic cleft by the process of exocytosis (**Figure 20.5B**). The ACh then diffuses across the synaptic cleft and binds to receptors on the sarcolemma, causing changes in the ionic permeability (especially to the inward flow of Na^+) and leading to the depolarization of the sarcolemma (**Figure 20.5C**). This change in permeability and subsequent depolarization leads to the generation of an action potential in the sarcolemma of the muscle fiber. The action potential spreads in all directions from the neuromuscular junction, depolarizing the entire sarcolemma. The action potential then spreads into the interior of the cell through the T tubules (**Figure 20.5D**) as described in [Chapter 17](#).

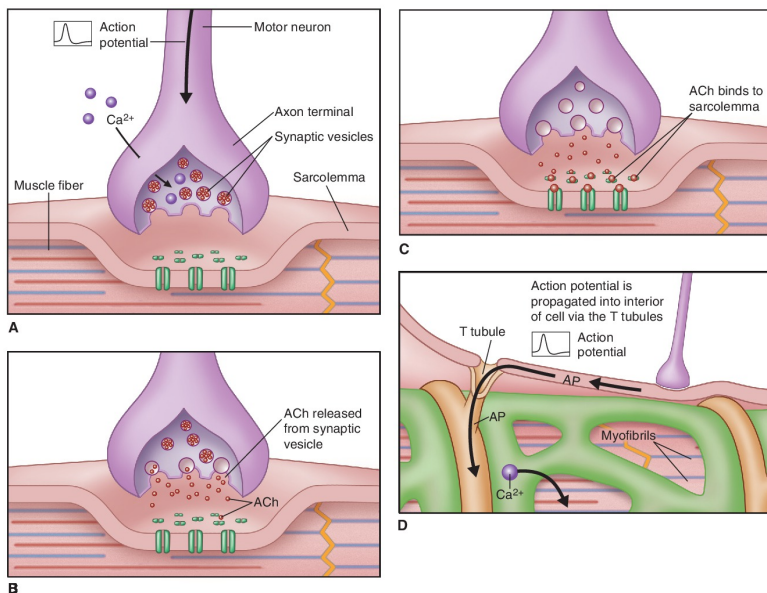


Figure 20.5 Events at the Neuromuscular Junction.

A. An action potential (AP) in the axon terminal causes the uptake of Ca^{2+} into the axon terminal and the subsequent release of the neurotransmitter. **B.** The neurotransmitter (ACh) is released from the synaptic vesicles and diffuses across the synaptic cleft. **C.** Generation of action potential: the binding of ACh to receptors on the sarcolemma causes a change in membrane permeability, causing an AP to be initiated in the sarcolemma. **D.** The AP is propagated into the interior of the cells via the T tubules.

Although the neuromuscular junction functions much like other synapses, there are three important differences.

1. At a neuromuscular junction, a single presynaptic action potential leads to a postsynaptic action potential.
2. The synapse can only be excitatory.
3. A muscle fiber receives synaptic input from only one motor neuron.

Note the two distinct roles that calcium plays in controlling

muscular contraction. The first is to facilitate the release of ACh from the synaptic vesicles in the motor neuron terminal. The second (and most often discussed) role of calcium is to control the position of the regulatory proteins troponin and tropomyosin on actin (as explained in [Chapter 17](#)).

Reflex Control of Movement

Reflexes play important roles in maintaining an upright posture, responding to movement in a coordinated fashion, and mediating responses to stretching. A **reflex** is a rapid, involuntary response to stimuli in which a specific stimulus results in a specific motor response. Reflexes can be classified as *autonomic reflexes*, which activate cardiac and smooth muscle and glands, or *somatic reflexes*, which result in skeletal muscle contraction. This section focuses on somatic reflexes, which play an important role in movement.

Reflex Rapid, involuntary response to stimuli in which a specific stimulus results in a specific motor response.

Many spinal reflexes do not require the participation of higher brain centers to initiate a response. Higher brain centers, however, are often notified of the resultant movement by neurons that synapse with the afferent neuron.

Spinal Cord

The spinal cord is involved in both involuntary and voluntary movements. The spinal cord connects the peripheral nervous system with the brain and serves as the site of reflex integration.

Figure 20.6 shows a cross-sectional view of the spinal cord. Note the spinal nerves extending from each side of the cord. Each spinal nerve contains afferent (sensory) fibers and efferent (motor) fibers. The cell bodies of the afferent neurons are located in the dorsal root ganglion of the spinal nerve, which is part of

the peripheral nervous system. The cell bodies of the efferent neurons, however, are located within the central nervous system, and the axon exits through the ventral root of the spinal nerve. The most familiar efferent neurons are the alpha motor neurons that innervate skeletal muscles leading to muscle contraction. The dorsal root and ventral root of the spinal nerve carry only afferent and efferent fibers, respectively. The two roots join to form a spinal nerve. Therefore, even though individual neurons have only sensory or motor functions, a nerve typically contains both afferent and efferent neurons and so has both sensory and motor functions.

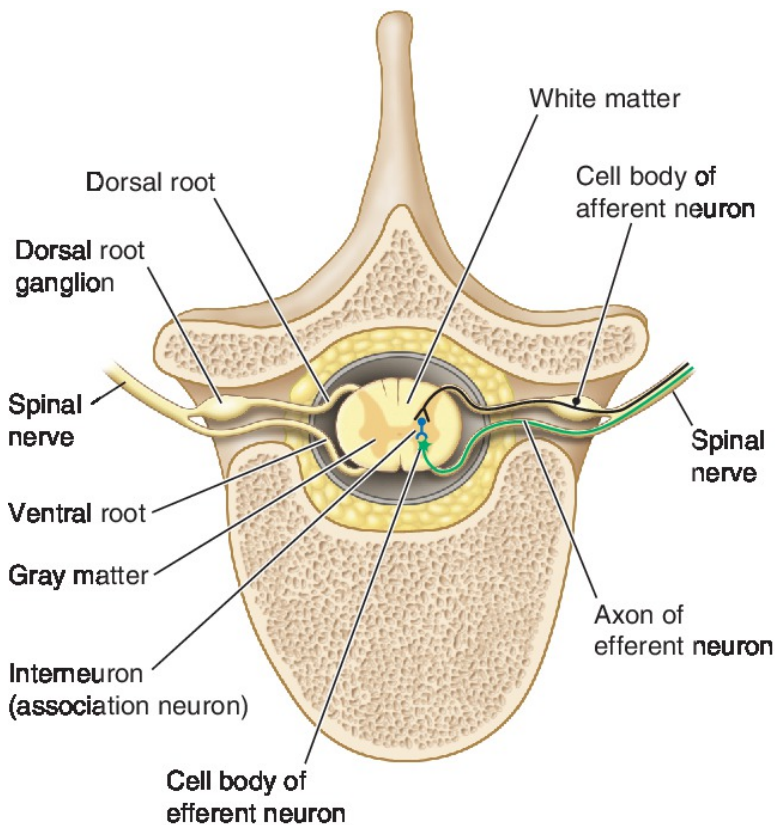


Figure 20.6 Cross-Sectional View of the Spinal Cord Showing the Relationships between the Spinal Cord and Vertebra.

Information is carried up and down the spinal cord through a series of tracts. A tract is a bundle of fibers in the central nervous system. The tracts that carry sensory (afferent) information are called ascending tracts. The tracts that carry motor (efferent) information are called descending tracts. Specific tracts are responsible for carrying different types of sensory information (such as pressure and temperature) and motor information (such as fine distal movement).

The descending pathways of the spinal cord can be divided into the pyramidal and extrapyramidal pathways. Both include several descending tracts that carry specific information. Voluntary motor impulses are transmitted from the motor area of the brain to somatic efferent neurons leading to skeletal muscles via the *pyramidal pathways*.

Pyramidal System

The pyramidal (corticospinal) system is composed of neurons with cell bodies that originate in the cerebral cortex and axons that travel through the spinal cord. The neurons that extend from the brain down through the descending tract are called the upper motor neurons (see **Figure 20.4**). These neurons synapse with the lower motor neurons, also known as the alpha (α) motor neurons, in the anterior gray matter of the spinal cord. The lower motor neurons then carry the message to specific skeletal muscles that control precise, discrete movement.

Both the lateral and anterior corticospinal (pyramidal) tracts decussate, or cross over, as they descend from the brain. Therefore, the motor cortex of the right side of the brain controls the muscles on the left side of the body, and vice versa. Thus, a patient who has had a cerebral vascular accident (stroke) on the right side of the brain may lose the motor function of the left side of the body.

Extrapyramidal System

The extrapyramidal system consists of all descending tracts not included in the pyramidal system. Typically, the neurons found in these tracts have cell bodies located in the basal nuclei or reticular formation of the brain. The extrapyramidal system

carries information that controls muscle tone and posture as well as head movements in response to visual stimuli and changes in equilibrium.

Components of a Reflex Arc

Many of our movements depend on spinal reflexes. Spinal reflexes also play an important role in stretching activities. The neural pathway over which a reflex occurs is called a reflex arc. The basic components of a reflex arc are as follows (**Figure 20.7**).

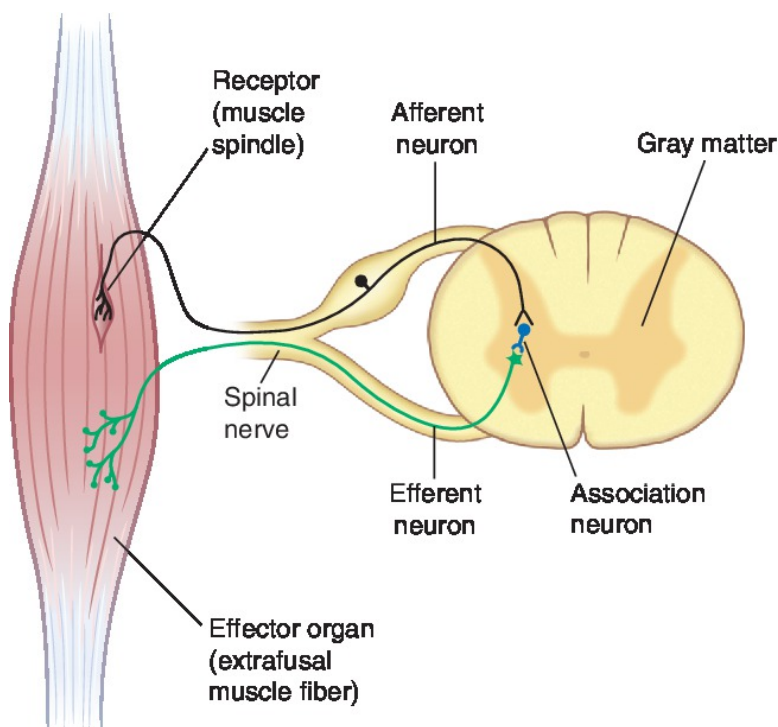


Figure 20.7 Components of a Reflex Arc.

1. The *receptor* organ responds to the stimulus by converting it into a neural (electrical) signal.
2. The *afferent* (sensory) neuron carries the signal to the central nervous system.
3. The *integration center* is located in the central nervous

system. Here, the incoming neural signal is processed through the connection of the afferent neuron with *association neurons* (also called interneurons) and efferent neurons. The incoming afferent neuron may synapse directly with the efferent neuron or with association neurons, depending on the complexity of the reflex.

4. The *efferent* (motor) neuron carries the impulse from the central nervous system to the body organ that is to respond to the original stimulus.
5. The *effector organ* responds to the original stimulus. The effector organ may be a muscle or a gland.

Proprioceptors and Related Reflexes

Proprioceptive sensations provide an awareness of the activities of the muscles, tendons, and joints and provide a sense of equilibrium. This section describes the role of these proprioceptive senses in order to explain the somatosensory system. However, it is important to remember that the other senses and receptors also play a major role in human movement. For example, we use the visual sense to gain important information regarding the speed and direction of a tennis ball during a tennis match. We also listen to the sound of the impact of the ball on the opponent's racket to help judge the return. - Remember that human movement is not only extremely complex but also affected by a host of factors, including sensory receptors of all types.

Stimulation of the proprioceptors gives rise to kinesthetic perceptions. Historically, *kinesthesia* was defined as a person's perception of his or her own motion, specifically the motion of the limbs relative to each other and the body as a whole. *Proprioception* was defined as the perception of movement of the body plus its orientation in space. Over the years, these terms have become practically synonymous ([Schmidt, 1988](#)). Relatively simple exercises using Swiss balls and stability balls can be used to as part of a proprioceptive training program (see accompanying Focus on Research Box).

Vestibular Apparatus


Specialized equilibrium receptors in the inner ear, called the vestibular apparatus, provide important proprioceptive sensations. The primary function of the vestibular apparatus is to maintain equilibrium and to preserve a constant plane of head position by modifying muscle tone (Sage, 1971). Receptors of the vestibular apparatus are located in the vestibule and in the semicircular canals.

The vestibule is comprised of two fluid-filled, sac-like structures called the utricle and saccule. The receptors in these structures, the maculae, sense information about the position of the head when the body is not moving or is accelerating linearly. The semicircular canals are oriented in three planes of space; that is, each of the semicircular canals is positioned at right angles to the other. This arrangement allows the equilibrium receptors in the semicircular canals, the crista ampullaris, to detect angular movement of the head in any plane. The receptors in the semicircular canals are sensitive to changes in the velocity of head movements—that is, to angular acceleration.

Information from the vestibular apparatus is transmitted to the brain via the vestibulocochlear nerve. The information is carried to the vestibular nuclear complex and the cerebellum. Here, it is processed along with information from visual receptors and the somatic receptors of the muscles, tendons, and joints. Although the vestibular receptors are very important, they can be overruled by information from other receptors. For example, the spotting technique (keeping the eyes focused on one spot) used by figure skaters allows them to perform spins without becoming dizzy.

Muscle Spindles and the Myotatic Reflex

Muscle spindles (sometimes called neuromuscular spindles, NMS) are located in skeletal muscle. They lie parallel to and are embedded in the skeletal muscle fibers. These receptors are stimulated by stretch and provide information to the central nervous system regarding the length and rate of length change in skeletal muscles. Stimulation of the muscle spindles results in reflex contraction of the stretched muscle via a myotatic reflex, also known as a stretch reflex (Hall and Hall, 2021). Thus, the muscle spindle performs both a sensory and a motor function.

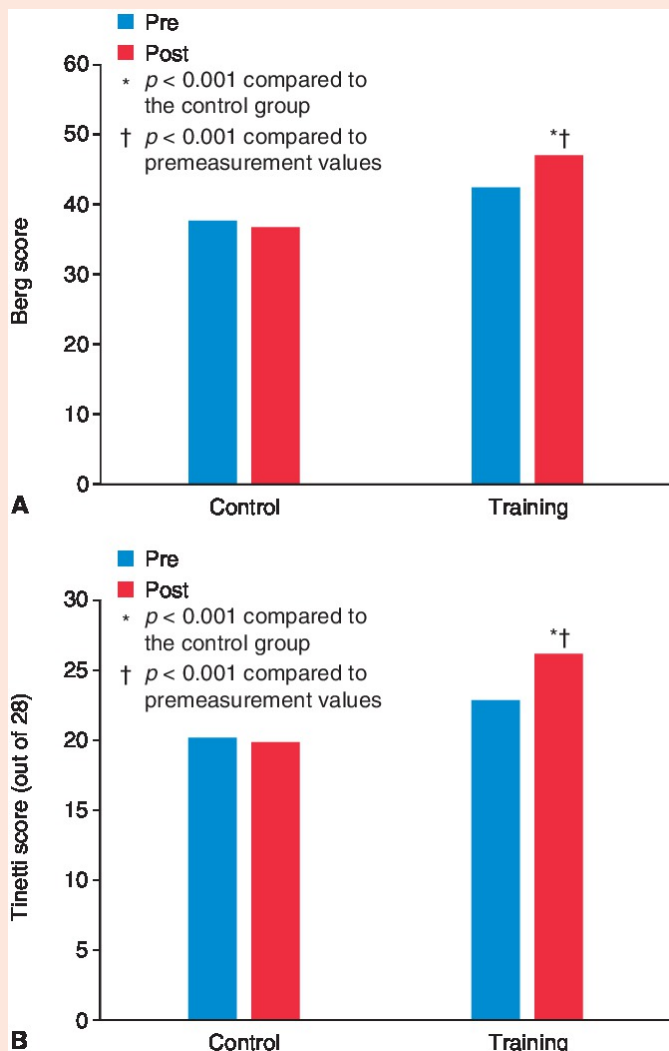


Proprioception Training to Improve Balance and Decrease Risk of Falls in Elderly

Falls are a significant cause of injury and fatality for older individuals. There are multiple factors that increase the risk for falls in older adults. Previous studies have investigated traditional strength training programs as an intervention to improve strength and decrease the risk of falls. Martínez-López and colleagues designed a study to investigate the effects of proprioceptive training programs on balance and the risk of falls in older adults. Study participants ($n = 56$) were divided into two groups, 28 participants (mean age = 79.3 years) received the intervention and the other 26 individuals (mean age = 77.0 years) served as a control group. The intervention group participated in a 12-week proprioception/balance training program that was performed twice a week for a total of 24 sessions, while the control group was prescribed general exercise on the same days. The proprioception exercise sessions contained six specific proprioceptive exercises utilizing a Swiss ball or a wobble ball for 5 minutes each. The proprioception program was progressive and consisted of an initial phase from weeks 1 to 5, an intermediate phase between weeks 5 and 8, and an advanced phase between weeks 8 and 12. A balance test was administered at the beginning of the study using the Berg test. The Berg test is a functional test of balance that consists of 14 observable tasks of daily life. The risk of falls was also assessed before and after the training program. Risk of falls was measured using the Tinetti scale, which assesses gait and balance. The balance component of the Tinetti scale involves testing the following maneuvers in a hard, armless chair: sitting balance, arise, attempts to arise, immediate standing balance, standing balance, nudged, eyes closed, turning, and sitting down. Each of these components can receive a score of 0, 1, or 2, meaning that a maximum score of 28 is possible.

Patients with a score of 24 or below are considered at risk of falls. A score below 19 is associated with a high risk of falls (Raiche et al., 2000). The accompanying figure shows the results of this study. The training program resulted in a significant increase in balance and a lower risk of falling (indicated by a higher score on the Tinetti scale).

The results of this study are very encouraging as they demonstrate that even a short training program that focuses on proprioceptive/balance training can increase dynamic balance and decrease risk of fall-related injuries in older adults.



Source: Martínez-López, E., F. Hita-Contreras, P. Jiménez-Lara, P. Latorre-Román, & A. & Martínez-Amat: The association of flexibility, balance, and lumbar strength with balance ability: Risk of falls in older adults. *Journal of Sports Science & Medicine [serial online]*. 13(2):349–357 (June 2014). Available from: SPORTDiscus with Full Text, Ipswich, MA. Accessed March 24, 2016. Raiche, M., R. Herbert, F. Prince, & H. Corriveau:.

Figure 20.8 represents a muscle spindle and its nerve supply. The muscle spindle consists of a fluid-filled capsule composed of connective tissue; it is long and cylindrical with tapered ends. The typical spindle is 4–7 mm long and approximately 1/5 the diameter of a muscle fiber (extrafusal fiber) (Sage, 1971). The capsule contains specialized muscle fibers called *intrafusal muscle fibers*. In contrast to intrafusal fibers, skeletal muscle fibers that produce muscular movement are sometimes called *extrafusal fibers*. Each end of the spindle is attached to extrafusal muscle fibers.

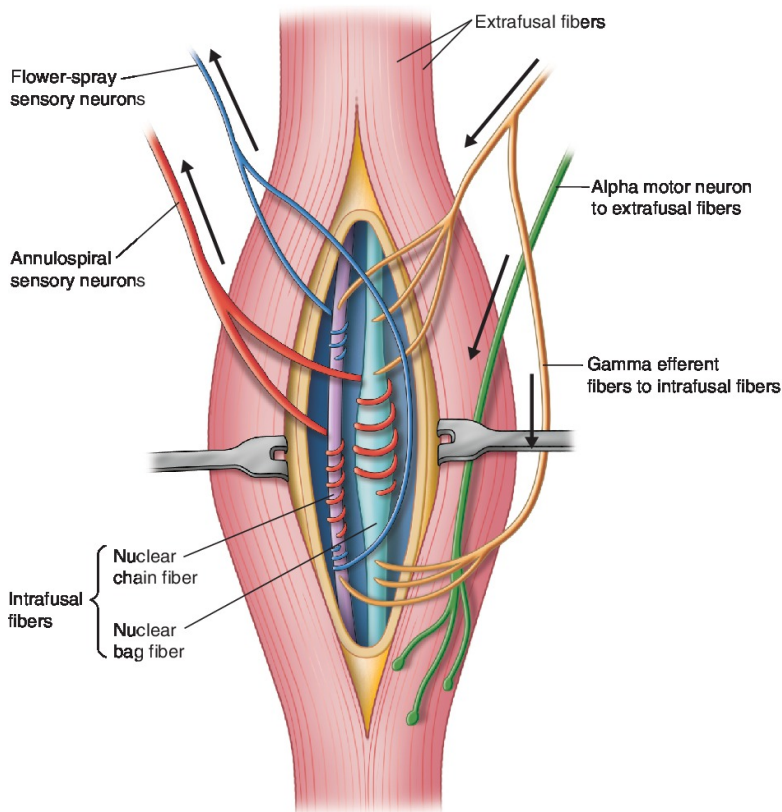


Figure 20.8 Muscle Spindle and Its Nerve Supply.

Muscle spindles transmit sensory information through flower-spray neurons and annulospiral neurons. The muscle spindle also receives motor (efferent) information from gamma efferent fibers.

Two types of intrafusal fibers are located within the muscle spindle: nuclear bag fibers and nuclear chain fibers. *Nuclear bag fibers* are thicker and contain many centrally located nuclei. These fibers extend beyond the spindle capsule and attach to the connective tissue of the extrafusal fibers. *Nuclear chain fibers* are shorter and thinner and have fewer nuclei in the central area of the fiber. Both types of intrafusal fibers contain contractile elements at their distal poles. The central region of the fibers does not contain contractile elements; this is the sensory receptor area of the spindle.

A typical muscle spindle contains 1–3 nuclear bag fibers and 3–9 nuclear chain fibers (Hall and Hall, 2021). The intrafusal fibers of the spindle are innervated by sensory nerves called annulospiral and flower-spray neurons. The branches of the *annulospiral neurons* wrap around the nucleated parts of both types of intrafusal fibers. These annulospiral fibers are large, myelinated fibers (Standring, 2005).

The branches of the *flower-spray neurons* wrap around only the nuclear chain fibers (Standring, 2005). The flower-spray fibers are smaller and conduct impulses slower than the annulospiral fibers. Both types of afferent fibers are stimulated when the central portion of the spindle is stretched. Since the intrafusal fibers are arranged in parallel with the extrafusal fibers, they are stretched or shortened with the whole muscle. The flower-spray nerve endings have a higher threshold of excitation than the annulospiral nerve endings. The flower-spray nerve endings provide information about relative muscle length; the annulospiral nerve endings respond primarily to the rate of length change.

The contractile intrafusal fibers also receive motor innervation from the central nervous system. The efferent fibers that terminate on the intrafusal fibers are called gamma efferents (γ motor neurons) or fusimotor neurons. The axons of the gamma motor neurons travel in the spinal nerve and terminate on the

distal ends of the intrafusal fibers. Stimulation of the gamma motor neurons produces contraction of the intrafusal fiber, which causes the central region of the spindle to be stretched. Gamma motor neurons are important enough to comprise almost a third of all motor neurons in the body.

MYOTATIC REFLEX The myotatic or stretch reflex has two separate components: a dynamic reaction and a static reaction. The dynamic reaction occurs in response to a sudden change in length of the muscle. When a muscle is quickly stretched, the annulospiral nerve endings (but not the flower-spray endings) transmit an impulse to the spinal cord, which results in an immediate strong reflex contraction of the same muscle from which the signal originated (Hall and Hall, 2021). This is what happens in the knee-jerk response, diagrammed in **Figure 20.9**, or in the head-jerk response when you fall asleep sitting up reading a book not nearly as interesting as this one. Because this reflex does not require that information be processed by the brain, it occurs very quickly.

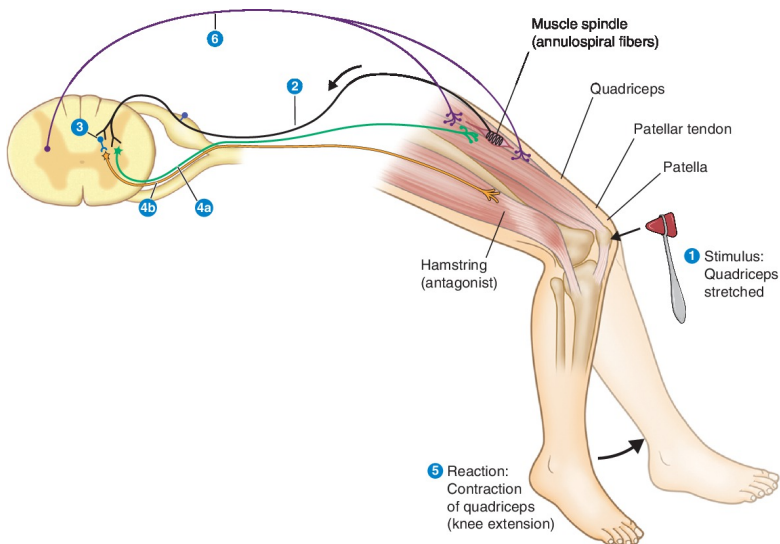


Figure 20.9 Myotatic (Stretch) Reflex.

A stimulus (1) is transmitted to the CNS via the afferent neuron (2). In the CNS, the afferent neuron bifurcates and

synapses with an alpha motor neuron (3a) and an association neuron (3b). The alpha motor neuron (4a) leads to contraction of the agonist (quadriceps). The efferent fiber (4b) that synapses with the association neuron results in relaxation of antagonist (hamstrings) and leads to knee extension (5). Gamma efferent neurons (6) innervate the muscle spindles and help provide smooth, coordinated movements.

Stretching of the skeletal muscle results in the stimulation of the muscle spindle fibers, which monitor changes in muscle length. In the knee-jerk response, the stretch is initiated by a tapping on the patellar tendon, causing a deformation that stretches the quadriceps muscle group ([1] in **Figure 20.9**). Sudden stretching of the muscle spindle causes an impulse to be sent to the spinal cord by way of the annulospiral nerve fibers ([2] in **Figure 20.9**). In the gray matter of the spinal cord, this sensory fiber bifurcates, with one branch synapsing with an alpha motor neuron ([3a] in **Figure 20.9**). The other branch synapses with an association neuron ([3b] in **Figure 20.9**). The alpha motor neuron exits the spinal cord and synapses with the skeletal muscle, which was originally stretched ([4a] in **Figure 20.9**), resulting in a contraction that is roughly equal in force and distance to the original stretch ([5] in **Figure 20.9**). The inhibitory association neuron synapses with another efferent neuron, which innervates the antagonist muscle (hamstring group in this example), where it causes inhibition; this reflex relaxation of the antagonist muscle in response to the contraction of the agonist is called **reciprocal inhibition**. This response facilitates contraction of the agonist muscle that was stimulated; the inhibited antagonist cannot resist the contraction of the agonist ([4b] in **Figure 20.9**). The muscle spindle is also supplied with a gamma efferent neuron; for clarity, this neuron is shown on the opposite side of the spinal cord ([6] in **Figure 20.9**).

Reciprocal Inhibition The reflex relaxation of the antagonist muscle in response to the contraction of the agonist.

As soon as the lengthening of the muscle stops increasing, the rate of impulse discharge returns to its original level, except for a small static response that is maintained as long as the muscle is longer than its normal length. The static response is elicited by both the annulospiral and flower-spray nerve endings. The resultant low-level muscle contractions oppose the force that is causing the excess length, with the ultimate goal of returning to the resting length. A static response is also invoked if the sensory receptor portion of the neuromuscular spindle is stretched slowly.

Normally, muscle spindles emit low-level sensory nerve signals that help maintain muscle tonus and postural adjustments. **Muscle tonus** is a state of low-level muscle contraction at rest. Muscle spindles also respond to stretch by an antagonistic muscle, to gravity, or to a load being applied to the muscle. The head jerk is an example of the response to gravity, but other muscles such as the back extensors and quadriceps function the same way for unconscious postural adjustments. As an example of the load stimulus, think about what happens if you stand with your elbows at 90 degrees, palms up, and someone places a 10-lb weight in your hands. Before you can consciously adjust to this weight, and because the weight stretches your biceps, the muscle spindles cause a reflex contraction that stops your hands from dropping too far.

Muscle Tonus A state of low-level muscle contraction at rest.

In addition to providing maintenance of muscle tone and adjustments for posture and load, the neuromuscular spindle also serves as a damping mechanism that facilitates smooth muscle contractions. This is accomplished by a *gamma loop*.

In a gamma loop, the stretch reflex is activated by the gamma motor neurons ([6] in **Figure 20.9**). Recall that the gamma motor neurons originate in the spinal cord and innervate the distal contractile portions of the intrafusal fibers. Pick up a rubber band and hold it at its maximal, unstretched length with your fingers. Now pull on both ends. What happens to the rubber band in the middle? Obviously, it is stretched. This is precisely what happens when the gamma motor neurons stimulate contractions at both

ends of the intrafusal fibers. The central, noncontractile portion of the fibers is stretched, deforming the sensory nerve endings and eliciting the myotatic stretch response.

What stimulates gamma motor neurons, and why? When signals are transmitted to the alpha motor neurons from the motor cortex or other areas of the brain, gamma motor neurons are almost always simultaneously stimulated. This action is called *coactivation*, and it serves several purposes. First, it provides damping, as mentioned earlier. Alpha and gamma neural signals to contract sometimes arrive asynchronously. But because the response to the gamma motor neuron stimulation is contraction anyway, the gaps can be filled in by reflex contractions, thereby smoothing out the force of contraction. Second, coactivation maintains proper load responsiveness regardless of the muscle length. If, for example, the extrafusal fibers contract less than the intrafusal fibers owing to a heavy external load, the mismatch would elicit the stretch reflex, and the additional extrafusal fiber excitation would cause more shortening (Hall and Hall, 2021). Similarly, because the intrafusal and extrafusal lengths are adjusted to each other, the neuromuscular spindle may be able to help compensate for fatigue by recruiting additional extrafusal fibers by reflex action. Finally, sensory information from neuromuscular spindles is always carried to higher brain centers, where it is unconsciously integrated with other sensory information. If the muscle spindles were not adjusted to the length of the extrafusal fibers during contraction, information on muscle length and the rate of length change could not be transmitted (Hall and Hall, 2021). Since gamma fibers do adjust the muscle spindle fibers, the gamma loop can assist voluntary motor activity, but it does not actually control voluntary motor activity.

PLYOMETRICS *Plyometrics*, also known as depth jumping or rebound training, is a training exercise in which a concentric contraction is immediately preceded by an eccentric contraction. Plyometrics is an explosive form of physical training used to enhance power output, force production, and velocity. It involves such activities as jumping off a box with both feet together and then immediately performing a maximal jump back onto the box. Plyometric training, alone or in combination with other training

modalities, has been shown to be effective in improving power output, force production, jumping height, athletic performance, and bone mineral density (Beato et al., 2018; Ribeiro et al., 2020; Vlachopoulos et al., 2018). While plyometric training has proven effective in improving lower extremity strength, power, and stretch-shortening muscle function, the mechanisms responsible for the improved performance have not been fully elucidated. However, it appears that the adaptive neuromuscular changes are the result of several factors, including increased neural drive, changes in muscle activation, changes in the mechanical properties of the muscle-tendon complex, and changes in muscle size and/or architecture (Markovic and Mikulic, 2010).

The stretch reflex is caused by activation of the muscle spindles in the agonist muscle. As the agonist is stretched during the eccentric phase of the movement, the muscle spindles activate a reflex arc culminating in stimulation of the alpha motor neuron and enhanced concentric contraction of the agonist. That is, stretching a muscle before concentrically contracting results in a more forceful contraction than if no prestretch occurred. In addition, as described in Chapter 18 (see Stretch Shortening Cycle, Figure 18.8), elastic proteins, such as titin, in the muscle act in a similar manner as a rubber band to enhance the force of contraction. During the eccentric phase, these series elastic components are stretched and stored as potential energy. The stored energy is released on initiation of the concentric phase, allowing a more forceful contraction (Potach and Chu, 2000).

Muscular strength changes are routinely found after plyometric training, and research indicates that increased drive to alpha-motor neuron pool posttraining may contribute to strength gains (Alkjaer et al., 2013; Taube et al., 2012b). Neural adaptations responsible for increased strength and power in jump and landing performance, such as during plyometric training, may be dependent on drop height. To maximize rebound height most efficiently, high drop heights should be implemented into training if time on the ground is not a factor. In contrast, low drop heights should be incorporated to improve time and power output (Taube et al., 2012b). During planned landing activity, such as during plyometric training, there is the potential for the CNS to predict the time of ground contact and to contribute to the control of muscular activity at the time of ground contact.

Such CNS-mediated programmed muscular activity may interact with components of reflex responses. Thus, the CNS may initiate jumping movement and participate with reflexes in modifying muscular response to landing (Taube et al., 2012a).

FOCUS ON APPLICATION

The Role of Plyometrics in the Prevention of Lower Extremity Injuries

Plyometric training is often used to increase muscle strength and improve athletic performance. However, there is compelling evidence that plyometric training can decrease the risk of lower extremity injuries, specifically, anterior cruciate ligament (ACL) tears in female athletes who participate in sports that involve jumping and pivoting. The results from training studies indicate a reduction in the *rate of injuries* or a *decrease in the risk factors* associated with ACL injuries (such as knee instability, imbalance between quadriceps, and hamstring ratio). Hewett et al. (1999) prospectively monitored high school female athletes (soccer, volleyball, and basketball) to assess the effectiveness of plyometric training on the rate of injuries. Fifteen sports team participated in a 6-week plyometric training program during their preseason training. Fifteen sports teams did not participate in the structured preseason plyometric training program and served as the control group. The plyometric training consisted of jump and landing techniques designed to increase vertical jump height and increase lower extremity strength. Training sessions lasted 6 weeks and were led by trained coaches and physical therapists. The training sessions lasted 60–90 minutes a day, 3 d·wk⁻¹. Following the training program, the researchers monitored the athletes during their competitive season by compiling weekly injury data. Injury data included reporting of all injuries and in-depth information regarding lower extremity injuries. Serious injuries were defined as a loss of at least 5 consecutive days

of practice or competition. All ACL ruptures were confirmed by arthroscopy. The study found a significantly lower rate of lower extremity injuries in female athletes who engaged in plyometric training compared to those who did not engage in plyometric training. In fact, untrained female athletes had a 3.6 times higher incidence of knee injury than the trained females. This study clearly suggests the preventative role plyometric training can play in reducing the number of knee injuries. Encouragingly, many other studies have replicated this finding.



Sources: [Hewett et al. \(1999\)](#); [Markovic and Mikulic \(2010\)](#); [Myer et al. \(2006\)](#).

Golgi Tendon Organs and Inverse Myotatic Reflex

Golgi tendon organs (GTOs) are receptors activated by stretch or active contraction of a muscle. They transmit information about muscle tension. Activation of these receptors results in a reflex inhibition of the muscle via the inverse myotatic reflex ([Marieb and Hoehn, 2016](#)). GTOs are located in the tendons, close to the point of muscular attachment. As shown in **Figure 20.10**, each Golgi tendon organ consists of a thin capsule of connective tissue enclosing collagenous fibers. The collagenous fibers within the

capsule are penetrated by fibers of sensory neuron whose terminal branches intertwine with the collagenous fibers. This afferent neuron relays information about muscle tension to the spinal cord. The information is then transmitted to muscle efferents and/or to higher brain centers, particularly the cerebellum.

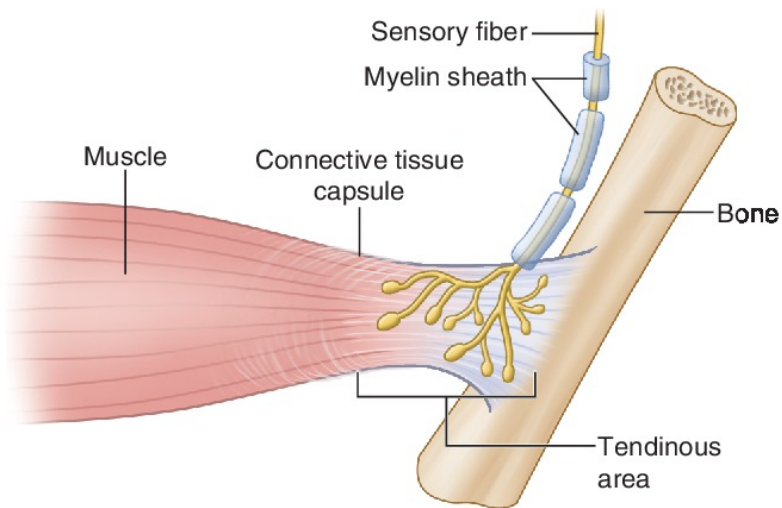


Figure 20.10 Golgi Tendon Organ.

The Golgi tendon organ is in series with the muscle. The Golgi tendon organ, therefore, can be stimulated by either stretch or contraction of the muscle. Because of elongation properties of the muscle during stretch, however, active contraction of a muscle is more effective in initiating action potentials within the Golgi tendon organ.

INVERSE MYOTATIC REFLEX As with the myotatic reflex, the inverse myotatic reflex has both a static and dynamic component. When tension increases abruptly and intensely, the dynamic response is invoked. Within milliseconds, this dynamic response becomes a lower-level static response within the GTO that is proportional to the muscle tension. The sequence of events in the inverse myotatic reflex is diagrammed in **Figure 20.11**.

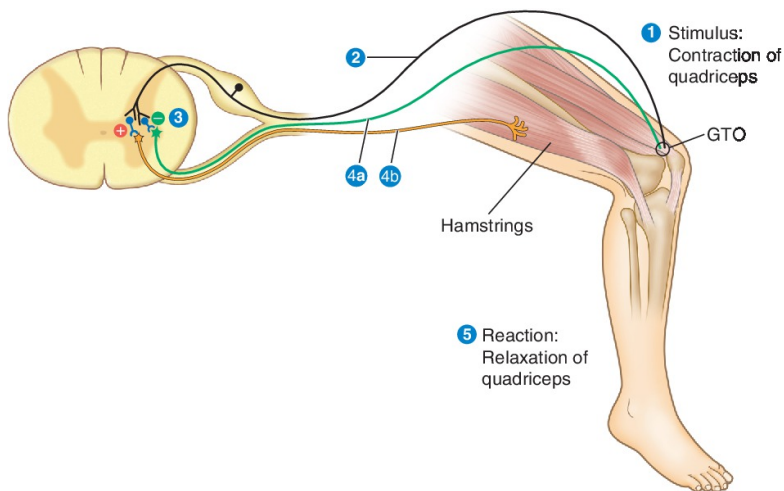


Figure 20.11 Inverse Myotatic Reflex.

A stimulus (1) is transmitted to the CNS via the afferent neuron (2). In the CNS, association neurons (3) activate efferent neurons (4a and 4b) that result in relaxation of the agonist (5).

Contraction of a skeletal muscle (or stretching) results in tension that stimulates the Golgi tendon organs in the tendon attached to the skeletal muscle ([1] in **Figure 20.11**). Stimulation of the Golgi tendon organ results in the transmission of impulses to the spinal cord by afferent neurons ([2] in **Figure 20.11**). In the spinal cord, the afferent neuron synapses with an inhibitory association neuron and an excitatory motor neuron ([3] in **Figure 20.11**). In turn, the inhibitory association neuron synapses with a motor neuron that innervates the muscle attached to the tendon; the inhibitory impulses lead to the relaxation of the contracted muscle ([4a] in **Figure 20.11**). The excitatory association neuron synapses with a motor neuron that innervates the antagonist muscle ([4b] in **Figure 20.11**).

Note that as the muscle group originally exhibiting the tension (the agonist) is relaxed, the opposing muscle group (or antagonist) is reciprocally activated. The relaxing action of the Golgi tendon organ serves several important functions. First, the Golgi tendon organ protects against muscle and tendon damage.

Excessive tension that might cause muscles and tendons to be torn or pulled away from their attachments is prevented by action of the Golgi tendon organ. For example, a weight lifter who manages to get a heavier barbell off the ground than he or she can really handle may suddenly find his or her muscle giving out because of the action of the Golgi tendon organs. It is speculated that increases in the amount of weight that can be lifted following resistance training are in part due to an inhibition of the Golgi tendon organs, allowing for a more forceful muscle contraction ([Gabriel et al., 2006](#)).

The second important advantage of Golgi tendon organ-mediated relaxation is that muscle fibers that are relaxed can be stretched further by opposing muscles or external force without damage. This response is useful in the development of flexibility.

Third, the sensory information regarding tension, which is provided to the cerebellum, allows for muscle adjustment so that only the amount of tension needed to complete the movement is produced. This feature ensures both a smooth beginning and a smooth ending to a movement and is particularly important in movements such as running that involve a rapid cycling between flexion and extension ([Biering-Sorensen, 1984](#)).

Volitional Control of Movement

Reflexes are important for controlling human movement, but the volitional control of movement required for skilled movement involves even more complexity. Furthermore, volition and reflexes often work simultaneously to ensure smooth, coordinated movement. This section discusses the volitional control of motor units and of whole muscles.

Volitional Control of Individual Motor Units

Healthy people often take the ability to move for granted; they assume that if their brain sends the proper signal, the desired action will simply occur. One discovers that it is not quite that

simple when attempting to learn a new sports or when illness or injury intervenes. We normally do not think about conscious control of single motor units. Most people consider reflex action the most basic, albeit unconscious, form of muscle action. But even reflexes involve more than one motor unit and result in muscle activity that can be felt by the individual. In reality, motor unit control is the most basic level of volitional control of movement that can be achieved.

Basmajian (1967) and colleagues performed a series of experiments using intramuscular electrodes attached to a tape recorder and audio amplifier for sound and an oscilloscope and camera for visual feedback. Initially, when subjects were asked to activate a motor unit, say, in the little finger, they moved the finger and got a typical EMG tracing such as the one shown in **Figure 20.12D**. Gradually, as movement was decreased to virtually nothing but neural signals were still being sent, a single motor unit (identifiable by a characteristic sound and spike pattern) (**Figure 20.12A**) was isolated. Once one motor unit had been isolated, the frequency of its recruitment could be varied. Other motor units could also be isolated (**Figure 20.12B and C**), and firings could be varied between the motor units. Thus, it has been shown that one truly can control motor units. Precisely, *how* this control is achieved is unclear both to the subjects doing it and to the scientists observing it, although some type of proprioceptive feedback is suspected.

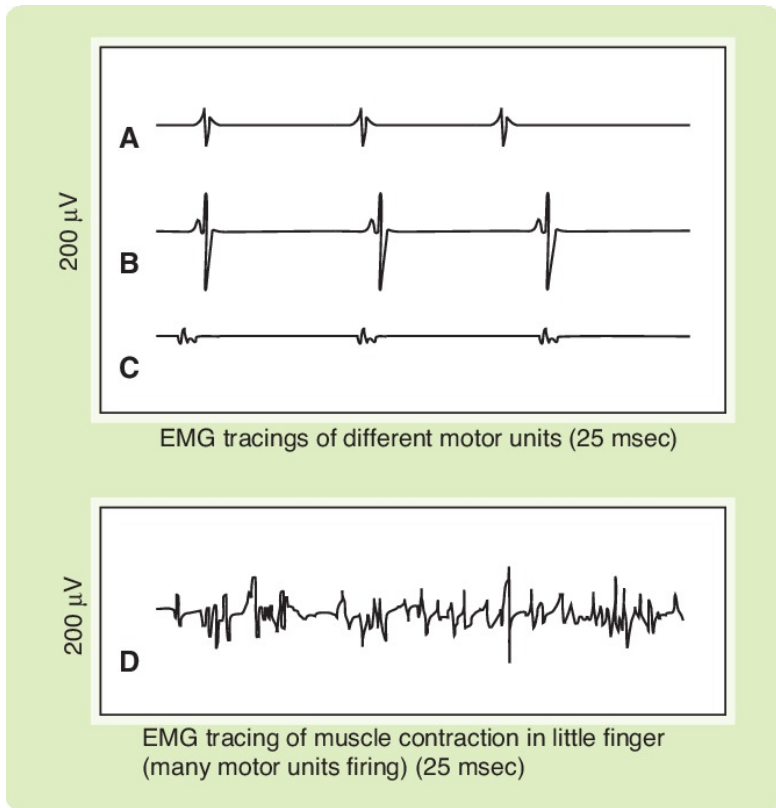


Figure 20.12 Volitional Control of a Single Motor Unit.

A. Single motor unit. B. A second single motor unit. C. A third single motor unit with different firing rate. D. EMG tracing when moving the little finger. **Source:** Reprinted with permission from Basmajian, J. V.: *Control of individual motor units. American Journal of Physical Medicine.* 46(1):480–486 (1967).

Such delicate, discrete control has obvious implications for the refinement of motor skills. Perhaps its greatest benefit, however, is in the area of therapeutic rehabilitation. For example, bioengineers can use trained motor units to control myoelectric prostheses and orthoses. Individuals disabled with conditions such as cerebral palsy can learn better motor control. And some individuals whose spinal cords have been injured but not totally

destroyed can learn through such biofeedback to control first one motor unit and then another to regain muscle movement.

Volitional Control of Muscle Movement

Although the way the nervous system initiates and controls muscle movement has been studied for over 100 years, it is still not fully understood. Volitional movement is the result of cognitive processes, which ultimately lead to muscular contraction (Schwartz, 2016). **Figure 20.13** provides a simplistic overview of volitional movement. The brain initiates movement ([1] in **Figure 20.13**); that is, the plan for a desired movement, whether to lift a book or perform the high jump, originates in the brain. This information is transmitted down the appropriate descending tract ([2] in **Figure 20.13**). The neurons of the descending tract synapse with the motor neurons in the gray matter of the spinal cord. The efferent motor neuron then carries the impulse to the muscle, the effector organ ([3] in **Figure 20.13**).

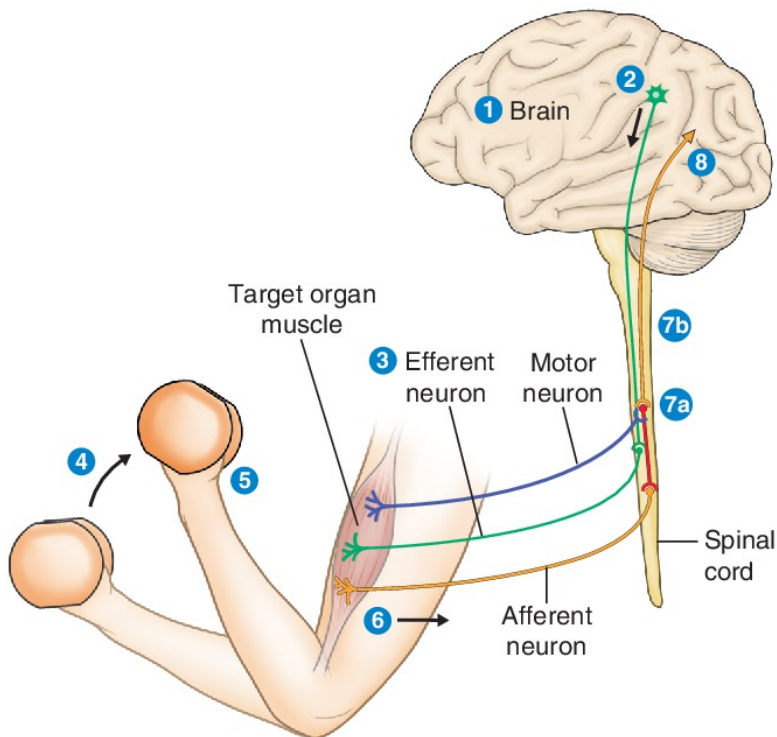


Figure 20.13 Overview of Volitional Movement.

Voluntary movement is initiated by the motor cortex (1). Neural signals are transmitted down the descending tracts (2) and carried to the muscles via efferent motor neurons (3) that synapse with the target muscles (4) to cause contraction (5). Movement causes stimulation of the afferent neurons (6) that synapse with association neurons (7a) in the spinal cord and then transmit the signal to neurons in the ascending tract (7b). Information from multiple sources, including multiple neurons in the ascending tract and sensory neurons, is integrated in the brain (8) and appropriate adjustments are made to ensure movements correspond with intended movement.

On receiving the signal from the nervous system, the muscle contracts and produces movement ([4] in **Figure 20.13**). Changes in muscle length, tension, and position stimulate receptors in the

muscles and joints of surrounding muscles ([5] in **Figure 20.13**). This information is transmitted to the central nervous system through afferent sensory neurons ([6] in **Figure 20.13**). The afferent neurons synapse with various association neurons in the gray matter of the spinal cord. In some instances, the neurons synapse with association neurons, which synapse with efferent motor neurons to reflexively control movement ([7a] in **Figure 20.13**). In other cases, the association neurons synapse with neurons of the ascending tract, which will carry the information to the brain ([7b] in **Figure 20.13**).

The signals from the ascending pathway are transmitted to the brain, where the information is perceived, compared, evaluated, and integrated in light of past experience, desired outcome, and additional sensory information ([8] in **Figure 20.13**). The brain then adjusts its original message, which is again sent to the muscles via the descending pathway and the motor neuron.

This cycle continues throughout the duration of a given activity. The speed at which the information is transmitted is as remarkable as the degree of integration that occurs. What seems an instantaneous response, on, say, a racquetball court (such as reaction to a powerful serve), actually requires a vast amount of communication within the neuromuscular system.

As already stated, many movements rely on both involuntary reflex action and volitional control of movement. An example is flexibility exercise. Although we decide to initiate stretches, the responses to them depend largely on reflexes.

Flexibility

Flexibility is the range of motion (ROM) in a joint or series of joints that reflects the ability of the musculotendon structures to elongate within the physical limitations of the joint (Hubley-Kozey, 1991). Neither pain nor injury should be involved in this range. The two basic types of flexibility are static and dynamic. *Static flexibility* is the range of motion about a joint without considering how easily or quickly the range of motion is achieved. *Dynamic flexibility* is the resistance to motion in a joint that affects how easily and quickly a joint can move through its

range of motion. Dynamic flexibility is also defined as the rate of increase in tension in a contracted or relaxed muscle as it is stretched. Thus, dynamic flexibility accounts for the resistance to stretch (Knudson et al., 2000). Dynamic flexibility is undoubtedly more important than static flexibility in relation to athletic performances (especially speed events) and the health or diseased condition of the joints (such as arthritis). Resistance to stretch is measured as *stiffness*. The opposite of stiffness is *compliance* (alteration in response to force). Stiffness is determined by the slope of a curve that plots the load (torque) against elongation (range of motion) for the individual. The steeper the line, the stiffer the muscle is. This testing requires specialized laboratory equipment (Gleim and McHugh, 1997). Muscle stiffness and dynamic flexibility can be assessed using shear wave elastography (SWE), which is an ultrasound technology (Sarto et al., 2021). However, no standardized measurement technique exists for evaluating dynamic flexibility in practical settings (Plowman, 1992). Therefore, unless specifically stated otherwise, the discussion that follows is limited to static flexibility.

Flexibility The range of motion in a joint or series of joints that reflects the ability of the musculotendon structures to elongate within the physical limits of the joint.

Several anatomical factors affect the range of motion in any given joint. The first is the actual structural arrangement of the joint—that is, the way the bones articulate. Each joint has a specific bony configuration that generally cannot and should not be altered. The soft tissue surrounding the joint, including the skin, ligaments, fascia, muscles, and tendons, also affects joint range of motion. The skin normally has very little influence on the range of motion. The ligaments provide joint stability, and their restriction of range of motion is generally considered necessary and beneficial. Thus, the muscles and their connective tissues are the critical factors that determine flexibility and that can be altered by stretching training.

Muscles actively resist elongation through contraction and passively resist elongation because of the noncontractile elements

of elasticity and plasticity. The difference between elastic and plastic properties is similar to the difference between a rubber band and a balloon. If the rubber band is stretched and then let go, it should rebound to its original length—at least when it is new and has not been frequently stretched. That is elasticity. Blowing up a balloon also stretches it, but when the air is let out of a previously fully inflated balloon, it does not return to its original size. That is plasticity. With stretching training, one attempts to influence the plastic deformation so that a degree of elongation remains when the force causing the stretch is removed (Plowman, 1992). Flexibility is affected by neuromuscular disorders characterized by spasticity and rigidity, any injuries resulting in scar tissue, and adaptive muscle shortening caused by casting.

Flexibility and stretching are important for everyday living (putting on shoes, reaching the top shelf) and for muscle relaxation and proper posture. Flexibility is obviously important also for sports performance. How important flexibility is depends on the sport. **Table 20.1** lists some popular sports and fitness activities according to the degree of flexibility required. The three degrees of flexibility listed are a normal range of motion, a slightly above-average range of motion in one or more joints, and an extreme range of motion in specific joints.

TABLE 20.1 Flexibility in Sports and Fitness Activities

| Skills Requiring Extreme Range of Motion in Specific Joints | Skills Requiring Greater-than-Normal Range of Motion in One or More Joints | Skills Requiring Only Normal Range of Motion in Involved Joints |
|---|--|---|
| Dancing (ballet, modern) | Jumping | Archery |
| Diving | Most team sports | Basketball |
| Figure skating | Racquet sports | Bicycling |
| Gymnastics | Sprinting | Boxing |
| Hurdles | Swimming | Cross-country skiing |
| Karate | Wrestling | Curling |
| Pitching | | Horseback riding |
| Yoga | | Long-distance jogging or running |
| | | Resistance training |
| | | Skating (in-line, roller) |
| | | Shooting |
| | | Stair stepping |

Note: If the skills involved with a particular sport do not require greater-than-normal flexibility, it does not mean that stretching exercises should not be included in the exercise program.

Source: Modified from Hubley-Kozey (1991).

Gymnasts obviously need to be more flexible than long-distance runners and cyclists. However, no definitive scientific studies directly link selected flexibility values with quality of performance in athletes who can move through the required range of motion (Gleim and McHugh, 1997; Plowman, 1992). For example, a bicyclist with normal range of motion in the ankle, knee, hip, and trunk will not become a better cyclist just by increasing flexibility in those joints.

Measuring Flexibility

The measurement of flexibility is not an exact, standardized procedure with a well-established criterion test. Direct measurement in the laboratory usually measures angular displacement (in degrees) between adjacent segments or from a reference point. Such measurements are usually performed with a goniometer or inclinometer.

A goniometer (**Figure 20.14A**) resembles a protractor with two arms, one of which is movable. The center of the goniometer is placed over the joint about which movement occurs, and the arms of the goniometer are aligned with the body segments on

both sides of the joint. For a measurement of the range of motion at the knee joint, the center of the goniometer would coincide with the knee, the stationary arm would be aligned with the upper leg, and the movable arm would be aligned with the lower leg. The angle would be measured at the extreme range of motion for extension and flexion—that is, with the leg completely extended and completely flexed. The range of motion for this joint is the difference between the angles measured.

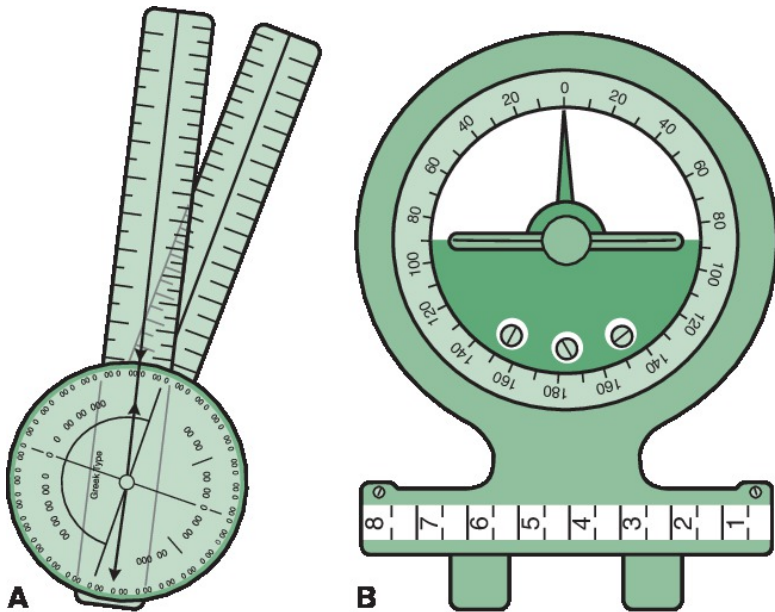


Figure 20.14 Instruments to Measure Flexibility.

A. Goniometer. **B.** Inclinometer.

Spinal and ankle movements are difficult to assess with a goniometer. For these, one or more inclinometers may be used (see **Figure 20.14B**). Inclinometers have a universal center of gravity as a starting point. To use an inclinometer, the instrument is held by the tester and zeroed at the standard starting position for any given joint. The participant then bends the joint through its ROM. For example, for cervical flexion, the inclinometer would be placed on the top of the head; the participant would then slowly bend the chin toward the chest. The inclinometer's

final reading (the angle at which the inclinometer is situated relative to the starting universal line of gravity) is the ROM measurement. Both mechanical and electronic inclinometers are available.

In recent years, wireless inertial measurement instruments have been used as motion capture devices. One such instrument is the WIMUTM (RealTrack Systems, Almeria, Spain). The WIMU consists of triaxial accelerometers, a 3D gyroscope, 3D magnetometer, barometer, and GPS. Data are processed through specific software. It is smaller than the typical inclinometer. It has been found to be valid and reliable for the assessment of joint angles (García-Rubio et al., 2020) and hamstring extensibility (Muyor, 2017).

Flexibility measurements can be made passively, when an external force causes the movement through the range of motion, or actively, when the individual uses muscle action to produce the movement. Testers should note which technique is used because passive measurements are generally higher than active ones (Plowman, 1992).

Field methods for measuring flexibility typically involve linear distances between segments or from an external object. Variations of the stand or sit-and-reach test are the most popular field test of flexibility. To perform the sit-and-reach test, the individual sits with one (now recommended) or both (previously used) stockings flat against a testing box. The hands are positioned fully extended from the shoulders onto a scale on top of the testing box. The individual flexes as far forward as possible in a controlled fashion, sliding the fingertips along the scale. The point of maximum reach is recorded.

From the mid-1940s until the mid-1980s, the stand or sit-and-reach test was described as a test of low back (lumbar) and hip (hamstring) flexibility, mobility, or extensibility. In the mid- to late 1980s, several studies were published showing clearly that although the stand or sit-and-reach test is a valid test of hamstring flexibility, it is *not* a valid test of low back flexibility (Biering-Sorensen, 1984; Jackson and Baker, 1986; Jackson and Langford, 1989; Kippers and Parker, 1987; Nicolaisen and Jorgensen, 1985). A recent meta-analysis of 34 separate studies (Mayorga-Vega et al., 2014) has confirmed these early individual

findings.

Because of its widespread use as the only flexibility test in physical fitness test batteries, the sit-and-reach test has often been misinterpreted as a measure of total body flexibility. That is, if an individual has good flexibility in this test, equally good flexibility is assumed in other joint-muscle units. Although this idea is appealing in terms of simplicity and ease of testing, it is not accurate (Clarke, 1975; Shephard et al., 1990). Joint flexibility is highly specific to individual locations; it is not a general trait common to all joints. Therefore, if the goal of testing is to determine whether an individual is flexible, a profile of major joints must be compiled because no one test indicates total body flexibility (de Araújo, 2004). Joint specificity for flexibility is true throughout the age span of childhood to old age.

The Influence of Sex and Age on Flexibility

Male-Female Comparisons

The specificity of flexibility to each joint makes it difficult to generalize about sex differences. One study has shown that across the entire age spectrum from 10 to 75 years, males exhibited greater anterior trunk flexion (also called lumbar mobility or low back flexibility) than females (**Figure 20.15A**). Conversely, across most of the same age span, females exhibited greater right lateral trunk flexibility than males (**Figure 20.15B**). Although not shown in this figure, females also had greater left lateral flexibility than males, at least in adults.

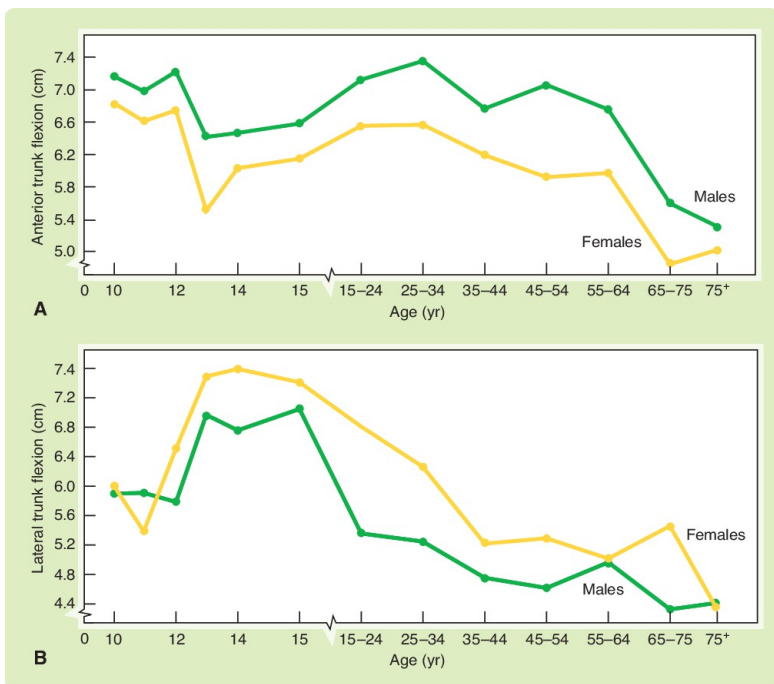


Figure 20.15 Flexibility in Males and Females.

A. Anterior trunk flexion (lumbar mobility). **B.** Lateral trunk flexibility. **Sources:** Plotted from data of [Moran et al. \(1979\)](#) and [Moll and Wright \(1971\)](#).

The differences graphed in **Figure 20.15A and B** did not reach statistical significance at all ages and/or were not always tested for significance, so it may be that there really aren't any differences in flexibility between the sexes. Either way, these observations contradict the usual assumption that females are more flexible than males. Other data have shown that adult males are more flexible than adult females in trunk extension ([Moll and Wright, 1971](#)) and left and right trunk rotation ([Gomez et al., 1991](#)). However, adult males and females do not significantly differ in trunk flexion, trunk extension, left or right lateral trunk flexion ([Gomez et al., 1991](#)), left or right head rotation, external shoulder rotation, or plantar or dorsi ankle flexion. Adult males are less flexible than adult females in internal shoulder rotation and hip flexion ([Sullivan et al., 1992](#)).

The often-stated but apparently insupportable assumption that females are more flexible than males can probably be attributed to results from the sit-and-reach test. Because studies have shown that females have greater hip flexibility (Shephard et al., 1990), females would be expected to score better than males on the sit-and-reach test. **Figure 20.16** clearly shows that from 5 to 18 years, girls do have a higher sit-and-reach score than boys.

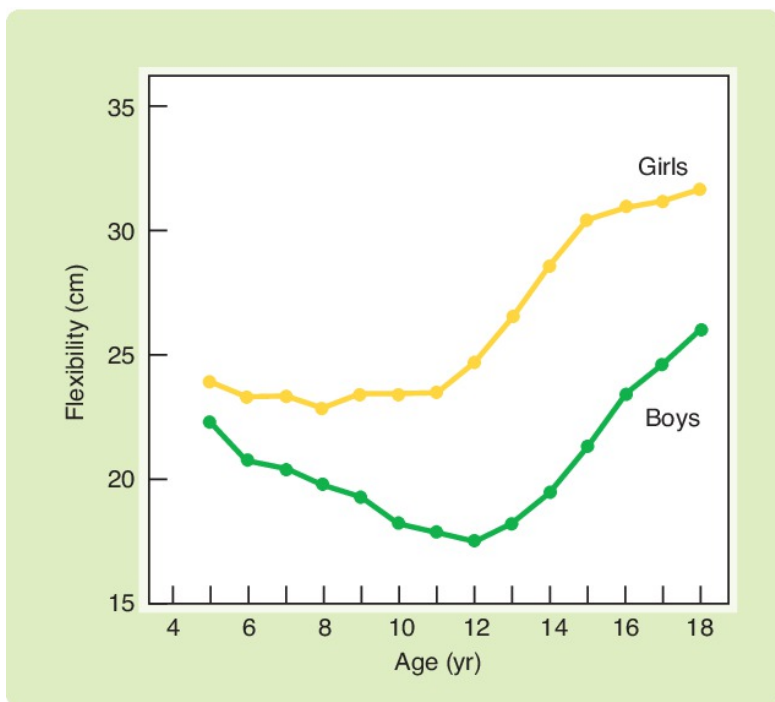


Figure 20.16 Flexibility as Measured by the Sit-and-Reach Test.

Source: Reprinted with permission from Malina, R. M., C. Bouchard, & O. Bar-Or: *Growth, Maturation and Physical Activity* (2nd ed.). Champaign, IL: Human Kinetics (2004); Data from Michigan State University Motor Performance Study.

Despite the sit-and-reach results, the results of many other studies show there is no consistent generalized pattern of sex

differences in flexibility. Depending on which joint is being measured, females may have a larger, equal, or smaller range of motion than males.

Influence of Age

The impact of age on flexibility is only slightly less confusing. **Figure 20.15A and B** illustrates specificity through the pubertal growth years. The general trend is for lumbar flexibility to decrease between 10 and 15 years of age, while lateral flexion generally increases during the same period. The sit-and-reach data (**Figure 20.16**) show an interaction of age and sex (Malina and Bouchard, 1991). Basically, girls show a consistent improvement from age 5 to 18 years, while boys show a U-shaped response—that is, a gradual decline from age 5 to 13 years and then an improvement from 13 to 18 years, such that they are more flexible in the hip and posterior thigh by adulthood. Whether and how range of motion changes through the growing years is, therefore, joint specific.

Although **Figure 20.15** shows a pattern of declining flexibility as people progress from young adulthood through middle age and into old age, other studies investigating different joints suggest that flexibility does not change (Gomez et al., 1991; Shephard et al., 1990). An increase in range of motion with age through the adult years has not been shown to occur without training. Therefore, flexibility either declines or stays the same through the adult years. This is probably joint specific in terms of both direction and magnitude. Changes in flexibility with aging may also be confounded by the decreasing activity that often accompanies aging. Most flexibility studies are cross-sectional rather than longitudinal, and this information has not been analyzed taking activity level into account.

All ages appear to be trainable in terms of flexibility (Clarke, 1975; Rider and Daly, 1991). This adaptation may be especially important to the elderly, for whom healthy, independent living is at stake (Zhou et al., 2019).

Flexibility and Low Back Pain

The theoretical link between muscle function and low back pain

(LBP) was described in [Chapter 19 \(Table 19.5\)](#), with particular emphasis there on muscle strength and endurance. Here, the emphasis is on the flexibility needs for a healthy, well-functioning back. Flexibility of the low back and hip area and strong and balanced lumbar, hamstrings, and hip flexor muscles are crucial for controlled pelvic movement. Controlled pelvic movement means having neither an exaggerated anterior tilt (lordotic curve) nor a restricted anterior tilt (no low back curvature). Either an exaggerated or a restricted pelvic tilt can increase vertebral disc compression and cause pain and strain in the low back area.

Recent research utilizing shear wave elastography has shown that individuals suffering from LBP have higher resting passive muscle stiffness of the erector spinae and superficial multifidus fibers than asymptomatic individuals ([Koppenhaver et al., 2019](#); [Murillo et al., 2019](#)). Typically LBP sufferers have limited range of motion in the low back and hamstring areas. However, the more important question is whether LBP can be predicted and remediation steps taken before acute and/or chronic pain becomes a problem. A 2017 meta-analysis ([Sadler et al., 2017](#)) involving 5,459 participants revealed that reduced lateral flexion range of motion (ROM), limited lumbar lordosis, and restricted hamstring ROM were significantly associated with the development of LBP. However, lumbar extension ROM, quadriceps flexibility, fingertip to floor distance, and lumbar flexion ROM showed nonsignificant results.

What exact values constitute too low or too high a range of motion are unknown. From the existing research, however, it can be recommended that flexibility of the hip, low back, and hamstrings be part of a general overall fitness program. Flexibility should be developed specifically for athletes as needed. However, flexibility should not be taken to extremes, nor should one expect that flexibility will mean absolute protection from LBP.

Stretching Techniques to Improve Flexibility

The biggest decision in setting up a *flexibility training program* is choosing a stretching technique. Four techniques have been used

to increase flexibility: ballistic stretching, dynamic stretching, static stretching, and proprioceptive neuromuscular facilitation (PNF). **Ballistic stretching (BS)**, characterized by an action-reaction bouncing motion, is a form of stretching in which the joints involved are moved to the extremes of the joint range of motion by fast, active contractions of agonistic muscle groups. As a result, the antagonistic muscles are stretched quickly and forced to elongate. This quick stretch distorts the intrafusal fibers of the neuromuscular spindle and activates the annulospiral nerve endings, which transmit an impulse to the spinal cord, resulting in an immediate strong reflex contraction (the myotatic reflex) of the muscles that had been stretched (Etnyre and Lee, 1987). This rebound bounce is proportional in force and distance to the original move. Although ballistic action occurs frequently in sports—for example, punting a football or a high kick in dance—and although ballistic stretching has been shown to be effective in increasing flexibility, this type of stretching is generally not recommended. Ballistic stretching may cause muscle soreness. In addition, although virtually no research or clinical evidence supports this, some fear that the forces generated by the series of pulls will exceed the extensibility limits of involved tissues and cause injury (Shellock and Prentice, 1985). Fortunately, alternative means of increasing flexibility do not invoke this (real or imagined) fear of injury.

Ballistic Stretching A form of stretching, characterized by an action-reaction bouncing motion, in which the involved joints are placed into an extreme range of motion by fast, active contractions of agonistic muscle groups.

Dynamic stretching (DS) involves moving the limb from its neutral position to its end range, where the muscles are at their greatest length, and then moving the limb back to its original position in a smooth, controlled manner for a specified time period. Typically, dynamic stretching consists of functional-based exercises, which use sports-specific movements that often work on balance at the same time utilizing both concentric and eccentric contractions. Dynamic stretching has the advantage of

allowing for a similarity between the stretching and exercise movement patterns without the rebound from the forceful activation of the neuromuscular spindle brought about by ballistic stretching. Conversely, it does not benefit from the relaxation brought about by the firing of the Golgi tendon organ as the movement is never stopped and held. However, the static component of the neuromuscular spindle that opposes excess lengthening and the feedback of sensory information to the cerebellum from the GTO that smooths the beginning and ending of each movement should occur. Dynamic stretching can elevate core temperature with all the positive physiological benefits of increased nerve conduction velocity, increased muscle compliance, decreased inhibition of antagonistic muscles, increased enzyme activity, and accelerated energy production that result (Behm et al., 2016a, 2016b). Examples of dynamic stretching activities include forward, backward and side lunges, inchworm, and arm circles. Many of these stretches can be performed either stationary or moving.

Dynamic Stretching A form of stretching that involves moving the limb from its neutral position to its end range, where muscles are at their greatest length, and then moving the limb back to its original position in a smooth, controlled manner.

Static stretching (SS) is a form of stretching in which the muscle to be stretched (the antagonist) is slowly put into a position of controlled maximal or near-maximal stretch. The position is held for 10–30 seconds. Because the rate of change in muscle length is slow as the individual gets into position and then stops as the position is held, the annulospiral nerve endings of the neuromuscular spindle (NMS) are not stimulated to fire, and a strong reflex contraction does not occur. That is, the dynamic phase of the NMS response is bypassed. Instead, if the stretch continues for at least 6 seconds, the Golgi tendon organs (GTOs) respond, leading to the inverse myotatic reflex and causing relaxation in the stretched muscle group. This response is called *autogenic inhibition* (Etnyre and Lee, 1987). This relaxation is easily felt by the exerciser, and it allows the muscle to be

elongated even further. The impulses from the GTO can override the weaker static response impulses coming from the NMS to allow this reflex relaxation and a continuous sustained stretch. If a maximal stretch is held long enough, the muscle being stretched ultimately reaches a point of **myoclonus**—twitching or spasm in the muscle group—indicating the endpoint of an effective stretch. Because no uncontrolled sudden forces are involved, injury is unlikely with this type of stretch ([Shellock and Prentice, 1985](#)).

Static Stretching A form of stretching in which the muscle to be stretched is slowly put into a position of controlled maximal or near-maximal stretch by contraction of the opposing muscle group and held for 30–60 seconds.

Myoclonus A twitching or spasm in a maximally stretched muscle group.

Proprioceptive neuromuscular facilitation (PNF) is a stretching technique in which the muscle to be stretched is first contracted maximally. The muscle is then relaxed and is either actively stretched by contraction of the opposing muscle or is passively stretched by an outside force. A number of different proprioceptive neuromuscular techniques are currently being used for stretching, but the two most popular are the contract-relax (CR) and contract-relax-agonist-contract (CRAC) techniques. In both the CR and CRAC techniques, the muscle to be stretched (the antagonist) is first placed in a position of maximal stretch by action of the agonist and then is contracted maximally, using either a dynamic concentric or static contraction. Both techniques also require the assistance of a partner or an implement to provide resistance and elongation.

Proprioceptive Neuromuscular Facilitation (PNF) A stretching technique in which the muscle to be stretched is first contracted maximally. The muscle is then relaxed and either is

actively stretched by contraction of the opposing muscle or is passively stretched.

The contraction phase, which originates from the position of maximal stretch, typically lasts for 6–10 seconds. Because of the slow rate of change of the muscle length as the individual gets to the maximal stretch position, the annulospiral nerve endings of the NMS are not stimulated to fire, and no reflex contraction occurs. As with a static stretch, the dynamic phase of the NMS response is bypassed. The exerciser then contracts the antagonists against the resistance provided by a partner. As tension is created in the muscle by the maximal contraction, the Golgi tendon organs respond and the inverse myotatic reflex is initiated, causing a relaxation in the stretched muscle group (Etnyre and Lee, 1987). At this point in the CR technique, the partner who has been resisting the contraction moves the relaxed limb into a greater stretch and holds it for 10–30 seconds. That is, the antagonist is further elongated passively until resistance to the stretch is again felt.

In the CRAC technique, the exerciser actively contracts the agonist to assist the stretching of the antagonist. By reciprocal inhibition, the contraction of the agonist is thought to aid in the relaxation of the antagonist, allowing it to be stretched further. PNF stretching carries some risk of injury if the partner attempts to push the relaxed limb too far. However, if the partner stops at the point of myoclonus, which can be easily felt with proper hand placement, injury should be avoided.

The role of the Golgi tendon organ in bringing about an inhibitory relaxation of stretched muscle has generally been supported by research studies (Etnyre and Abraham, 1988; Etnyre and Lee, 1987; Hutton, 1992). When needle electrodes were implanted in the stretched muscles, very little EMG activity was recorded, indicating relaxation.

Table 20.2 summarizes the four stretching techniques. Although you have probably used all four techniques in the past, try them again now as described below for the hamstring muscles. Be very conscious of what you are feeling and why.

TABLE 20.2 Summary of Stretching Techniques

| | Ballistic | Dynamic | Static | PNF:CR | PNF:CRAC |
|---------------|--|---|---|---|---|
| Action | Antagonist stretched by dynamic contraction of agonist | Antagonist stretched by dynamic contraction of agonist | Antagonist moved slowly to limit of ROM and held | Antagonist moved slowly to limit of ROM by action of agonist, where it contracts maximally for about 6–10 sec | Antagonist moved slowly to limit of ROM by action of agonist, where it contracts maximally for about 6–10 sec |
| Reaction | Bounce back proportional to force of original contraction | Low-level static contraction opposing lengthening; sensory information provided to cerebellum to smooth muscle action | Relaxation and further elongation, usually gravity assisted | Relaxation and further passive elongation by partner or implement, such as towel or jump rope | Relaxation and further passive elongation by active dynamic concentric contraction of agonist |
| Mechanism | Neuromuscular spindle (myotatic reflex) | Neuromuscular spindle and Golgi tendon organ | Golgi tendon organ, autogenetic inhibition (inverse myotatic reflex) | Golgi tendon organ, autogenetic inhibition (inverse myotatic reflex) | Golgi tendon organ, autogenetic inhibition (inverse myotatic reflex) plus reciprocal inhibition |
| Advantages | Improves flexibility; may mimic action in sports performance | Improves flexibility; increases body temperature | Improves flexibility and is safest; may provide relief from delayed-onset muscle soreness | Improves flexibility and is safe | Improves flexibility and is safe |
| Disadvantages | Muscle soreness or injury may result | | | Requires partner or implement, such as towel, jump rope, or sweats | Requires partner or implement |

Note: ROM, range of motion.

- 1. Ballistic:** stand with your feet shoulder's width apart, legs straight, and quickly attempt to place your palms (or if that is easy, your elbows) on the floor. You should bounce back up, but might not have if you didn't go down forcefully because you didn't want to pull your hamstring. Review the action of the NMS in your mind.
- 2. Dynamic:** assume a full push-up position. Walk your feet to your hands, keeping your legs straight. Walk your hands out to the push-up position again in inchworm fashion. Continue for 10 yards.
- 3. Static:** stand up with your feet shoulder's width apart, legs straight but knees not locked, and bend over at the waist with your head and arms dangling down. Hold that position until you feel your hamstring muscles relax and allow you to bend even further. Repeat until your hamstrings start to quiver (myoclonus) or you can't go any further. If you can easily touch the floor initially, stand on a stable box that allows you to reach beyond your feet. Think about the interaction of the GTO and NMS.

4. Proprioceptive neuromuscular facilitation: lie supine on the floor and place a towel around the heel of one foot so that you can pull on it. Elevate that leg straight until you feel resistance. Statically, contract the hamstrings for 6 seconds against the resistance being provided by your towel. Then pull on the towel with your arms to further stretch the hamstrings. This is the CR PNF technique. From the new position, repeat the static contraction for another 10 seconds. This time, stretch the hamstrings by actively contracting the quadriceps of that leg. This is the CRAC PNF technique. Think about the interaction of the NMS, GTO, and reciprocal inhibition needed to perform these actions.

Work through the exercises provided in the [Check Your Comprehension box](#) to ensure that you understand these concepts.

CHECK YOUR COMPREHENSION 1

Describe an exercise using each of the following techniques for the gastrocnemius (calf) muscle. Then perform the exercise.

1. Ballistic
2. Dynamic
3. Static
4. CR PNF
5. CRAC PNF

Check your answers in Appendix C.

Acute Physiological Response to Stretching

Coaches and exercise professionals often recommend stretching before activity to improve performance, prevent injury, and prevent delayed-onset muscle soreness (DOMS). Unfortunately,

these recommendations, though common, are not entirely based on research evidence. This section describes the acute effects of stretching on flexibility, performance, injury protection, and the sensation of DOMS. The evidence may surprise those of you who are familiar with long-standing traditions in this area. As you read, it is important to distinguish between acute responses to preexercise stretching and a general chronic program of flexibility training done at another time in terms of positive versus negative responses or adaptations.

Range of Motion

The obvious and well-known response to an acute bout of stretching exercises is increased range of motion of muscles around a joint—an increase in flexibility. To achieve this, muscle relaxation actually occurs within the sarcomeres. Evidence seems to suggest that this relaxation is the combined result of a decline in passive tension that results from the mechanical viscoelastic properties of the muscle and the neural actions for the inverse myotatic reflex ([Gleim and McHugh, 1997](#); [McHugh et al., 1999](#); [Smith, 1994](#)). All types of stretching appear to result in an increased range of motion in individuals of all ages including older adults, with no clear evidence suggests that one type of stretching (BS, SS, DS, PNF) is better than the others ([Behm et al., 2016a, 2016b](#); [Cayco et al., 2019](#); [Fields et al., 2007](#); [Iwata et al., 2019](#); [Larouche and Connolly, 2006](#); [Zhou et al., 2019](#)). Holding a SS or PNF stretch for 15–30 seconds appears to be more effective than shorter periods, but there is no apparent additional benefit for longer duration stretches. The increased range of motion resulting from an acute bout of stretching lasts for approximately 30–120 minutes ([Behm et al., 2016a, 2016b](#); [Fields et al., 2007](#); [Fowles et al., 2000](#); [Power et al., 2004](#)).

Performance

Many coaches and exercise leaders suggest that preexercise stretching can enhance performance. However, the scientific literature appears to contradict this claim when the performance has a major strength or power component. This phenomenon has

been termed the *stretching-induced force deficit* (Fowles et al., 2000). Indeed, in 2006, the European College of Sports Medicine (Magnusson and Renström, 2006) concluded that there was firm evidence that an acute bout of stretching could diminish performance in tests requiring maximal muscle efforts, and the American College of Sports Medicine (ACSM, 2010, 2014) suggested removing static stretching from a warm-up if strength and/or power were important to performance. It should be noted that the ACSM recommendation was based on references from 2010 to 2011. Now there is a larger body of evidence and it has been reexamined.

Evidence also suggested that SS protocols as part of a warm-up routine might adversely affect endurance performance. A 2010 study reported that 16 minutes of static stretching prior to a 60-minute treadmill run significantly decreased performance and increased the energy cost of exercise (Wilson et al., 2010). Similarly, a 2014 study found that static stretching, compared to a dynamic treadmill warm-up, significantly decreased performance on a 1-mile uphill run (Lowery et al., 2014).

A 2012 systematic review (Kay and Blazevich, 2012) on the effect of acute static stretch (SS) on maximal muscle performance determined that in 149 findings from 106 articles, only 44% indicated significant reductions in maximal strength-, power-, or speed-dependent performance with a pooled reduction of -3.7% . More importantly, there was a curvilinear relationship between stretch duration and the mean reduction. The magnitude of the reduction was -1.1% for less than 30-second stretch, -1.9% for 30- to 45-second stretch, -4.2% for 1- to 2-minute stretch, and -7.0% for greater than 2-minute stretch. The decrement did not continue to increase beyond 2 minutes. Thus, there appears to be clear evidence that there is basically no performance decrement in strength, power, or speed dependent tasks with short stretch durations. In general, where decrement was seen, it was higher for strength-dependent than power- or speed-dependent tasks. Additionally, significant improvements in jumping, cycling, and sprinting performance were reported with short duration stretches. The authors concluded that SS totaling less than 45 seconds can be used in preexercise routines without risk of significant decreases in strength-, power-, or speed-dependent performance.

A 2013 meta-analysis ([Simic et al., 2013](#)) of 104 studies showed that an acute bout of preexercise SS decreased maximal muscle strength by -5.4% , muscle power by -1.9% , and explosive muscular performance by -2.0% . Further, it was shown that the acute effects of SS were task specific in that maximal isometric tests were more affected than maximal dynamic tests. Finally, as with the Kay and Blazevich review, the smallest negative acute effects were evident with a stretch duration of ≤ 45 seconds. These findings were universal, that is, the same regardless of the individual's sex, age, or training status. Acknowledging that SS also has certain positive acute effects during warm-up, such as increased range of motion and reduced incidence of muscle strains, the authors recommended that although the use of SS as the only activity during warm-up should be avoided, its incorporation into a comprehensive warm-up could be a practical way to minimize the negative effects while maintaining the positive effects on performance.

A 2016 systematic review ([Behm et al., 2016a, 2016b](#)), acknowledging the shift away from SS and/or proprioceptive neuromuscular facilitation (PNF) stretching within a warm-up to a greater emphasis on dynamic stretching (DS), compared the effects of SS, PNF, and DS on performance. Data from 125 studies revealed a -3.7% performance reduction from SS, a -4.4% reduction from PNF stretching, but a $+1.3\%$ increase with DS in performance immediately (3–5 minutes) after stretching. As with the other two major reviews, the dose-response relationship again showed that longer static stretches (>60 seconds) were more detrimental than shorter stretches. However, in studies that conducted tests greater than 10 minutes after stretching, performance changes were typically statistically trivial unless extreme stretch protocols were used.

Finally, there is a strong body of evidence supporting the positive or neutral effects of dynamic stretching on range of motion and subsequent muscular force, power, jump, and sprint performance ([Opplert & Babault 2018](#)). These effects appeared to be amplified when the stretches were performed while walking and the length of the stretches (at least until fatigue intervened) was not important. Dynamic stretching was found to be more efficient than static stretching and more beneficial than ballistic stretching.

It is important to note that the effects of SS on performance are influenced by the proximity of the SS bout relative to exercise as well as the duration of SS itself (see the Focus on Research Box). For example, when short duration SS (<60 seconds) is performed in combination with other warm-up techniques (aerobic exercise, dynamic stretching, sport specific exercises), SS shows trivial decrements in strength performance. However, when performed for longer duration immediately prior to strength training, data suggest that SS may induce significant reductions in strength performance. However, although not universally agreed upon, SS has also been shown to improve muscular flexibility and reduce the risk of musculotendinous injury (Chaabene et al., 2019). Therefore, the question whether SS should be included as part of a warm-up prior to exercise is dependent on the proximity of SS to exercise, the combination of warm-up techniques used, as well as the desirable outcome from the particular exercise bout.

Taken together, these comprehensive analyses have the same takeaway message. Short bouts of SS or PNF stretching ($\leq 45\text{--}60$ seconds) per muscle group per limb can be incorporated into a comprehensive warm-up (aerobic activity, stretching, performance-related dynamic activity) at least 10 minutes before training or competition. DS may be done closer to the time of the event.

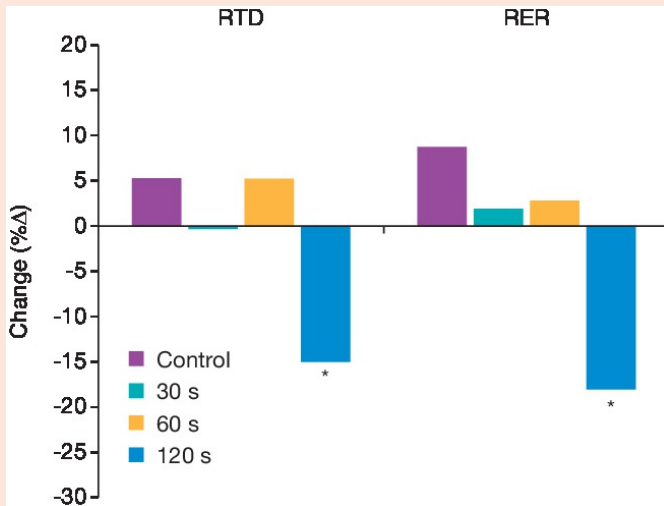
There are several primary hypotheses to explain stretching-induced force deficit if it occurs. One suggests that mechanical factors within the musculotendinous unit, such as decreases in muscle stiffness or a deformation in the connective tissue, may limit the muscles' maximal force production. A second proposes that contractile fatigue caused by reduced blood flow and oxygen availability may be responsible. A third hypothesis proposes that central nervous system factors, such as activation of motor units or reflex sensitivity, is altered by static stretching in a way that impairs maximal force production (Behm et al., 2001, 2016a, 2016b; Herda et al., 2008). The lack of stretch-induced force deficit caused by dynamic stretching is possibly related to the fact that changes in musculotendinous stiffness are not as great during DS as in SS or PNF. Conversely, the muscle contractions could induce postactivation potentiation (an increase in muscle force and rate of force development that occurs as a result of previous

activation of the muscle) or be related to temperature increases (Kallerud and Gleeson, 2013).

FOCUS ON RESEARCH

Should Weight Lifters Stretch during Their Warm-Up?

For many years, including static stretching as part of a warm-up prior to lifting has been the norm. Some reasons for the inclusion of static stretching include increasing range of motion and reducing risk of injury (although there is not much evidence to support a reduced injury risk). However, studies suggest that this may not be ideal, since stretching prior to training may decrease an individual's ability to produce force, and thus, decrease performance. A 2019 study evaluated the effects of different static stretching durations (30, 60, or 120 seconds) on the physical performance of the hamstrings. Thirteen female participants performed two isometric maximal voluntary contraction of the hamstrings after each of the stretching periods. The results demonstrated that the 120 seconds stretching protocol resulted in reduced rapid strength (rate of torque development [RTD]) and power development (rate of EMG rise [RER]). However, decrements in performance were not observed in the 30 or 60 seconds stretching conditions. Overall, these results demonstrate that the potential negative effects of static stretching prior to resistance training are intensity and duration dependent. Perhaps, short stretching periods of approximately 30–60 seconds may not be detrimental to strength and power output and can be included as part of a general warm-up technique.



Source: Adapted with permission from Palmer T. B., J. G. Pineda, M. R. Cruz & C. C. Agu-Udemba: Duration-dependent effects of passive static stretching on musculotendinous stiffness and maximal and rapid torque and surface electromyography characteristics of the Hamstrings. *Journal of Strength & Conditioning Research*. 33(3):717–726 (2019). Copyright © 2019 National Strength and Conditioning Association..

Health

Flexibility is one of the four major components (along with strength, muscular endurance, and power) of the musculoskeletal portion of Health-Related Physical Fitness (HRPF) ([Chapter 1](#), [Figure 1.5](#)). As such, flexibility is important to health. A principal component analysis of musculoskeletal, cardiovascular, and body composition elements of HRPF showed that all of these elements except flexibility were strongly associated with each other. Therefore, flexibility represents something unique ([Dumith et al., 2012](#)), and it may/may not relate to health differently than do the other elements. Despite, or possibly, because of this uniqueness historically little research has even attempted to document specific cardiovascular, metabolic, and/or body composition benefits of flexibility. This prompted the Institute of

Medicine (as indicated in [Chapter 1](#)) to exclude flexibility from the recommended core fitness test items suitable for use in a national survey of youth fitness ([Institute of Medicine, 2012](#)). [Nuzzo \(2020\)](#) presented a case for retiring flexibility (and specifically the sit-and-reach test) as a major component of HRPF and stretch training primarily (but not exclusively) based on the fact that flexibility has little relationship to cardiovascular health, is not related to mortality, and stretch training is not the only activity that can improve flexibility. The idea that a cardiovascular component may be involved in human skeletal muscle stretching is relatively new ([Kruse, 2020](#); [Kruse & Scheuermann, 2017](#)). However, emerging evidence suggests that both acute and/or chronic stretching exercise may bring about a variety of cardiovascular responses.

At the onset of a stretch, the mechanical deformation of the vascular bed coupled with stimulation of muscle afferent nerve fibers results in peripheral vasodilation, a heart-rate driven increase in cardiac output, blood pressure, and muscle blood flow ([Kruse & Scheuermann, 2017](#)).

The increase in heart rate is both minimal ($4\text{--}5\text{ b}\cdot\text{min}^{-1}$) and transient (lasting only 30–60 seconds) before decreasing during recovery. Although some evidence suggests that acute stretching can reduce systemic BP in recovery ([Kruse & Scheuermann, 2017](#); [Kruse et al., 2016](#)), this relationship remains inconclusive. However, one longitudinal study that followed 14,805 males and 8,167 females (median age 49 year), all initially normotensive, showed an interesting predictive ability. Each individual was tested with the standing forward bend and then followed for a median of 5.6 year. During that time, 4,235 individuals developed hypertension. A high initial level of flexibility was associated with a lower incidence of developed hypertension, independent of other confounding factors ([Gando et al., 2020](#)).

Although stretching does not appear to decrease blood flow at the arterial level, flow may be impeded within the microvasculature in a muscle-intensity stretch-dependent manner. Upon release of the stretch, a large increase in blood flow occurs ([Kruse & Scheuermann, 2017](#); [Kruse et al., 2016](#)). Vascular endothelial function and peripheral circulation have been shown to improve in patients with acute myocardial infarctions with a

single session of stretching exercise ([Hotta et al., 2013](#)).

Arterial stiffness (the ability of arteries to expand and recoil with cardiac pulsation and relaxation) is functionally related to vascular tone of arteries and as such is positively related to cardiovascular risk; greater stiffness equals greater risk. Arterial stiffness is measured as pulse wave velocity (PWV) between specific arteries. A body of evidence is building that suggests that arterial stiffness is reflected in flexibility scores (that is, poor sit-and-reach scores are associated with high arterial stiffness and vice versa) and acute stretching decreases arterial stiffness temporarily ([Gando et al., 2017](#); [Komatsu et al., 2017](#); [Kruse & Scheuermann, 2017](#); [Logan et al. 2018](#)).

Finally, heart rate variability (HRV) is affected by stretching. HRV is the change in the interval between consecutive R waves on the electrocardiogram and can be used to determine the balance between the sympathetic and parasympathetic neural control of the heart. A reduction in HRV is associated with increased risks of cardiovascular events (heart attacks and strokes), especially in the postexercise recovery period. Stretching has been shown to positively impact HRV undoubtedly due to increased parasympathetic activity during the stretch and decreased sympathetic activity after stretching ([Farinatti et al., 2011](#); [Kruse & Scheuermann, 2017](#); [Wong & Figueroa, 2021](#)).

Injury Prevention

Exercise professionals routinely recommend preexercise stretching to prevent injury. The rationale for this is that stretching increases the compliance (decreases the stiffness) of the tendon unit and thus theoretically makes it less prone to injury ([Witvrouw et al., 2004](#)).

However, despite this theoretical rationale and current widespread recommendations, the precise effects of preactivity stretching on injury are not fully documented. A recent review ([Behm et al., 2016a, 2016b](#)) found only 12 studies that investigated the effect of preactivity stretching on injury risk. Neither static stretch nor PNF stretch showed an overall effect on all-cause injury or overuse injuries. There was an indication of a benefit in reducing acute muscle injury with running, sprinting,

or other repetitive contraction activities. There was also limited evidence indicating a potentially greater effect of static stretching and PNF stretching on injury risk if the stretching duration was greater than 5 minutes total and it involved task-related multiple muscle groups. There was no evidence as to whether dynamic flexibility influences injury risk or not. Importantly, there was no evidence that stretching increased injury risk.

A systematic review evaluating the association between flexibility and musculoskeletal injury in military and civilian athletic populations found that there was moderate yet conflicting evidence for a link between hamstring and/or ankle flexibility and injury. One high-quality study determined that athletes with poor hamstring flexibility had an approximately 30% greater risk of injury than those with high hamstring flexibility. A second good quality study found a similar relationship in male basic Army trainees, but not females. Finally, a third good quality study also on Army trainees revealed that both the individuals with the highest and lowest flexibility scores were more likely to suffer a hamstring injury than those with average flexibility. The evidence remains equivocal on the influence of flexibility on training and sport-related injuries (De La Motte et al., 2019).

Delayed-Onset Muscle Soreness

Static stretching has been advocated by coaches and exercise professionals as a way to prevent or minimize delayed-onset muscle soreness (DOMS). Unfortunately, the research literature does not support this contention. Meta-analyses investigating the effect of stretching immediately before or after exercise on subsequent delayed muscle soreness found that stretching had no significant effect on soreness (Herbert and Gabriel, 2002); (Herbert and de Noronha, 2007). Similarly, although the number of studies is low, neither postexercise PNF stretching nor dynamic stretching have been shown to prevent or significantly reduce the symptoms of DOMS (Krityakiaran et al., 2014; McGrath et al., 2014).

In summary, acute stretching increases range of motion around a joint. Acute stretching also appears to elicit several

beneficial cardiovascular changes. However, research suggests that stretching immediately before exercise may result in force decrements that impair other aspects of performance. Stretching does not appear to prevent or minimize muscle soreness. Overall, the cost-benefit ratio of muscle stretching preexercise seems to favor muscle stretching in some form in terms of performance, range of motion, and injury outcomes, but the type of stretching chosen and the make-up of the stretch routine must depend upon the context in which it is used. That is, neither static nor PNF stretching is recommended if prolonged stretching (>60 seconds total per individual muscle) is conducted within 5 minutes of training or competition unless the necessity for an increase in range of motion and/or possible reduction in injury outweigh the requirement for optimal performance (Behm et al., 2016a, 2016b).

Therefore, at this point it appears that preexercise bouts of acute stretching should not also be used as individual chronic training bouts for the development of flexibility. The best advice is to conduct flexibility training at times other than before exercise.

Application of the Training Principles to Flexibility Training

The application of the training principles to flexibility development has not received as much research attention as has the application of training principles to aerobic training programs and resistance training programs. Nonetheless, there is sufficient evidence of the benefits of flexibility training that the American College of Sports Medicine recommends that flexibility training be included in a well-rounded fitness program (ACSM, 2011, 2022). Guidelines for developing a flexibility training program are given in the following sections. The cardiovascular changes will occur naturally as the stretching is done.

Specificity

Flexibility is joint specific (Marshall et al., 1980; Shephard et al.,

1990) and, therefore, is also task or sports specific. The first step in developing a flexibility program is to analyze the task or sports to determine the degree of flexibility needed, the specific joint(s) involved, and the plane of action involved. For example, hurdling requires flexion and extension at both the hip and the knee joints and also hip adduction, abduction, and rotation. Swimming requires the same hip flexibility as hurdling, but instead of knee flexion and extension, swimming requires ankle flexion and extension plus inversion, eversion, and shoulder flexion and extension, adduction, abduction, and rotation (Hubley-Kozey, 1991). A general fitness participant should emphasize a total body workout of the major joints and muscle groups including the shoulder girdle, chest, neck, lower back, hips, posterior and anterior legs, and ankles. Although flexibility training has historically been used by dancers and a few athletes, it is becoming more common place (**Figure 20.17**).



Figure 20.17 Flexibility Training.

The response to stretching is not specific to the type of stretching performed or the type of movement that is to follow. How muscle and connective tissue are elongated does not matter as long as the elongation occurs. Because a movement will be done ballistically does not mean that ballistic stretching work

should be done (Etnyre and Lee, 1987; Hardy and Jones, 1986).

Overload

Overload in flexibility training is achieved by placing the muscle and connective tissue at or near the normal limits of extensibility and manipulating the NMS and GTO by holding the position or contracting the muscle to achieve an elongation or by performing dynamic stretching exercises to the limits of the range of motion. A static stretch should be held between 10 and 30 seconds (ACSM, 2011, 2022; Fields et al., 2007; Knudson, 1998), except for older individuals where a 30- to 60-second stretch may confer greater benefit. PNF stretches with 3–6 seconds of contraction at 20–75% of maximal voluntary contraction, followed by 10–30 seconds of assisted stretch, is recommended (ACSM, 2011, 2022). Dynamic stretching exercises are typically performed for 3–5 repetitions, 10 yd (3 m) of distance, or 15–90 seconds per set for a total of 10–15 minutes of activity (Behm et al., 2016a, 2016b).

A high number of repetitions are not necessary for stretching to be effective. Two to five repetitions are frequently recommended for both the static and PNF flexibility techniques. A reasonable target is to perform 60 seconds of total stretching time for each flexibility exercise (ACSM, 2011, 2022).

The intensity of the stretching exercises should be monitored by both myoclonus and pain. Although stretching to the point of feeling tightness or slight discomfort is good, stretched muscles that begin to twitch or spasm (myoclonus) are stretched too far and are fighting that stretch by reflexively trying to contract. Before proceeding, such a muscle should be shortened to the point where the myoclonus ceases. Pain also means that the stretch is too intense; pain should not be tolerated. Both the rate of stretch and amount of force should be minimized.

The frequency of the workout should be at least 2–3 d·wk⁻¹ in the development phase (ACSM, 2011, 2022; Sharman et al., 2006). However, stretching 3–5 days a week is an effective way to improve flexibility, and reasonable daily stretching should have no detrimental effects. Indeed, daily stretching is associated with the greatest gains in flexibility.

Rest/Recovery/Adaptation and Progression

Short-term improvements in flexibility have been shown to occur after as little as 1 week of daily sessions ([Hardy and Jones, 1986](#)). On the other hand, anecdotal evidence suggests that some people do not improve at all. At any rate, since the individual begins both static and PNF stretching exercises at the limit of extensibility and moves through their range of motion for DS, progression will naturally follow whatever adaptation does occur. Beyond this, methods to optimize progression have not been established. Most importantly, except in the case of specific athletic requirements, progression should not continue to extreme flexibility.

Individualization

As stated previously, the most important consideration in flexibility training is that the individual's goals and technique preferences be considered. In a school program, the maturity of the individuals might also need to be considered. For example, if a PNF technique with a partner is going to be used, the partner has to be able to detect the onset of myoclonus and not try just to push as far as possible. Otherwise, individualization is inherent in the flexibility exercises themselves. Each individual stretches to his or her own limits at his or her own rate. Joint looseness is an individual characteristic ([Marshall et al., 1980](#)).

Maintenance

Once the appropriate or desired level of flexibility has been attained, it can apparently be maintained by just 1 d-wk⁻¹ of training at the same intensity level. These data are based on a study that trained participants three times per week, using five reps of PNF stretching, over a 30-day period. Improvements in flexibility were maintained with only 1 day a week of training, although training three times per week resulted in continued improvements in range of motion ([Wallin et al., 1985](#)).

Retrogression/Plateau/Reversibility

Little is known about when or even if a plateau occurs in flexibility training, although there will be a point, probably set by genetics, when further improvement ceases (Etnyre and Lee, 1987). Improvements in flexibility have been shown to continue for at least 8 weeks after the cessation of exercise (Clarke, 1975).

Warm-Up and Cooldown

Considerable confusion exists about the relationship between warming up and stretching before an activity and flexibility training. A *flexibility training program* is a planned, deliberate, and regular program of exercises that can permanently and progressively increase the usable range of motion of a joint or set of joints over time (Alter, 1988). A *warm-up and cooldown stretching program* is a planned, deliberate, and regular program of exercises that are done immediately before and after an activity, with the intent to improve performance and reduce the risk of injury (Alter, 1988). Considerable evidence shows that a flexibility training program can and does improve flexibility, and some evidence suggests that it may decrease injuries in some activities. However, as previously discussed, stretching immediately before an exercise may cause performance decrements even while improving joint range of motion if the stretch is SS or PNF ≥ 45 –60 seconds.

SS and PNF stretching do not cause an elevation in body temperature and, therefore, do not constitute a warm-up. A cardiovascular warm-up to elevate body temperature should precede stretching exercises regardless of the reason for stretching. The warm-up increases body temperature and, therefore, makes the muscles and joints more viscous and responsive to stretch. Stretching in conjunction with a warm-up is likely more important and beneficial to individuals whose sports or activity requires greater than normal or extreme range of motion (see **Table 20.1**) but not maximal force production. Flexibility training should be done as a stand-alone activity after a warm-up or as part of the cooldown where the structures benefit from the elevated temperatures of the work-out.

Elevated body temperature is believed to decrease the viscous resistance of muscle fibers and tendons, thus leading to increased

range of motion. Increased body temperature is also associated with increased nerve conduction rate, which may be important for complex motor tasks or those tasks that require a fast reaction time (Bishop, 2003). Despite the fact that increased body temperature should make stretching more effective, studies have not shown that flexibility gains after 3–4 minutes of warm-up are any different from flexibility gains after 3–4 minutes of warm-up plus 20–30 minutes of aerobic work (Cornelius et al., 1988).

Adaptation to Flexibility Training

Improved Range of Motion

Stretching exercises and flexibility training improve range of motion, whether the technique used is ballistic, dynamic, static, or one of the PNF techniques (Etnyre and Lee, 1987; Hutton, 1992; Shellock and Prentice, 1985). A randomized controlled clinical trial found that the current ACSM recommendations for flexibility training are effective for improving flexibility in young adults (Sainz de Baranda and Ayala, 2010). Many studies have explored the question of which technique brings about the greatest improvement. All types of stretching appear to be effective in bringing about changes in range of motion around a joint. Stretching programs using PNF techniques twice a week for up to 12 weeks have resulted in increases in range of motion of 21–32 degrees in long-lever hip flexion (Sharman et al., 2006). However, research suggest that PNF stretching techniques are not superior to traditional static stretching for improving range of motion, at least in the hamstrings (Lempke et al., 2018). The physiological basis of training-induced changes in range of motion is not well understood. It has been speculated that relatively permanent anatomical changes occur in connective tissue, but these changes have not been documented. A 2018 meta-analysis of longitudinal static, dynamic, and PNF stretching found that programs of 3–8 weeks do not change either the muscle or the tendon properties, although they did increase the extensibility and tolerance to a greater force. Adaptation to programs shorter than 8 weeks seemed to occur primarily at a

sensory level supporting that idea that changes in neural sensitivity or simply an increase in “stretch perception or tolerance modulation” may be involved ([Freitas et al., 2018](#); [Gleim and McHugh, 1997](#); [McHugh et al., 1999](#); [Sharman et al., 2006](#); [Smith, 1994](#)).

Despite expressed concern about the effect of resistance training on flexibility, little scientific or empirical evidence suggests that resistance training decreases flexibility. In fact, studies have shown that heavy resistance training results in either an improvement or no change in flexibility in some joints. The effects of resistance training on range of motion seem to be movement specific, where joints that are trained under heavy load and full range of motion increase mobility over time, whereas other joints, which are not directly involved in the particular movements performed by the athlete, likely do not improve range of motion and flexibility ([Schoenfeld and Grgic, 2020](#)).

Health

Research on the impact of predominantly static stretch training on cardiovascular variables is growing. A review of studies published up to January 2020 revealed 8 studies where the primary and secondary outcomes were changes from stretching training in hemodynamics (HR and BP), arterial stiffness, and vascular endothelial function in males and females greater than 40 years old. The meta-analysis of these studies ([Kato et al., 2020](#)) revealed that the stretch training (which increased muscle flexibility) decreased resting HR and diastolic blood pressure (DBP) but not systolic blood pressure (SBP) compared with the control groups.

In this same meta-analysis, flexibility training was shown to significantly improve endothelial function compared to the controls. This improvement in endothelial function may be helpful in preventing the progression of atherosclerosis (the build-up of plaque inside blood vessels) ([Kato et al., 2020](#)). In one of those studies ([Hotta et al., 2019](#)), patients with peripheral arterial disease (PAD, which makes walking painful) also improved walking ability.

The primary outcome investigated by [Kato et al. \(2020\)](#) was arterial stiffness. The measurement of arterial stiffness is the most common noninvasive method for detecting atherosclerotic-related changes in the arteries. Arterial stiffness was significantly reduced by stretching exercise training compared with the controls. The authors concluded that “these results suggest that stretching exercises might lead to a comparable reduction in arterial stiffness compared with aerobic exercise” (p. 28).

Finally, two of three separate studies (in healthy male body builders and untrained obese postmenopausal females) found positive changes in heart rate variability (HRV) measures after stretch training through increasing parasympathetic activity and decreasing sympathetic tone. In the obese females, the improvement in sit-and-reach partially explained the neural changes ([Wong & Figueroa, 2021](#)). These training adaptations may be especially important in the aging and clinical populations rather than for young and middle-aged individuals. Stretching can be an alternative, low-intensity intervention for the many individuals who do not have the cardiovascular, metabolic, or psychological ability to do more vigorous aerobic or resistance training. Some exercise is always better than no exercise ([Kato et al., 2020](#); [Kruse & Scheuermann, 2017](#); [Wong & Figueroa, 2021](#)).

Injury Prevention

Muscles, tendons, and ligaments are the tissues injured most frequently in work and in fitness and sports participation. While there are data on flexibility training and injury rates, there is no conclusive evidence that high levels of flexibility (as indicated in the previous section on acute changes) or improvements in flexibility either protect against injury or reduce the severity of injury, including low back pain ([Hart, 2005](#); [Park and Chou, 2006](#); [Plowman, 1992](#); [Shrier, 2004](#); [Small et al., 2008](#); [Thacker et al., 2004](#); [Witvrouw et al., 2004](#)). Conversely, there is some indication that hypermobility, or loose ligamentous structure, may predispose some individuals to injury or low back pain ([Gleim and McHugh, 1997](#); [Plowman, 1992](#)).

Importantly, individuals with poor flexibility for the task they will perform probably have a greater risk of exceeding the

extensibility limits of the musculotendon unit. Such individuals should work on improving their flexibility. Likewise, individuals whose sports may cause maladaptive shortening in certain muscles should perform stretching exercises to counteract this tendency. Individuals who are shown to be hypermobile need to concentrate on strengthening the musculature around those joints.

Therefore, developing methods to reduce the risk of injury in athletes may be an effective strategy to improve long-term performance. Researchers investigated the effect of both dynamic and dynamic plus static stretching prior to training in over 500 high-school soccer athletes ([Zakaria et al., 2015](#)). Dynamic stretching included exercises such as walk and touch right knee to ground, walk and abduct and adduct hip with knee flexed at 90 degrees, and jog and adduct left hip with knee flexed at 90 degrees. The dynamic plus static stretching group performed the same dynamics stretches with the inclusion of static stretching such as quadriceps stretch standing, modified hurdlers stretch seated, and calves stretch standing with a partner. The results showed that there were a total of 17 injuries among those performing dynamic stretching and 20 injuries among those performing dynamic plus static stretching. However, there were no statistically significant differences between the two groups, indicating that neither dynamic nor dynamic plus static stretching protocols were superior to the other for reducing the risk of injury during the regular season ([Zakaria et al., 2015](#)). One major limitation of this study was the lack of a control group. While this study properly demonstrates that the inclusion of static stretching on top of dynamic stretching does not reduce the risk of injury, it does not discern whether stretching itself provides any injury reducing benefits compared to not performing any stretching and should certainly be a topic of further research ([Zakaria et al., 2015](#)).

What advice should an exercise professional give about stretching before an event to decrease risk of injury? At this point, it seems prudent to recognize that stretching has not consistently been shown to prevent injury, but that there is some evidence that it may. However, the evidence clearly suggests that the application of the training principles, discussed throughout this text, is appropriate. In this case, it is particularly important

to progress gradually, that is, to build fitness level progressively as a way to lessen risk of injury, and to accept that individuals may respond to stretching differently.

Delayed-Onset Muscle Soreness

Muscle damage and resulting soreness and performance impairments are common in exercise and sports situations. As discussed in [Chapter 18](#), delayed-onset muscle soreness (DOMS) is characterized by muscle soreness and stiffness that appears approximately 8 hours after exercise and increases and peaks over the next 24–72 hours. Because of the unpleasant sensations of muscle soreness and its interference with subsequent training and performance, there is considerable interest in finding modalities to lessen muscle soreness. As discussed earlier in this chapter, several authors have suggested that an acute bout of stretching may lessen the soreness associated with exercise—especially eccentric exercise. However, research studies have not supported the notion that acute stretching can attenuate muscle soreness. In contrast, a program of flexibility training has been shown to decrease muscle soreness and muscle damage (measured by plasma CK levels) following a bout of eccentric exercise ([Chen et al., 2011](#)). Participants were assigned to a SS, PNF, or control group. Participants in the stretching groups performed stretching activity three times a week for 8 weeks. Both stretching groups improved their range of motion compared to the control group. Furthermore, both groups had smaller decrements in force production following the eccentric exercise than the control group and both stretching groups suffered less soreness. These results demonstrate that a chronic flexibility training program, consisting of either SS or PNF, may be protective against muscle damage and soreness.

Balance

All human movement requires continual muscular adjustments in posture to accommodate changes in the center of gravity as we move. Even the apparently simple act of standing motionless

requires a continual process of minute adjustments of body position to keep the center of gravity over the base of support. As with flexibility, postural control and balance may be classified as static or dynamic. **Static balance** is the ability to make adjustments to maintain posture while standing still, whereas **dynamic balance** is the ability to make necessary adjustments while the center of gravity and/or the base of support are in motion. Clearly dynamic balance is more important during activities, from relatively simple activities like walking to more complex movements like a balance beam routine. Balance has traditionally been viewed as an important component of sports-specific fitness, and in fact, many sports do require a great deal of balance. For example, static balance is essential in archery and other shooting sports; dynamic balance dominates in sports such as snowboarding, skateboarding, and windsurfing; center of mass control is important in climbing, figure skating, and rowing where a narrow area of support exists; regulation of center of mass movement in a predefined direction matters in ballet, dancing, and gymnastics; and side-to-side balance in sports games is important in injury prevention (Zemková, 2014). However, balance is also an essential skill of daily living. Good balance is critical for preventing falls, a leading cause of injury and mortality among older adults. Some authors have, therefore, argued that balance should be included as a component of health-related fitness (Claxton et al., 2006), but at present it is not (Chapter 1).

Static Balance The ability to make adjustments to maintain posture while standing still.

Dynamic Balance The ability to make necessary postural adjustments while the center of gravity and/or the base of support are in motion.

Balance is highly dependent on the nervous system and involves continuous feedback from visual, vestibular, and

somatosensory inputs as well as rapid and coordinated activation of the muscular system. Postural control is a complex motor skill involving multiple sensorimotor processes. The two main functional goals of postural control are postural orientation and postural equilibrium. Postural orientation involves the control of body alignment and tone with respect to gravity, the support surface, the environment, and internal senses. This orientation relies heavily on sensory information received from the visual, somatosensory, and vestibular sensory organs. Postural equilibrium involves the integration and coordination of sensorimotor strategies to stabilize the body's center of mass during both voluntary and externally triggered disturbances in postural stability (Horak, 2006). When considering factors that affect an individual's risk of falling, it is important to realize that balance is a complex skill influenced by multiple factors. Not surprisingly, no single test adequately assesses balance, and probably no single type of exercise training can improve all components of balance.

Measurements of Balance

The most sophisticated laboratory measurements of static balance include the use of a force platform that can measure movement of the center of pressure (COP) and provide an indication of postural sway (Figure 20.18). Software used with these instruments can calculate various parameters during a stationary balance test, such as area outlined by the center of pressure path, the total distance traversed, and the maximum excursion in a particular plane, which are assumed to provide an indication of balance (Hrysomallis, 2007).

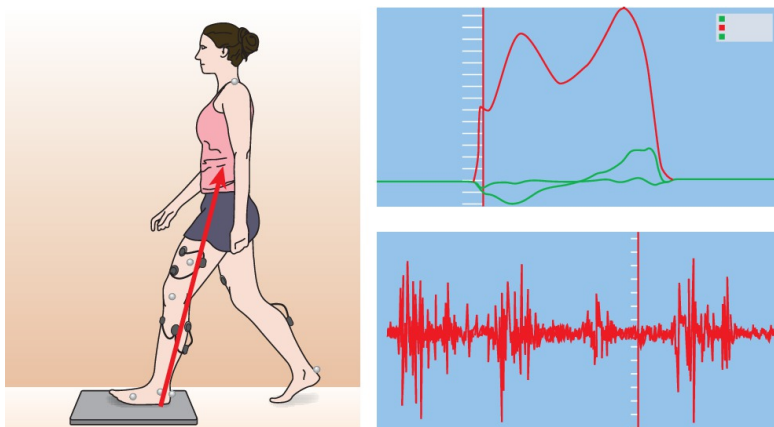


Figure 20.18 Force Platform.

A force platform can be used to measure ground reaction forces at rest or during movement. The force platform provides information about balance and gait and can be integrated with EMG measures.

Clinical scales such as the Balance Error Scoring System (BESS) also evaluate static balance. In BESS, three stances (double-leg support, nondominant single-leg support, and tandem or one foot in front of the other support) are held for 20 seconds, first on a solid floor and then on a medium-density foam pad. Eyes are closed and the hands are held on the hips. The number of specified errors for all six conditions is summed into a single score. Normative percentile data are available for ages 20–70 years (Miller, 2012).

Static field tests are also available. The most common technique is the timed, single-limb balance test. The individual stands one foot on a flat, stable surface with eyes closed for as long as he or she can without moving. Another technique to assess static balance is the flamingo balance test in which balance is assessed in terms of the number of trials it takes for the participant to balance on one leg on a narrow balance beam for 1 minute with eyes open.

Dynamic balance testing typically involves some level of expected movement around a base of support such as jumping or hopping and immediately attempting to remain as motionless as

possible or reaching as far as possible without compromising a base of support. These are considered to be proactive tests. In a reactive test, the individual attempts to regain balance following a disruption by an outside force of normal motion.

Reactive tests can be laboratory based. In this situation, an individual is put into a safety harness and with eyes closed begins walking on a treadmill. At some point, the individual is pushed from behind, an obstruction causes a trip, or a slippery surface causing loss of balance is encountered. In each case, recovery to a stable upright position is judged. If the individual is not able to recover, the safety harness catches them. Such manipulations can also be used as balance training ([Grabiner et al., 2014](#)).

The Modified Bass test is an example of a hopping test in which the individual is required to jump from square to square in a pattern somewhat similar to the old hopscotch layout but using only one leg. Hands must remain on the hips, and upon landing, the individual must look straight ahead and remain motionless. Error points are assessed and normative data are available for balance and landing error totals ([Miller, 2012](#)).

The Star Excursion Balance Test (SEBT) is conducted on a floor marked with a star pattern in eight directions 45 degrees from each other: anterior (A), posterior (P), medial (M), lateral (L), posterolateral (PL), posteromedial (PM), anterolateral (AL), and anteromedial (AM) (**Figure 20.19**). One foot is placed in the middle of this pattern, and the individual attempts to reach as far as possible, sequentially, in all eight directions with just a light tap on the floor with the other foot. The results are adjusted for leg length, and normative values are expressed as a percentage of leg length. The SEBT has been shown to be a reliable and valid dynamic test to predict risk of lower extremity injury, to identify dynamic deficits in individuals with a variety of lower extremity conditions, and to be responsive to training programs in both healthy people and those with lower extremity injuries ([Gribble et al., 2012](#); [Miller, 2012](#)).

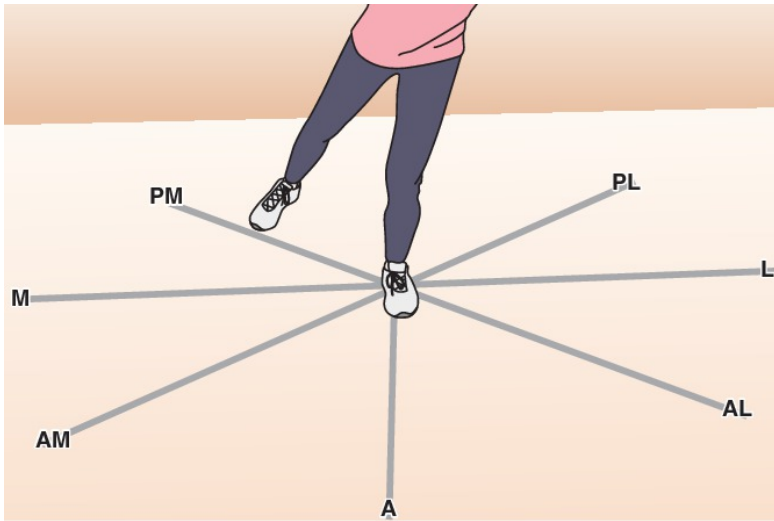


Figure 20.19 Star Excursion Balance Test.

The Influence of Sex and Age on Balance

Balance improves as children mature. Both the sensory and motor processes involved with balance seem to undergo developmental maturation. The influence of proprioceptive function on stance stability has been reported to be completely developed by 4 years, whereas visual and vestibular influence reached adult levels by 16 years (Steindl et al., 2004). Research suggests possible differences between the sexes in balance parameters in children. A study that investigated several balance parameters in children who were approximately 10, 13, and 16 years found that boys exhibited greater and faster movements in the center of pressure than girls in the youngest age group. Furthermore, the boys showed age-related improvements in sway parameters (Nolan et al., 2005).

Balance, its maturational development and decline, is associated with falls. Children and adolescents have more falls that require medical attention than young adults (18–44 years) who exhibit the lowest rate but about as many as middle-aged adults (45–65 years) (Centers for Disease Control and Prevention, 2012). Older adults have an increased risk of falling, making balance a particular concern in this population. Falls are a

common and devastating problem, causing tremendous amounts of morbidity, mortality, and use of health care services, including nursing home admissions (Rubenstein, 2006). One out of three individuals ≥ 65 years falls each year. One out of every five falls causes a serious injury such as broken bones or a head injury. Each year, 2.5 million elderly are treated in emergency departments for fall injuries; more than 700,000 are hospitalized. The age-adjusted fall injury death rate increased from 29.6 per 100,000 in 2000 to 56.7 per 100,000 in 2013 (226.1/100,000 aged ≥ 85 years; 59.0/100,000 aged 75–84 years; 14.1/100,000 aged 65–74 years) in the United States (Centers for Disease Control and Prevention, 2016; Kramarow et al., 2015). In 2015, the direct medical costs of falls were \$637.2 million dollars and \$31.3 billion for nonfatal falls, which is equivalent to roughly \$9,780 per fall (Burns et al., 2016). Given the dramatic effects of a serious fall and related injury on an individual, and the staggering health care costs associated with serious injury, decreasing the risk of falling has become a major public health concern. Good balance depends on feedback from the visual, somatosensory, and vestibular senses and relies on muscle strength, reflex control, and reaction time. With increasing age, a progressive loss of functioning occurs in these systems, causing the increased risk of falling (Carter et al., 2001; Lord and Sturnieks, 2005).

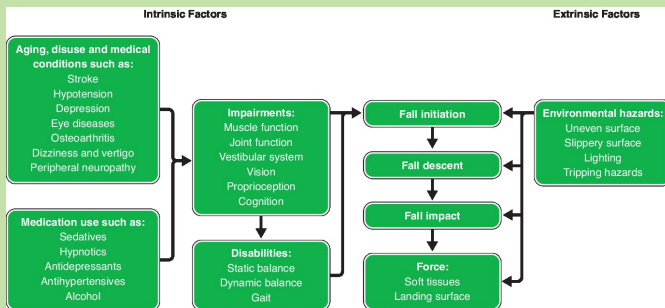
Studies investigating balance in adults routinely report that balance decreases as an individual ages (Carter et al., 2001; Era et al., 2002; Holviala et al., 2006; Punakallio, 2003). A cross-sectional study found that older adults (over 50 years) had a lower functional balance and higher postural sway balance than younger adults (< 39 years) (Punakallio, 2003). A longitudinal study that investigated balance in a group of older adults at age 75 years and again 5 years later (at age 80 years) found that balance deteriorated markedly in both sexes. However, women in this study scored better on the balance test than the men at both points in time (Era et al., 2002). Underscoring the importance of balance, the simple balance test of having individuals stand with their eyes open was a significant predictor of survival over the 5-year follow-up period.

FOCUS ON APPLICATION

A Safe Exercise Environment

The diagram outlines how intrinsic factors (related to the individual) and extrinsic factors (related to the environment) may influence the risk of falling and the risk of serious injury from a fall. This figure distinguishes between impairments (the loss or abnormality of psychological, physiological, or anatomical structure or function) and disability (a restriction or lack of ability to perform an activity in the manner or within the range that is considered normal) (Schuntermann, 1996).

Exercise professionals have a responsibility to do what they can to minimize the risk of falls and injury when working with any individuals, especially older adults or those at risk for falling. This requires understanding intrinsic factors that increase the risk for falling and being mindful of environmental factors (such as a slippery floor, poor lighting) that may increase the risk of injury.



Source: Adapted by permission from Springer: Carter, N. D., P. Kannus, & K. M. Khan: Exercise in the prevention of falls in older people: A systematic literature review examining the rationale and the evidence. *Sports Medicine*. 31(6):427–438 (2001). Copyright © 2012 Springer Nature.

Acute Physiological Responses to Balance Exercises

The vast majority of studies related to balance focus on long-term adaptations in the neuromuscular system related to injury reduction. The short-term physiological responses to a single bout of balance training exercises have received little attention.

Application of the Training Principles to Balance

Balance involves the nervous and muscular systems and is influenced by flexibility and strength; therefore, balance training programs can be expected to follow the well-established training principles with important specific indications. Although there is more research needed in some areas, the following applications have been documented.

Specificity

There is some evidence that measures of muscular strength are associated with balance and falls. However, a recent systematic review and meta-analysis has shown that balance and lower extremity muscle strength/power are independent of each other and task specific and should, therefore, be tested and trained independently from each other across the lifespan. Thus, if balance is needed, then, as with most physical fitness attributes, the training must be specific to balance.

Balance training programs may involve performing basic balance tasks such as alternating one-leg stands, standing on one leg with eyes closed, backward walking, and the use of a wobble ball (**Figure 20.20**). Balance training may also use a specialized training aid such as the wobble board. The wobble board consists of a round section fixed to the top of a hemispherical section allowing for multiplanar movement. The wobble board is common in rehabilitative settings and has traditionally been used to help injured athletes work on strength, flexibility, and balance following a lower limb injury.



Figure 20.20 Wobble Ball Training to Improve Balance.

Tai Chi is a form of training that employs self-initiated slow but continuous rhythmical movements and emphasizes multidirectional weight shifting, awareness of body alignment, and abdominal and lower extremity muscle function. Both cross-

sectional and controlled studies have shown that Tai Chi can lead to improved functional balance, with a reduction in falls in older persons (Li et al., 2004). Pilates may also be used to improve balance (Barker et al., 2015).

Overload

Results from two meta-analyses (Lesinski et al., 2015a, 2015b) indicate that an effective balance training program for younger (16–40 years) and older (≥ 65 years) individuals of both sexes is characterized by a training frequency of 3 per week for 11–15 minutes per session plus additional time for warm-up and cooldown for a total of 31–45 minutes per session or 91–150 minutes per week. Although data were insufficient to determine the effective number of exercises and sets for older individual, at least four separate exercises with a duration of 21–40 seconds in two sets for a total of 16–19 training sessions were found to be effective for younger individuals. Older adults benefited most from at least 36–40 training sessions. There is no methodological sound approach available for assessing intensity during balance training relative to an individual's balance ability.

Rest/Recovery/Adaptation

There is no specific research information available in this area, but there is also no reason to assume that rest and recovery are not important for adaptation to occur.

Progression

It is essential to sufficiently and persistently challenge postural control during the course of balance training. Increasingly difficult activities can be achieved by narrowing the base of support (both feet side by side, to step placement, to tandem heel-to-toe positioning, to one-legged stance) and by limiting the use of sensory information (eyes open to eyes closed; standing on solid ground to standing on unstable supports) (Lesinski et al., 2015a).

Individualization

As always, the exercises need to be selected to match the ability of each individual, and progression must be matched to the increasing balance exhibited by the individual.

Maintenance

Training-induced gains in balance are not stable in older individuals but appear to decline after 6 weeks of detraining. Therefore, it is recommended that participation in balance training be ongoing for older individuals to counteract age-related declines in balance performance ([Lesinski et al., 2015b](#)).

Retrogression/Plateau/Reversibility

There is no research information on either retrogression or plateau on balance training. Reversibility does occur with detraining. [Toulotte et al. \(2006\)](#) showed that simply not participating in a physical exercise program resulted in worse balance outcomes over time and that just 3 months of detraining following a structured exercise program in older individuals showed signs of reversibility of physical fitness and balance ([Toulotte et al., 2006](#)).

Warm-Up and Cooldown

There is a lack of definitive information as to whether balance training should be done before or after exercise and/or sports training sessions. Balance exercises are not typically included in general warm-up or cooldown activities beyond their link to certain flexibility activities. When balance training is conducted in isolation, both a warm-up and cooldown should be part of that training session ([Lesinski et al., 2015a, 2015b](#)).

Adaptation to Balance Training

Balance training following the training principles outlined above

is an effective means of improving static steady-state, dynamic steady-state, proactive and reactive balance, as well as performance in balance test batteries in older adults. Age does not appear to negatively impact balance adaptations when compared to young adults (Lesinski et al., 2015b). There is similar evidence of balance improvement for measures of proactive and steady-state balance in healthy young adults but insufficient evidence for reactive balance. Favorable effects have also been shown on motor performance and injury prevention (Lesinski et al., 2015a; Muehlbauer et al., 2015). Athletes of different specializations exhibit an enhanced ability to maintain balance in the specific conditions associated with their sports (Zemková, 2014).

Balance training may be particularly effective in reducing risk of injury in older adults. A meta-analysis investigating the effects of balance training on balance performance in healthy older adults concluded that balance training significantly improves proxies of static/dynamic steady-state, proactive, and reactive balance in this population (Lesinski et al., 2015b). Importantly, this may contribute to decreased falls, fractures, and hospitalizations of older adults. Thankfully, researchers are investigating ways to incorporate balance training at home for older adults that is convenient, affordable, and easy to implement by using video game systems. For example, researchers have investigated the use of the Kinect for the Xbox 360, which uses the movement of arms and limb throughout space to interact with the game, to implement balance specific training. A 2016 pilot study concluded that the use of this device at home can significantly improve measurements of balance at home for older adults (Bieryla, 2016).

Summary

1. The spinal cord performs the essential functions of connecting the peripheral nervous system with the brain and serving as a site of reflex integration.
2. Voluntary motor impulses are transmitted from the motor area of the brain to somatic efferent neurons, leading to

skeletal muscles via the pyramidal pathways.

3. Reflexes play an important role in maintaining an upright posture and responding to movement in a coordinated fashion. Reflexes are rapid, automatic responses to stimuli in which a specific stimulus results in a specific motor response.
4. The myotatic reflex is initiated in response to a sudden change in length of the muscle. When a muscle is quickly stretched, the annulospiral nerve endings in the NMS transmit an impulse to the spinal cord, which results in an immediate strong reflex contraction of the same muscle from which the signal originated.
5. The inverse myotatic reflex is initiated when tension increases abruptly and intensely and stimulates Golgi tendon organs; it results in inhibition of the tensed muscle group, causing relaxation.
6. Volitional control of movement can function at the level of the motor unit as well as at the whole muscle level.
7. Flexibility is joint specific, and the degree of flexibility is specific to the individual. Flexibility may be either static or dynamic, but dynamic cannot easily be measured. Dynamic stretching, static stretching, or proprioceptive neuromuscular facilitation techniques are recommended to enhance flexibility.
8. Newer research has demonstrated that stretching protocols may help improve cardiovascular function.
9. An acute bout of stretching:
 - a. Increases range of motion
 - b. Does not adversely impact performance if dynamic stretching is utilized and/or static stretching or proprioceptive neuromuscular facilitation stretching is limited to less than 45–60 seconds per muscle group incorporated into a comprehensive warm-up at least 10 minutes prior to training or competition
 - c. Does not appear to mitigate injury
 - d. Has no beneficial effect on delayed-onset muscle soreness
 - e. May negatively influence both strength and endurance performance if performed for long duration (> 120 seconds) directly prior to exercise.

10. Flexibility training adaptations:

- a. Include an improvement in range of motion
 - b. Have not conclusively been shown to protect against injury
 - c. May decrease eccentrically induced muscle damage leading to delayed-onset muscle soreness
11. Despite popular belief, scientific evidence suggests that resistance training is not detrimental to flexibility. In fact, studies have shown that heavy resistance training results in either an improvement or no change in flexibility.
12. Balance is an important fitness component of particular interest because of its relationship to falls in the elderly. Balance is both static and dynamic, and the type of balance is situational.
13. Training programs specifically aimed at improving balance in older adults have shown improvements in balance-related parameters and may reduce the risk of injuries.

Review Questions

1. Describe the anatomical relationship between nerves and muscles. What is the functional significance of this relationship?
2. Diagram the sequence of events that occur at the neuromuscular junction.
3. Diagram the components of a generalized reflex arc.
4. Diagram the components of the myotatic reflex. Pay careful attention to the afferent and efferent neurons involved.
5. Diagram the components of the inverse myotatic reflex.
6. Outline the sequence of events involved in volitional control of movement.
7. Provide a rationale for incorporating a flexibility training program into an overall fitness program.
8. Critique the appropriateness of the sit-and-reach test as a measure of lumbar flexibility and a predictor of low back

pain. What are the anatomical requirements of a healthy low back?

9. Differentiate between ballistic and dynamic stretching. Which technique is preferred and why?
10. Describe static stretching, and explain the involvement of reflexes in muscle elongation during this type of stretching.
11. Describe the proprioceptive neuromuscular facilitation technique, and explain the involvement of reflexes in muscle elongation during this type of stretching.
12. Describe the responses to an acute bout of static stretching.
13. Describe the importance of different static stretching durations (e.g., 30, 60, 120 seconds) on both endurance and resistance training.
14. If you decide to include static stretching as part of a warm-up routine, what are some measures you can take to decrease the risk of hindering performance?
15. Discuss the application of the individual training principles to the development of a flexibility program and the training adaptations that occur.
16. Discuss the importance of balance across the age span.
17. Discuss the application of the training principles for the development of balance and the training adaptations that occur.

For further review and study tools, visit Lippincott Connect.

Literature Search

In this chapter, we discussed neuromuscular aspects of movement. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search neuromuscular adaptations to training, this will yield a huge selection of articles.

- b.** Refine your search using key terms that may reflect your interest in this area. For example,
- ii.** Neuromuscular adaptations to training and Parkinson's disease.
 - ii.** Neuromuscular adaptations to training after stroke.
 - iii.** Neuromuscular adaptations and hypertrophy.
 - iv.** Neuromuscular adaptations to balance training.
 - v.** Continue your search for aspects of this topic that are of particular interest to you.

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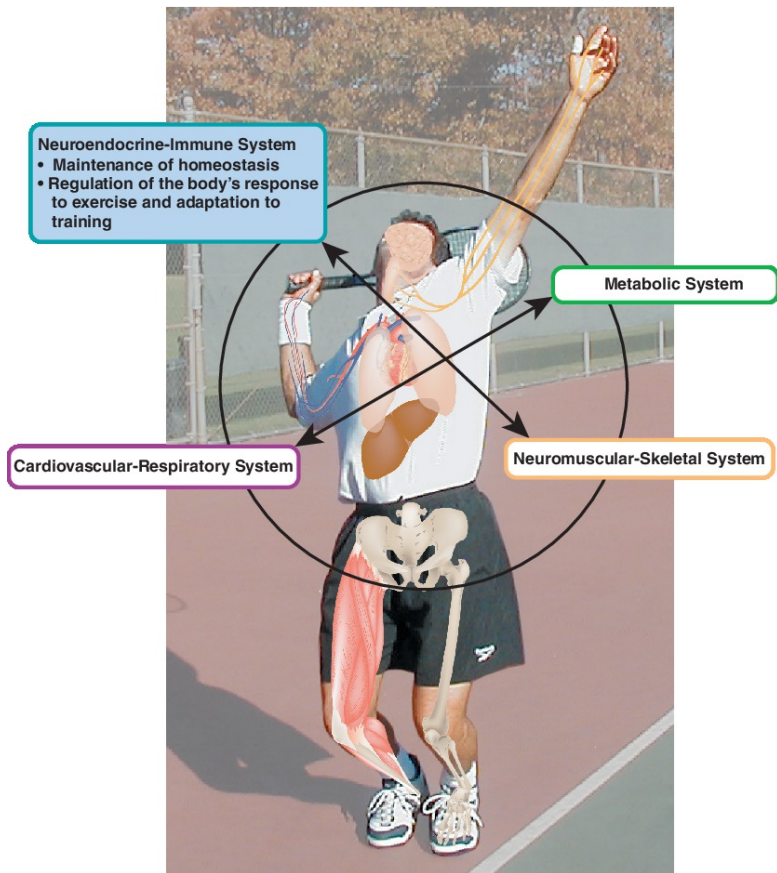
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Neuroendocrine- Immune System Unit



The neural, endocrine, and immune systems play a vital role in regulating and coordinating the body's response to exercise and the adaptations to exercise training. The autonomic nervous system and endocrine systems function to control cardiovascular and metabolic responses and adaptations. Recent evidence has made it increasingly clear that there is considerable communication between the chemical mediators of the endocrine system (hormones) and the cells and chemical mediators (cytokines) of the immune system. While these systems are essential for coordinating the positive adaptations to exercise training, there is also evidence that these same systems can reflect the pathological condition of overtraining.

21 Neuroendocrine Control of Exercise



CHAPTER OUTLINE

Introduction

Exercise as a Stressor That Activates the Neural and Hormonal Systems

The Nervous System

The Autonomic Nervous System

Neural Communication and Responses

Assessing Autonomic Nervous System Activity

Autonomic Nervous System Control during Exercise

The Endocrine System

The Basic Structure of the Endocrine System

Hormonal Communication and Responses

Role of the Endocrine System in Exercise

Hormonal Regulation of Metabolism

Hormonal Regulation of Cardiovascular Function

Hormonal Involvement in Muscle, Bone, and Adipose Tissue

Hormonal Responses to Exercise

Metabolic and Cardiovascular Hormones

Fluid Balance Hormones

Metabolic Hormones

Hormones Related to Muscle, Bone, and Adipose Tissue

Hormonal Adaptations to Training

Adaptations Related to Metabolic Function

Adaptations Related to Cardiovascular Function

Adaptations Related to Muscle, Bone, and Adipose Tissue

Hormonal Adaptations to Resistance Training

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Identify and briefly describe the overlapping roles of the

autonomic nervous system and endocrine system in maintaining homeostasis.

- Identify changes that may occur in a target cell as a result of the binding of a neurotransmitter or hormone to a cell receptor (receptor activation).
- Describe the functions of the autonomic nervous system that relate directly to regulating the exercise response.
- Identify the primary hormones involved in regulating the exercise response and describe the exercise response of these hormones.
- Identify adaptations in the autonomic nervous system as a result of exercise training.
- Identify the adaptations that occur in the hormonal system as a result of exercise training.

Introduction

You know from your own experience that when you exercise, certain changes take place in your body. You consciously initiate the most obvious change—contraction of the muscles—by activating the nervous system, which in turn signals the muscles to contract. You may also be aware of other changes, such as an increased breathing rate, increased heart rate, and increased sweating; these responses you do not consciously initiate. Still other changes—fuel mobilization, enzyme actions, and energy utilization—also occur during exercise without conscious initiation or even awareness of their occurrence. These changes are all part of an ongoing internal effort to maintain homeostatic balance. **Homeostasis** is the dynamic state of equilibrium in the internal functioning of the body. It is controlled and coordinated by the nervous and endocrine (hormonal) systems of the body. Furthermore, these two complementary and often overlapping systems regulate the body's response to any disruption in homeostasis, such as the disruption created by exercise. The nervous system is the faster-acting regulator of the body, whereas the endocrine system is the slower-acting regulator. The two systems interact and overlap in multiple ways to support exercise. Because these two systems function so closely together, they are

often referred to as the *neuroendocrine* (or *neurohormonal*) system. While [Chapter 20](#) examines the role of the somatic nervous system in initiating movement, this chapter addresses the role of the neuroendocrine system in regulating the body's responses to exercise and adaptations to training. Because the neuroendocrine system controls body systems, including those described in this book (cardiorespiratory, metabolic, and neuromusculoskeletal), the basic principles of neuroendocrine control are critical for understanding the exercise responses, training adaptations, and the integrated nature of all the systems studied in exercise physiology.

Homeostasis A dynamic state of equilibrium in the internal functioning of the body.

Both the nervous system and the hormonal system rely on chemical messengers to communicate with target cells. While neurons rely on electrical signals to conduct messages within a cell, they communicate with adjacent cells via chemical messengers called **neurotransmitters**. **Figure 21.1** depicts the cells of the nervous and hormonal systems and the chemical messengers they use. Neurons are secretory cells that release neurotransmitters at the site of the target cell. Endocrine glands, or hormone-producing tissues, release chemical messengers called **hormones** into the bloodstream or another body fluid. These hormones, in turn, have systemic or local effects. Thus, a hormone can be defined as a chemical substance that originates in glandular tissue (or cells) and is transported through body fluids to a target cell to influence physiological activity.

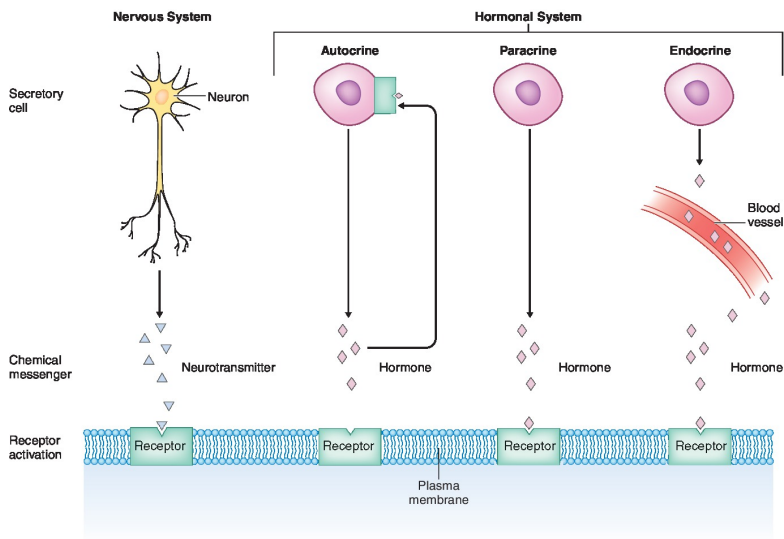


Figure 21.1 Chemical Messengers of the Nervous and Hormonal Systems.

Both the nervous system and the hormonal system rely on chemical messengers to communicate with target organs. Neurons release neurotransmitters in close proximity to the target organ. Endocrine glands release hormones into body fluids (often the blood) and may have an effect on target organs throughout the body.

Neurotransmitters Chemical messengers with which neurons communicate with target cells of either other neurons or effector organs.

Hormones Chemical substances that originate in glandular tissue (or cells) and are transported through body fluids to a target cell to influence physiological activity.

The effect of hormones on target cells can be further delineated as autocrine, paracrine, or endocrine. Autocrine

function refers to the action of a hormone on the cell that secreted the hormone. Paracrine function refers to the action of a hormone released into the body fluids that has an effect on a nearby cell, and endocrine function refers to a hormone released into the bloodstream that has an effect on target cells at a distant site. If a neuron releases a chemical substance into the bloodstream, this substance is called a *neurohormone*.

The effect of chemical messengers (neurotransmitters, hormones, and neurohormones) on target cells is mediated by the substance's binding to a receptor on (or in) the target cells. The chemical messenger binds to the receptor because of the complementary shape of the messenger and the receptor. Thus, chemical messengers bind only to very specific receptors. The messenger-receptor binding is known as *receptor activation*. It results in one or more changes within the target cell:

1. Change in permeability, electrical state, or transport properties of the cell
2. Change in enzyme activity of the cell (altering metabolism)
3. Change in secretory activity of the cell
4. Muscle contraction
5. Protein synthesis

Table 21.1 summarizes the nervous and endocrine systems within a comparative framework.

TABLE 21.1 Outline of Nervous and Endocrine Systems

| | Nervous System | Endocrine System |
|-------------------------------|--|--|
| Basic structure | Central and peripheral nervous system composed of neurons | Endocrine gland/tissue, which releases hormones |
| Chemical messenger | Neurotransmitter (NT) | Hormones |
| Mechanism of chemical release | Action potential in axon causes release of NT | Endocrine gland/tissue secretes hormone into blood/body fluid |
| Chemical/receptor binding | NT binds to receptor on target cell because of complementary shape and affinity | Hormone binds to receptor on target cell because of complementary shape and affinity |
| Mechanism of action | Change in membrane permeability Second messenger system Direct gene activation | Second messenger system Direct gene activation |
| Role in exercise | <ol style="list-style-type: none">1. Contraction of skeletal muscle2. Regulation of cardiovascular-respiratory systems3. Coordination with endocrine system to:<ul style="list-style-type: none">• Mobilize fuel for energy production• Transport fuel, oxygen, and waste• Maintain fluid and electrolyte balance• Maintain thermal balance | <ol style="list-style-type: none">1. Regulate metabolic system<ul style="list-style-type: none">• Mobilize fuel for energy production• Increase rate at which fuel is broken down to produce ATP• Maintain blood glucose levels2. Regulate cardiovascular system<ul style="list-style-type: none">• Transport fuel, oxygen, and waste• Maintain fluid and electrolyte balance• Maintain thermal balance |

Exercise as a Stressor That Activates the Neural and Hormonal Systems

As detailed in [Chapter 1](#) and discussed throughout this text, exercise causes a disruption in homeostasis and simultaneously creates the need for the body to increase oxygen and nutrient delivery to the working muscles to support the metabolic demands of activity. These needs are met by the joint action of the neural and hormonal systems. Understanding the neurohormonal response to exercise is necessary for understanding how the bodily systems respond to exercise or any stressor ([Hackney, 2006](#); [Selye, 1956](#)).

Both the neural and the hormonal regulatory systems are involved in the stress response system, as shown in the schematic outline in **Figure 21.2**. The primarily neural component is called the brainstem (locus ceruleus)-sympathetic nervous system (SNS) pathway and is shown on the left. The hypothalamus-pituitary-adrenal axis constitutes the primarily hormonal component and is shown on the right side of the figure. Notice, however, the overlap of the two systems. The *hypothalamus* is both a neural and an endocrine organ, and activation of the SNS stimulates many target organs by direct innervation and causes the release of epinephrine (E) and norepinephrine (NE) from the adrenal medulla. When the individual encounters a stressor, the hypothalamus coordinates the response. As both a neural structure and an endocrine gland, the hypothalamus orchestrates

the body's response by stimulating both the SNS and the endocrine glands.

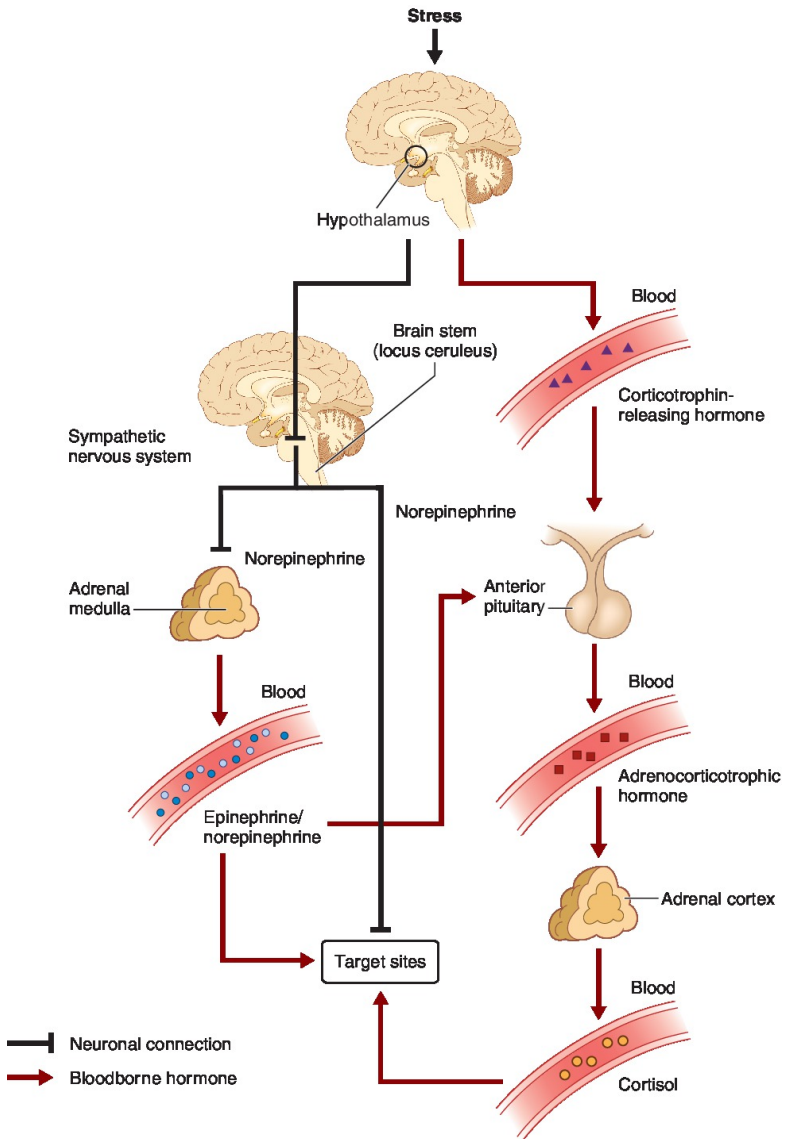


Figure 21.2 Neurohormonal Regulation of the Exercise Stress Response.

The nervous and hormonal systems have multiple, and often

overlapping, roles in regulating the exercise response. The hypothalamus serves as both a neural tissue (activating the brainstem) and a hormone-secreting gland.

The sympathetic nerve fibers originate in the brainstem and travel throughout the body to a variety of target sites. The sympathetic nerve fibers also go to and control the adrenal medulla. NE is released by sympathetic nerve fibers, and both NE and E are secreted by the adrenal medulla. The NE released from the sympathetic nerve endings and from the adrenal medulla is the same chemical. However, when NE is released by the nerves, it is considered to be a neurotransmitter, and when it is released by the adrenal medulla, it is considered to be a hormone. The NE and E secreted by the adrenal medulla circulate in the bloodstream to the target sites, where they mimic and reinforce the actions of the sympathetic nerve fibers that innervate these same target sites, for example, in elevating the heart rate. This response pattern is often referred to as sympathoadrenal activation. Both NE and E may also influence other endocrine glands, as indicated by the connecting line to the anterior pituitary gland in **Figure 21.2** (Chrousos and Gold, 1992).

The hypothalamus directly regulates endocrine glands both neurally and hormonally through a series of releasing factors, sometimes called releasing hormones. In the generic stress response, CRH is released by the hypothalamus and stimulates the anterior pituitary to secrete adrenocorticotrophic hormone (ACTH). ACTH in turn stimulates the adrenal cortex to release cortisol, which acts on specific target sites.

The following sections discuss the neural and hormonal responses to exercise separately in greater detail, but it is useful to remember the considerable overlap between these systems.

The Nervous System

The nervous system is a fast-acting control system that regulates an almost endless list of bodily functions. In general, the nervous system has three primary functions:

1. Monitoring the internal and external environment through sensory receptors
2. Integrating the information it receives
3. Initiating and coordinating a response by activating muscles (skeletal, smooth, and cardiac) and glands (including endocrine glands and sweat glands)

These functions are accomplished by the cells of the nervous system (neurons) communicating with each other and with *effector organs* (muscle and glands). Communication within a neuron occurs by electrical signals (action potentials). Communication between neurons or between neurons and an effector organ (e.g., skeletal muscle, cardiac muscle, glands) occurs by chemical signals (neurotransmitters).

The Autonomic Nervous System

As described in [Chapter 20](#), the nervous system can be structurally divided into the *central nervous system* (CNS) and the *peripheral nervous system* (PNS) (see **Figure 20.1**). Recall that functionally, the *somatic nervous system* innervates skeletal muscles and is responsible for controlling their contraction. In contrast, *the autonomic nervous system* (ANS) provides subconscious neural regulation of the internal environment of the body by innervating cardiac muscle, smooth muscle, and endocrine glands. It is the ANS that interacts so extensively with the endocrine system to control the body's response to exercise as well as adaptations to exercise training and will be discussed in this chapter.

The ANS has two branches, which work in opposition to each other through dual innervations. **Figure 21.3** is a schematic overview of major body organs influenced by the two divisions of the ANS; organs most directly involved in the response to exercise are highlighted by being shown in color. The sympathetic nerves exit the spinal cord in the thoracic and lumbar region, whereas parasympathetic nerves exit the spinal cord from the cranial nerves and the sacral area. The *sympathetic nervous system* (SNS) supports activities associated with the “fight-or-flight” stress response and is vital in regulating the body's integrated response

to exercise (including increased oxygen delivery to the working muscles, fuel utilization in support of increased metabolic demands, and heat dissipation). The *parasympathetic nervous system* (PSNS) supports activities associated with “rest and digest” and is vital in the process of recovering from exercise. Changes in the balance of SNS and PSNS activity are responsible for many of the adjustments made during exercise and adaptations resulting from exercise training.

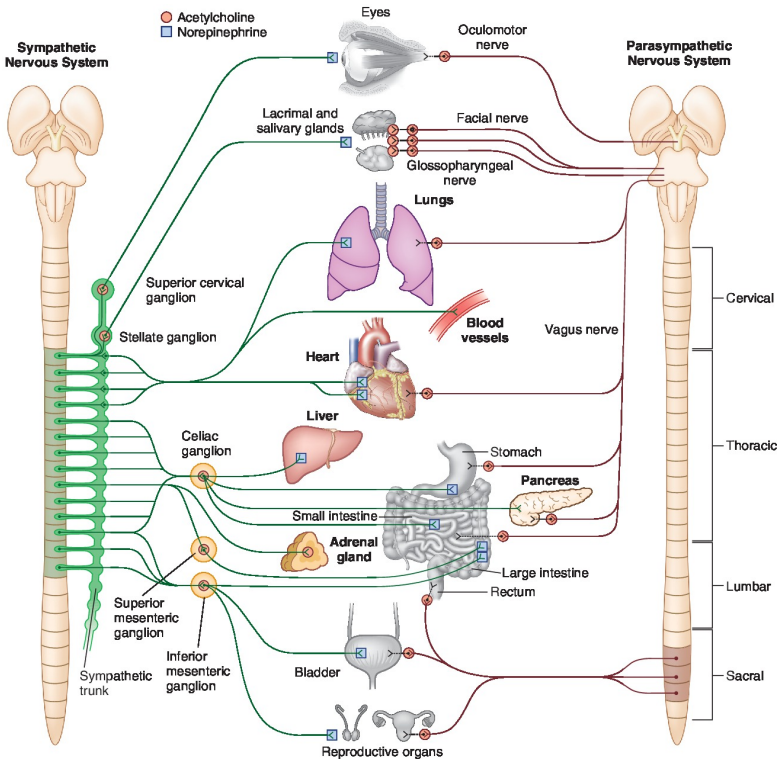


Figure 21.3 Components of the Autonomic Nervous System.

Sympathetic fibers exit the spinal cord at the thoracic and lumbar regions, whereas parasympathetic fibers exit from cranial nerves and the sacral region of the spinal cord. Parasympathetic preganglionic neurons are longer than sympathetic preganglionic neurons and synapse with postganglionic fibers near or within target organs.

Neural Communication and Responses

Neurons are the functional unit of the nervous system. Excitation of a neuron leads to the generation of an electrical signal—termed an action potential (see [Chapter 20](#) for full discussion). Once generated in the axon, the action potential moves along the entire length of the axon, causing a neurotransmitter to be released from the terminal end of the axon. The terminal end of the axon communicates with other neurons, muscle cells, or glands across junctions known as synapses. A synapse between a neuron and a muscle cell is known as a *neuromuscular junction*.

The dominant form of **synapse** is a chemical synapse, which involves the release of a neurotransmitter from the neuron. (The sequence of events involved in the release of a neurotransmitter at the neuromuscular junction is detailed in [Chapter 20](#).) Acetylcholine (ACh) is the neurotransmitter released from somatic motor neurons, parasympathetic and sympathetic preganglionic fibers, and parasympathetic postganglionic fibers (**Figure 21.4**). ACh is always excitatory to skeletal muscle but may be inhibitory or excitatory to target cells of the ANS (smooth muscle, cardiac muscle, glands), depending on the target cell receptors to which it binds. NE is the neurotransmitter released by most sympathetic postganglionic neurons; neurons innervating sweat glands are an important exception. NE is also inhibitory or excitatory depending on the receptors to which it binds. Neurons that release ACh are called *cholinergic fibers*; neurons that release NE are called *adrenergic fibers*.

Synapse The gap, or junction, between terminal ends of the axon and other neurons, muscle cells, or glands.

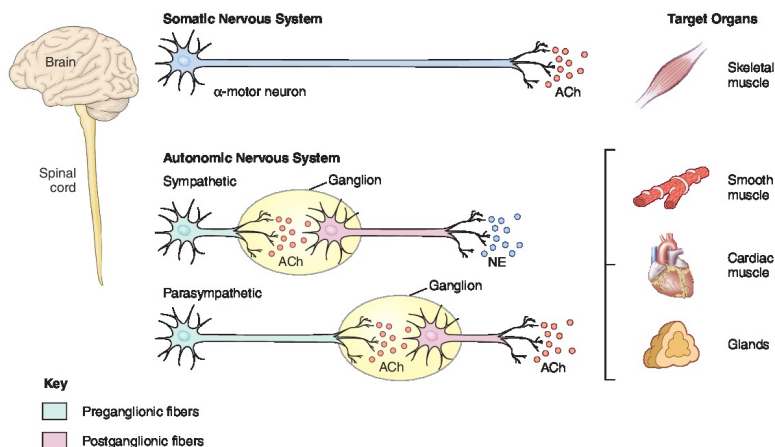


Figure 21.4 Neurotransmitters of the Somatic and Autonomic Divisions of the Nervous System.

Neurons of the somatic nervous system (α -motor neurons) release acetylcholine (ACh), which causes skeletal muscle to contract. Neurons of the parasympathetic division of the autonomic nervous system secrete ACh. Preganglion fibers of the sympathetic nervous system also secrete ACh, but postganglionic fibers secrete norepinephrine (NE). Autonomic fibers innervate smooth muscle, cardiac muscle, and glands.

The response of the target organ depends not only on the neurotransmitter but also on the receptor to which it binds. Receptors that bind ACh are called cholinergic receptors, and receptors that bind NE are called adrenergic receptors. The two types of *cholinergic receptors* are nicotinic and muscarinic receptors, and the two types of *adrenergic receptors* are termed alpha (α) and beta (β). These latter are further subdivided into α_1 , α_2 , β_1 , β_2 , and β_3 receptors (Hall and Hall, 2021). Because target organs may have more than one type of receptor, they may have different responses. Table 21.2 outlines the effects of the SNS and PSNS on selected effector organs. This table shows the general principle that activation of the SNS supports activities associated with the “fight-or-flight” stress response, while the PSNS is associated with the “rest and digest” mode.

TABLE 21.2 Effect of Autonomic Nervous Stimulation on Various Organs

| | | Effect of Sympathetic Stimulation | Effect of Parasympathetic Stimulation |
|------------------------------------|---------------------------|--|---------------------------------------|
| Cardiovascular | Heart | ↑ Rate of contraction | ↓ Rate of contraction |
| | Arterioles | ↑ Force of contraction | Slight ↑ force of contraction |
| | Coronary | Dilation (β_2), constriction (α) | Dilation |
| | Skeletal | Constriction (α), dilation (β_2 and cholinergic) | None |
| | Skin and internal organs | Constriction (α) | None |
| Metabolic | GI motility | Decrease (β_2) | Increase |
| | Adipose tissue | Lipolysis | None |
| | Liver | Glycogenolysis | Glycogen synthesis |
| | | Gluconeogenesis | |
| Fluid balance and thermoregulation | Sweat glands | ↑ Sweating (cholinergic) | None |
| | Kidney | ↑ Renin secretion (α , β_1) | None |
| | Posterior pituitary | ↑ ADH secretion | None |
| | Adrenal medulla secretion | Increase | None |
| | Coagulation | Increase | None |

Sources: Seifter et al. (2005); and Hall and Hall (2021).

Assessing Autonomic Nervous System Activity

Changes in parasympathetic and sympathetic activity have profound effects. However, directly measuring autonomic responses is difficult and must be done in a laboratory. The most direct measure, muscle sympathetic nerve activity (MSNA), measures electrical activity, typically in the peroneal nerve (located in the lower leg); this is an invasive method that provides an index of sympathetic excitement (Vallbo et al., 1979). However, ANS function can be easily assessed by measuring heart rate variability (HRV) and heart rate recovery (HRRec) from exercise.

FOCUS ON RESEARCH | *Clinically Relevant*

Effect of Training Status on ANS Recovery

Exercise results in activation of the SNS and a shift in

autonomic balance. The rate of recovery from this shift in autonomic balance depends on the intensity and duration of the activity and the training status of the individual. The quantification of HRV is a noninvasive tool to investigate changes in autonomic balance. HRV analysis can provide several useful variables, including mean HR, mean HR interval, and the root mean square of sequential deviations (RMSSD). RMSSD is often expressed as a percentage of preexercise values. A return to 100% indicates that preexercise autonomic balance has been restored.

As part of a larger study to investigate autonomic recovery from different exercise training regimens, Sieler et al. compared autonomic recovery from a high-intensity training session in highly trained athletes with recreationally trained athletes. The highly trained group ($n = 9$) had a mean age of 23 years, HRmax of $189 \text{ b}\cdot\text{min}^{-1}$, and a

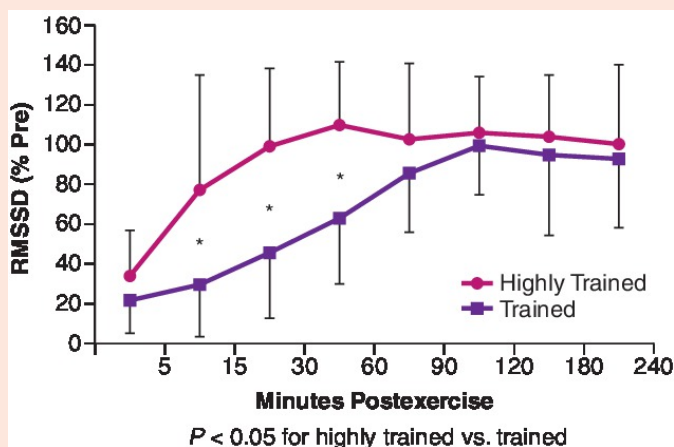
$\dot{V}\text{O}_2\text{max}$ of $72 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$; they trained an average of $14 \text{ hr}\cdot\text{wk}^{-1}$. The recreationally trained group ($n = 8$) had a mean age of 27 years, HRmax of $189 \text{ b}\cdot\text{min}^{-1}$, and a

$\dot{V}\text{O}_2\text{max}$ of $60 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$; they trained an average of $7 \text{ hr}\cdot\text{wk}^{-1}$. Both groups performed high-intensity interval exercise on a treadmill. Participants first performed a 20-minute warm-up and then completed a 30-minute interval session consisting of 6×3 minute intervals at a velocity

eliciting 95–100% of $\dot{V}\text{O}_2\text{max}$ with 2 minutes of active recovery periods, followed by a 10-minute cooldown. The RPE associated with the exercise session was 18 in both groups. The figure presents the RMSSD for the group of recreationally and highly trained athletes for the 4-hour postexercise period. The recovery of parasympathetic control after the intense interval exercise session was significantly slower (60–90 minutes) in the recreationally trained versus the highly trained subjects.

Highly trained subjects had a more accelerated recovery from intense interval work than recreationally trained athletes. This adaptation may be an important factor in the ability of highly trained athletes to tolerate typical twice-daily training regimens common among elite endurance

athletes.



Source: Reprinted with permission from Seiler, S., O. Haugen, & E. Kuffel: Autonomic recovery after exercise in trained athletes: Intensity and duration effects. *Medicine & Science in Sports & Exercise*. 39(8):1366–1373 (2007). Copyright ©2007 The American College of Sports Medicine.

Heart rate variability is the beat-to-beat variation in the time of the R-to-R intervals, reflecting fluctuation in time intervals between successive heart beats, called interbeat intervals (IBI) (Shaffer and Ginsberg, 2017). HRV provides important information about ANS control of the cardiovascular system at rest, during exercise, and during recovery from exercise. A healthy heart rate is not fixed but rather varies moment to moment in response to physiological stimuli. For instance, in a healthy individual with a resting heart rate of 60 b·min⁻¹, the interval between beats is not exactly 1 second but varies in a range of different times that averages 1 second. In fact, higher variability reflects the ability of the ANS to change quickly in response to changing internal stimuli. An increase in SNS activity occurs when there is a need to increase blood flow or blood pressure, and an increase in PSNS activity occurs when there is lower demand for blood flow or blood pressure.

Heart Rate Variability The beat-to-beat variation in the time of the R-to-R intervals.

There are several HRV measurements that can be reported from resting or exercise beat-to-beat variability. HRV can be described over 24-hour periods, short-term periods (~5 minutes), and ultrashort periods (<5 minutes). When comparing HRV measures, it is important to have standard time periods for obtaining the measures. HRV measurements are categorized as time domain measures, which report various time intervals between beats and frequency domain measures that rely on spectral methods for the analysis of several variables ([Draghici and Taylor, 2016](#); [Task Force of European Society of Cardiology and North American Society for Pacing and Electrophysiology, 1996](#)).

HRV reflects the balance of the ANS and is a strong predictor of cardiovascular and overall health. Lower variability reflects poor autonomic tone and is associated with a poor cardiovascular risk profile, elevated risk of coronary heart disease, increased risk of metabolic disease, and increased risk of all-cause mortality ([Singh et al., 2018](#); [Thayer et al., 2010](#); [Wulsin et al., 2015](#)). A meta-analysis found that lower HRV is associated with a 32–45% increase in risk of a first cardiovascular event in patients without known cardiovascular disease ([Hillebrand et al., 2013](#)). HRV is also considered a convenient, noninvasive assessment tool for monitoring recovery from exercise and individual adaptation to training. With the wide spread availability of wearable fitness devices with sophisticated analytic ability (such as Polar chest straps and WHOOP wrist bands [Figure 4.7]) several authorities have suggested that HRV is one of the most promising methods to monitor cardiac ANS status. Nonfunctional overtraining and/or negative adaptation to training are associated with reduced vagal-related indices of HRV, and increases in fitness and exercise performance are associated with increases in vagal-related indices ([Plews et al., 2013](#)). Increasingly, athletes are using commercially available units that calculate HRV to monitor recovery and to guide training programs.

Heart rate recovery (HRRec) following a graded exercise test

is an easily obtained measure that can be used to estimate autonomic response to a challenge. HRRec is the difference between maximum heart rate and the heart rate after a specified time (usually 1 or 2 minutes) of recovery. The primary determinant of HRRec is thought to be parasympathetic reactivation; thus, a faster HRRec indicates a greater parasympathetic response ([Carnethon and Craft, 2008](#)).

A delayed decrease in heart rate during the first minute of recovery from a graded exercise test (slower HRRec) is a powerful predictor of overall mortality and cardiovascular death, independent of workload, presence or absence of ischemia, and changes in heart rate during exercise. This is true in persons with cardiovascular disease and those who have no known cardiovascular disease ([Nishime et al., 2000](#); [Sydo et al., 2018](#)). Research suggests that the predictive power of HRRec is independent of sex, age, obesity, hypertension, and diabetes mellitus. However, HRRec is less predictive in patients taking β -blockers, current smokers, and individuals with normal cardiorespiratory fitness ([Sydo et al., 2018](#)). Research involving over 40,000 individuals found that the HRRec during the first 10 seconds of recovery (based on ECG tracings) is even more predictive of overall and cardiovascular mortality than the HRRec at 1 minute ([van de Vegte et al., 2018](#)).

Autonomic Nervous System Control during Exercise

The role of the ANS in regulating the exercise response is very diverse. As previously stated, the exercise response is mediated primarily through the sympathetic branch of the ANS. In addition to direct nerve stimulation via innervation of multiple organs (see [Figure 21.2](#)), sympathetic nerve stimulation leads to the release of E and NE from the adrenal medulla. These circulating hormones serve to augment the response of the sympathetic nervous fibers that directly innervate various organs. As summarized in [Figure 21.5](#), the primary functions of the sympathetic branch of the ANS during exercise are to:

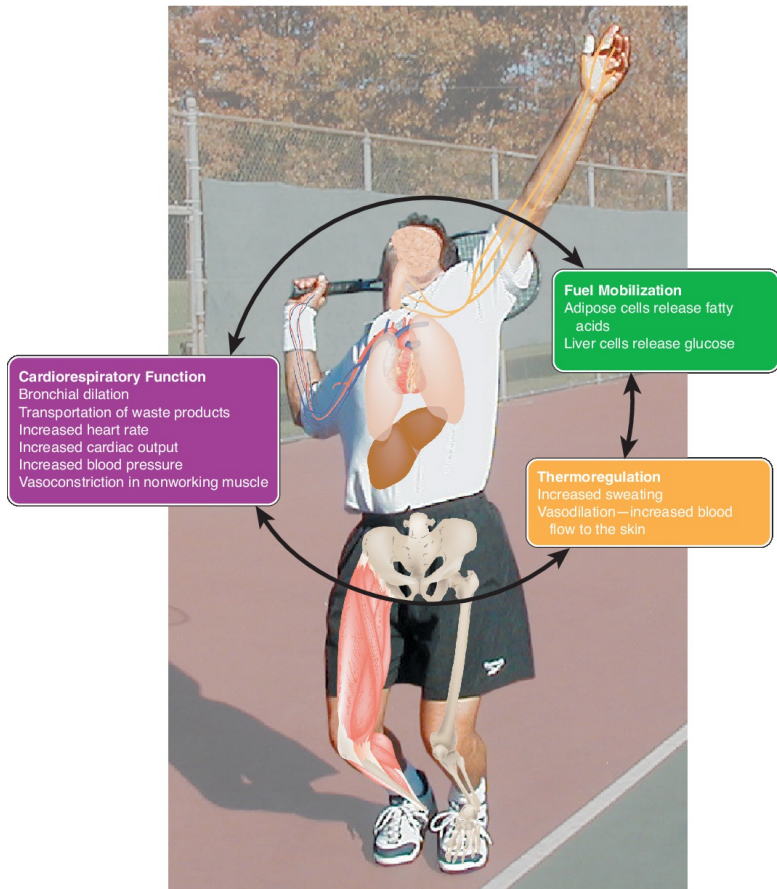


Figure 21.5 Primary Results of Sympathetic Nervous System (SNS) Stimulation during Exercise.

Activation of the sympathetic nervous system during exercise is responsible for many of the acute exercise responses.

1. Enhance cardiorespiratory function:
 - a. Increase cardiac output
 - b. Regulate blood flow and maintain blood pressure and blood volume
 - c. Ensure that the body can stop any unnecessary bleeding

2. Maintain thermal balance
3. Increase fuel mobilization for the production of energy

Autonomic nerve fibers innervate the respiratory system and the heart. Stimulation of the SNS causes a decrease in airway resistance (bronchial dilation), facilitating movement of air into and out of the lungs. Stimulation of the SNS also increases heart rate and force of contraction, causing an increase in cardiac output and an increase in blood pressure. The increased cardiac output is important for transporting fuel and oxygen necessary to support muscular contraction and for transporting waste products that build up as a result of contraction and must be eliminated from the body.

Related to enhancing cardiovascular function, the SNS also plays an integral role in redirecting blood flow during exercise and maintaining blood pressure. These functions are achieved by controlling the diameter of blood vessels. Sympathetic nerve stimulation causes blood vessels (arterioles) in visceral organs, skin, and nonworking muscles to vasoconstrict (decrease in diameter). This allows more blood to be redirected to the contracting muscles to support their metabolic needs. Sympathetic nerve stimulation also plays an important role in maintaining fluid balance during exercise by causing the release of renin and antidiuretic hormone—two hormones directly involved in fluid balance (see [Chapter 11](#)).

Additionally, in response to SNS activation, the blood has an increased tendency to clot. This appears to be a protective mechanism associated with the increased risk of injury in situations in which the body is stressed. Furthermore, the increased clotting potential during SNS activation is balanced by an increased ability to breakdown clots (fibrinolysis) in healthy individuals. However, in individuals with cardiovascular disease, especially endothelial dysfunction, this increased coagulatory potential can be dangerous (see [Chapter 15](#)).

The ANS also helps maintain thermal balance by controlling blood flow to the skin and by regulating sweat glands. During exercise, particularly exercise performed in the heat, vasodilation of skin blood vessels facilitates heat loss by increasing blood flow in cutaneous vascular beds near the surface of the body where

heat can be more easily dissipated. Sympathetic nerve stimulation also causes sweat gland secretion. Interestingly, the sympathetic nerve fibers that innervate sweat glands release ACh (unlike most sympathetic nerve fibers that release NE). The evaporation of sweat is the primary mechanism for heat loss during most exercise (see [Chapter 14](#)).

Finally, sympathetic nerve stimulation also helps to support the increased metabolic needs of exercise. The metabolic effects of the SNS are broad and include an increase in lipolysis in adipose tissue and an increase in glycogenolysis and gluconeogenesis in the liver. Thus, both fatty acids and glucose are mobilized to support the working muscles.

In addition to the activation of the SNS described above, the PSNS is simultaneously inhibited in the immediate preexercise anticipation stage and during exercise. The inhibition of the PSNS is often referred to as parasympathetic withdrawal. It is the withdrawal of parasympathetic nervous input that is responsible for the initial rise in heart rate seen with exercise and why immediate preexercise heart rates (such as might be taken before a treadmill test) are not accurate resting values.

The Endocrine System

The endocrine system, along with the nervous system, regulates the body's response to exercise. Although many hormones have changed blood concentrations during exercise, this textbook concentrates only on those hormones that play a primary role in regulating the exercise response or training adaptations. The primary role of the hormonal system during exercise is to help regulate the metabolic and cardiovascular systems ([Bunt, 1986](#)). Secondly, hormones are involved in muscle, bone, and adipose tissue functions.

The Basic Structure of the Endocrine System

The endocrine system is composed of a series of ductless glands,

other tissues, and the hormones they secrete. Hormones are chemical substances that originate in glandular tissue (or cells) and are transported through body fluids to a target cell to influence physiological activity. The major endocrine glands involved in exercise or training (**Figure 21.6**) include the hypothalamus, pituitary, thyroid, parathyroid, adrenal, pancreas, and gonads (ovaries and testes). Other tissues that also function as endocrine glands and secrete hormones include the heart, kidneys, liver, adipose tissue, muscle tissue, and the gastrointestinal tract, as well as endothelial and immunological cells. Hormones released from ductless glands are released directly into the bloodstream and travel throughout the body. Hormones released from tissue cells are excreted into the surrounding extracellular fluid, from which they may diffuse into nearby cells to exert an autocrine or a paracrine function (see **Figure 21.1**). Although a complete inventory and description of all hormones is beyond the scope of this book, **Table 21.3** lists those hormones directly involved in regulating exercise responses, training adaptations, or other functions discussed in this text.

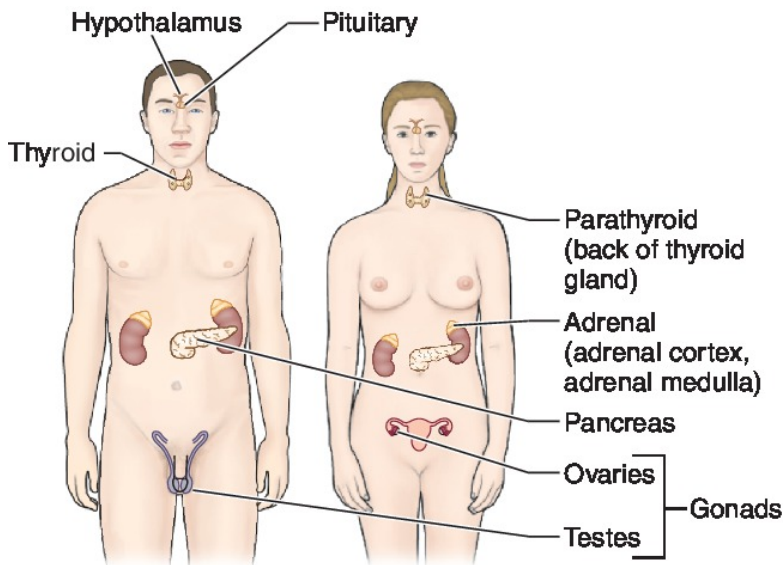


Figure 21.6 Major Endocrine Glands.

TABLE 21.3 Hormones Involved in Regulating Exercise

| Site Produced (Endocrine Gland/Tissue) | Hormone(s) | Selected Functions Relative to Exercise Physiology |
|--|--|--|
| Adipose tissue | Leptin | Food intake, metabolic rate |
| Adrenal gland | | |
| • Adrenal cortex | Cortisol, aldosterone | Metabolism, stress response, immune function, anti-inflammatory, catabolic to muscle tissue |
| • Adrenal medulla | Epinephrine, norepinephrine | Na ⁺ , K ⁺ , and acid secretion by kidneys, fluid balance |
| Gonads | | |
| • Ovaries (female) | Estrogen | Fat deposition, bone remodeling |
| • Testes (male) | Progesterone | Catabolic to muscular tissue, bone remodeling |
| | Testosterone | Bone and muscle growth and development |
| Hypothalamus | Hypothalamic trophic hormones (general) | Controls secretions of hormones of anterior pituitary |
| | • Corticotrophin-releasing hormone (CRH) | (in general): see anterior pituitary (Ant.Pit.) |
| | • Thyrotrophin-releasing hormone (TRH) | Stimulates anterior pituitary to secrete adrenocorticotrophic hormone (ACTH) |
| | • Growth hormone–releasing hormone (GHRH) | Stimulates Ant.Pit. to secrete thyroid-stimulating hormone (TSH) |
| | • Growth hormone–inhibiting hormone (GHIH) | Stimulates Ant.Pit. to secrete growth hormone (GH) |
| | • Gonadotropin-releasing hormone (GnRH) | Inhibits secretion of GH |
| | Production of antidiuretic hormone (ADH), which is released by posterior pituitary gland | Stimulates Ant.Pit. to secrete luteinizing hormone (LH) and follicle-stimulating hormone (FSH) |
| Kidneys | Erythropoietin | Erythrocyte (RBC) production |
| Leukocytes (WBC) and endothelial cells | Cytokines | Immune functions |
| Liver | Somatomedins (insulin-like growth factors [IGF]) | Anabolic to muscle tissue |
| Pancreas | Insulin, glucagon | Metabolism, regulates blood glucose levels |
| Parathyroid | Parathyroid hormone (PTH) | Plasma Ca ²⁺ , PO ₂ levels |
| Pituitary | | |
| • Anterior | Growth hormone | Bone and muscle growth, metabolism, stimulates IGF release |
| | Thyroid-stimulating hormone (TSH) | Secretion of hormones from thyroid gland |
| | Adrenocorticotrophic hormone (ACTH) | Secretion of hormones from adrenal cortex |
| | Follicle-stimulating hormone (FSH) and Luteinizing hormone (LH) | Sex hormone secretion |
| • Posterior | Antidiuretic hormone (ADH, also called vasopressin) | Water excretion by kidneys, fluid balance, cardiovascular function |
| Thyroid | Thyroxine (T ₄) triiodothyronine (T ₃) | Metabolic rate |
| | Calcitonin | Plasma Ca ²⁺ levels |

Activation of the Hormonal System

The hormonal system can be stimulated to release hormones (chemical mediators) through multiple pathways. Once secreted, hormone levels in the blood are dependent on multiple factors, and their biological activity depends on still other factors. The following section provides a brief overview of the activation of the hormonal system.

SECRETION OF HORMONES Endocrine glands and tissues can be activated to secrete hormones in three ways: neural, hormonal, or humoral. Regardless of the type, the activation of an endocrine gland or hormone-secreting tissue always depends on a chemical

signal. Furthermore, the synthesis and release of most hormones are regulated by *negative feedback* mechanisms; that is, the output (the hormone or a variable controlled by the hormone) shuts off the original stimulus or reduces its intensity. Negative feedback mechanisms cause the variable to change in a direction opposite to the original change, returning the variable to its “set point” and thus helping to maintain homeostasis.

Neural activation occurs directly when a neuron releases a neurotransmitter that signals the endocrine tissue to release a hormone. For example, sympathetic neurons release the neurotransmitter NE, which stimulates the adrenal medulla to release the hormones E and NE.

Hormonal activation literally means that one hormone (sometimes called hormone-releasing factor) stimulates another gland to release a hormone in a *feed-forward* control system. Hormones that stimulate the release of another hormone are called trophic hormones. The hypothalamus secretes numerous *trophic* hormones, including CRH. CRH in turn stimulates the anterior pituitary to release ACTH. ACTH then stimulates the adrenal cortex to release cortisol (the long-term stress hormone). When hormones from one gland cause the target gland to secrete a hormone, which affects yet another gland, this is called an axis. The example given above describes the hypothalamus-pituitary-adrenal axis diagrammed in **Figure 21.2**.

The term *humoral* refers to blood or other body fluids. *Humoral activation* thus refers to stimulation of an endocrine gland by blood levels of nutrients, electrolytes, water, ions, or other factors. For example, the pancreas responds to blood levels of glucose (a nutrient) by releasing the hormone insulin. Likewise, the thyroid gland and parathyroid glands are stimulated to release calcitonin and parathyroid hormone, respectively, in response to blood levels of calcium (an electrolyte).

BLOOD HORMONE LEVELS The plasma concentration of a hormone depends on several factors including:

1. The rate at which the hormone is secreted
2. The rate at which it is broken down and removed from the blood

3. For some hormones, the effective, or biologically active amount; that is, how much of the hormone is bound to a protein versus how much is circulating in the free or unbound state

Hormones can be secreted in pulsatile (or rhythmic), circadian (day/night or 24-hour periodization), entropic (random moment-to-moment variation), or cyclical (often monthly) patterns as well as on demand. Some hormones act instantaneously; others require minutes to hours to days to have an impact. Hormones can be broken down and cleared from the body by the kidney, liver, or target cells. In addition, many hormones (primarily the steroids) circulate in blood bound to protein. However, to interact with a receptor, the hormone must be unbound, or “free.” In these cases, it is the amount of free hormone, not the total amount circulating that determines the biological effectiveness of the hormone.

In addition to the factors listed above, hormonal blood levels are affected by disease, temperature, altitude, nutritional status, age, sex, exercise, hydration level, and training status.

EXTENT OF CELLULAR RESPONSE As stated earlier, hormones affect a target cell by binding to a receptor on or in the target cell. This binding requires complementary shapes between the hormone and the receptor. The hormone-receptor binding is known as *receptor activation*. The extent of cellular response to receptor activation depends on three factors (Marieb and Hoehn, 2018):

1. The blood levels of the hormone
2. The relative number of receptors
3. The strength (affinity) of the bond between the hormone and receptor

Factors influencing the blood level of the hormone are described in the preceding section. The number of receptors on a cell for any given hormone can vary over time and in response to acute exercise and chronic exercise training. Hormone-receptor binding often destroys the receptor; as a result, replacement

receptors are needed. High-affinity receptors produce a more pronounced hormonal effect than low-affinity receptors. High blood levels of a hormone may cause either an increase in the number of receptors (*up-regulation*) or a decrease in the number of receptors (*down-regulation*). Up-regulation increases the cell's ability to bring about the hormone-specific cellular response; down-regulation prevents the target cells from overreacting to persistently high hormonal levels. Low blood levels of a hormone may also result in up-regulation. The more receptors, the lower the hormone concentration required to cause any given physiological response.

Mechanism of Hormonal Action

Hormones are divided into three general classifications: (1) proteins and polypeptides, (2) steroid-based hormones, and (3) derivatives of the amino acid tyrosine. Protein and polypeptide hormones include those released from the anterior and posterior pituitary gland, the pancreas, the parathyroid gland, and dozens of others. Steroid-based hormones are derived from cholesterol and include only those hormones secreted from the adrenal cortex (aldosterone and cortisol) and gonads (estrogen and testosterone). Derivatives of tyrosine include the hormones released from the thyroid (T3 and T4) and the adrenal medulla (E and NE).

For a hormone to bring about an action within a cell, it must first bind to a cellular receptor. Receptors may be on the cell membrane (often a transmembrane protein), in the cytoplasm, or in the nucleus of the cell. Once a hormone binds to a receptor on or in the target cell, a response is initiated. The sequence of steps from receptor activation to cellular response is known as the *signal transduction pathway* or *mechanism of action*. A hormone may exert its action through numerous transduction pathways; a full description of these pathways is beyond the scope of this text. However, as shown in **Figure 21.7**, signal transduction pathways may be broadly categorized into second messenger systems and gene activation pathways (Hall and Hall 2021, 2016; Marieb and Hoehn, 2018).

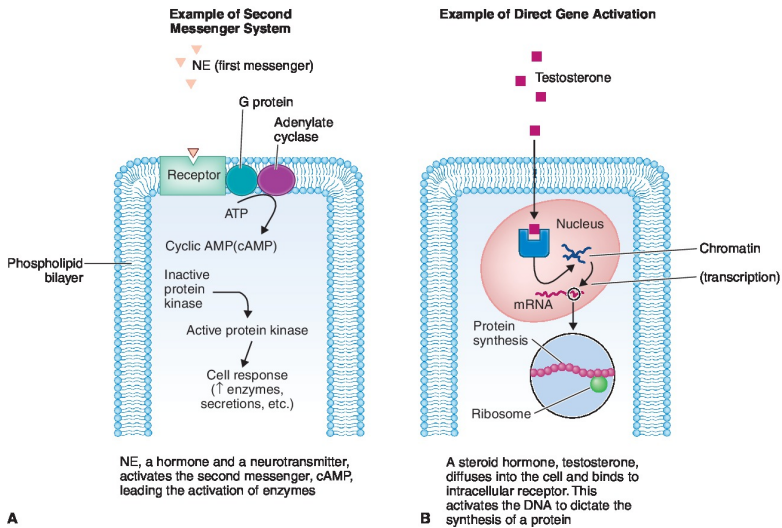


Figure 21.7 Mechanisms of Hormonal Action.

Hormones may exert their effect on target organs (A) using a second messenger system (one example is shown) or (B) through direct gene activation.

Because they are lipid insoluble and cannot diffuse into a cell, many hormones exert their influence primarily through a *second messenger system*. This mechanism begins when a hormone, known as the *first messenger* because it is delivering a chemical message from another tissue, binds to its specific receptor on the cell membrane. The binding of the first messenger to the receptor activates another membrane protein (e.g., G protein), which in turn activates a *second messenger* within the cell (e.g., adenylate cyclase). The second messenger initiates a cascade of chemical reactions (e.g., changing a protein kinase enzyme in the cell from an inactive to an active form) as shown in **Figure 21.7A**.

Steroid hormones and a few protein hormones are lipid soluble and diffuse easily into target cells. Once inside the cell, they operate by *direct gene activation*. Lipid-soluble hormones enter the cell and bind to receptors either in the nucleus or in the cell cytoplasm (forming a complex that must then migrate into the nucleus). The activated hormone-receptor complex signals the chromatin portion of the DNA, a gene, to be transcribed to

messenger RNA (mRNA). The mRNA carries the code from the nucleus to the cytoplasm, where a specific protein is synthesized (see **Figure 21.7B**). The protein produced may be a muscle filament or an enzyme that regulates cell metabolism.

Hormonal Communication and Responses

The hormonal system regulates and integrates a dizzying array of bodily functions. All hormones exert their influence by binding to a specific receptor on or in the cell. Several additional principles help explain how these hormone-receptor complexes translate into physiological responses that result from exercise. **Figure 21.8** summarizes these essential principles:

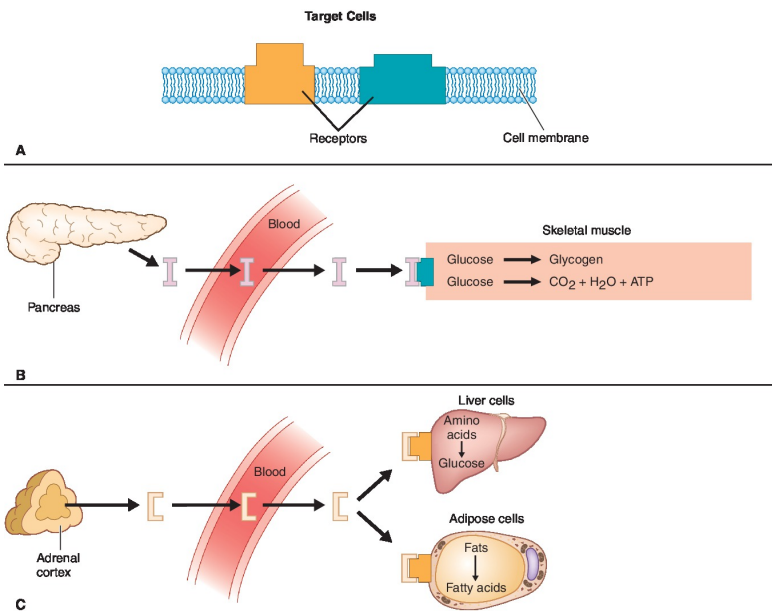


Figure 21.8 Principles of Hormonal Response.

A. Hormones bind to receptors because of complimentary structures. **B.** A hormone may have multiple effects within a single target organ. **C.** A hormone may affect multiple target organs.

- A target cell may (and usually does) have many different

receptor types on its surface (**Figure 21.8A**). Furthermore, as noted earlier, the number of any given type of receptor may change over time based on cell needs.

- A hormone may have several different functions within a cell (**Figure 21.8B**). For example, when insulin binds to receptors on skeletal muscle cells, it may cause several things to happen, depending on metabolic conditions within the cell. These include an increase in the uptake of glucose into the cell, the formation of glycogen from glucose (by a process known as glycogenesis), or an increase in the rate at which glucose is broken down to produce ATP within the cell (glycolysis).
- A hormone may affect multiple target cells (**Figure 21.8C**). For example, when cortisol binds to receptors on the membrane of liver cells, it causes glycogen to be broken down into glucose through the process of glycogenolysis. Glucose levels in the blood thus increase, and glucose can be utilized as a fuel for active muscle cells and nervous tissue. However, when cortisol binds to receptors on adipose cells, it stimulates the breakdown of fats into free fatty acids and glycerol through the process of lipolysis. Thus, free fatty acid levels in the blood increase and can be utilized as a fuel for active muscle cells.

Interaction of Hormones

Many cellular responses require the joint action of many hormones. In a *synergistic response*, the combined effect of the hormones may be greater than the sum of their individual effects. In a *complementary response*, both or all hormones are needed to accomplish the task. *Permissive* hormones facilitate or potentiate the actions of another hormone, making their response more effective. In some situations, the actions of one hormone oppose another. These hormones are said to be antagonistic.

Role of the Endocrine System in Exercise

Exercise presents a physical stress that challenges homeostasis. In response, the neuroendocrine system, particularly the ANS and

the hypothalamic-pituitary-adrenal axis, reacts to help maintain homeostasis ([Mastorakos and Pavlatou, 2005](#)). This section describes the hormonal response to exercise. The primary role of the endocrine system during an acute bout of exercise is to regulate the metabolic and cardiovascular systems, to conserve fluid, and to regulate stress-reactivity reactions ([Bunt, 1986](#); Hackney and Lane, 2015). Exercise affects almost every endocrine gland, including reproductive glands, and endocrine responses to exercise play an important role in the adaptations to exercise training, including increased muscle size and regulation of body composition. The following sections primarily describe how the endocrine system helps regulate the metabolic and cardiovascular systems, with secondary emphasis on muscle, bone, and adipose tissue. Importantly, the hormonal system provides a major connection among various organs and tissues ensuring coordinated responses and adaptations to exercise. A full discussion of all the hormonal responses to various types of exercise is well beyond the scope of this textbook, but this is an important area of research that many students of exercise physiology may wish to explore further.

Hormonal Regulation of Metabolism

Hormones play a critical role in regulating metabolism by controlling GI tract motility and the absorption of foodstuff, the storage and release of fuel, and the metabolic activity of cells. The overall goal of hormonal regulation of metabolism is to ensure that metabolic fuels (carbohydrate, fat, and protein) are available continuously to meet the metabolic needs of the organism and that excess fuels are stored. Additionally, the hormonal regulation of metabolism ensures that blood glucose levels are maintained at levels necessary to supply the nervous tissue with required amounts of glucose. The challenge of maintaining appropriate blood glucose and mobilizing lipids and amino acids when needed occurs because we ingest food only periodically (typically 2–6 times a day), but the cells constantly need fuel available to support metabolic activity—and the metabolic needs of the body may change drastically, as they do with exercise.

The goals of the endocrine system relative to metabolic

activity during exercise are to:

1. Mobilize fuel for the production of ATP energy needed to support muscle contraction
2. Maintain blood glucose levels (because neural tissue can use only glucose to produce energy)

Glucagon, epinephrine, norepinephrine, growth hormone (GH), and cortisol operate together under the permissive influence of triiodothyronine (T₃) to accomplish the first goal. Glucagon and insulin act antagonistically; glucagon levels increase, and insulin levels are simultaneously suppressed during exercise. As shown in **Figure 21.9**, glucagon, NE, E, GH, and cortisol affect three primary target cells: adipose, liver, and skeletal muscle cells. When these hormones bind to certain (beta adrenergic) receptors on adipose cells, fat storage is inhibited and fat mobilization and uptake enhanced. When these hormones bind to receptors on the liver, glycogen (chains of glucose molecules chemically linked together) is broken down to glucose, and additional glucose is synthesized from other sources, such as alanine (an amino acid), glycerol, or lactate. When these hormones bind to receptors on skeletal muscle, stored glycogen is broken down to glucose. These hormones also cause skeletal muscles to increase their uptake and utilization of fatty acids, which aids in providing substrate for ATP resynthesis.

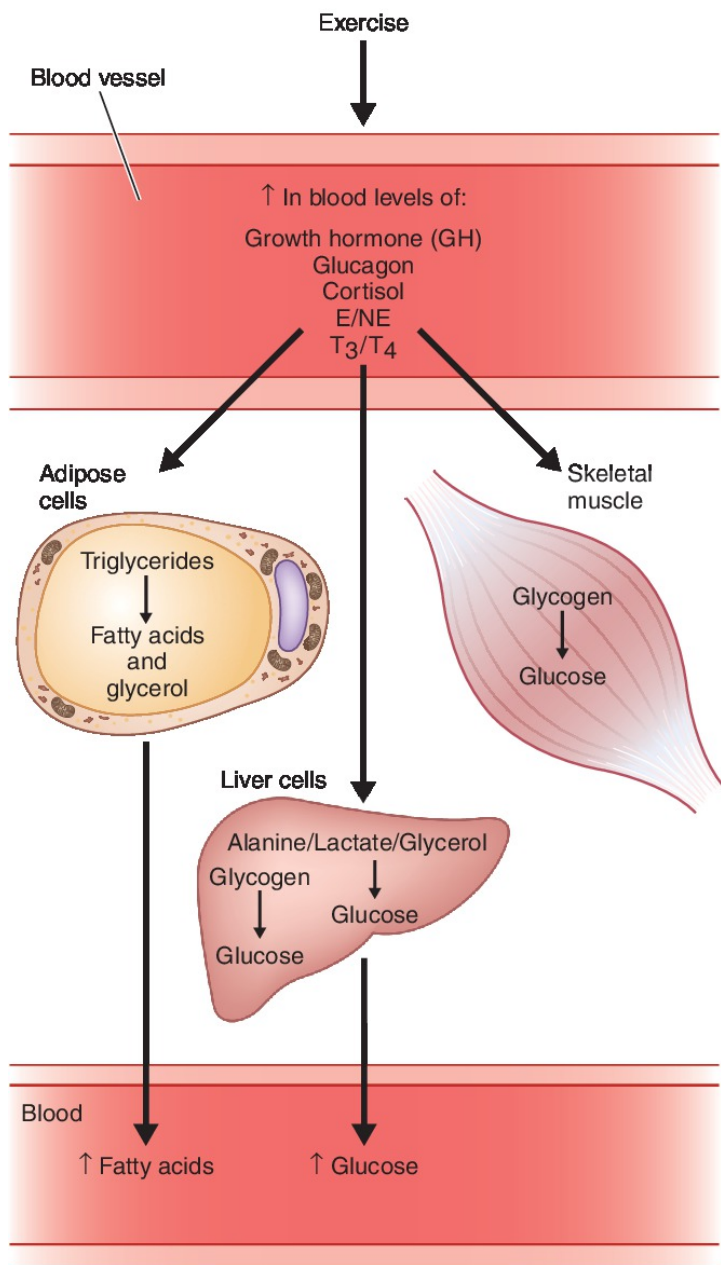


Figure 21.9 Effect of Metabolic Hormones.

Metabolic hormones affect adipose cells, liver cells, and skeletal muscle. The net effect of the metabolic hormones is

to increase fuel availability.

Hormonal Regulation of Cardiovascular Function

The goals of the hormonal system relative to cardiovascular function during exercise are to:

1. Enhance cardiac function
2. Distribute blood to active tissues
3. Maintain blood pressure by stabilizing fluid and electrolyte balance

Enhanced cardiac function and distribution of blood to working muscles are primarily accomplished by E and NE released from the adrenal medulla. These adrenal hormones reinforce the actions of the SNS (see earlier section on ANS Control During Exercise). In general, the two hormones have similar effects, but E is more important in increasing blood flow to skeletal muscle and the heart, and NE is more influential in causing vasoconstriction and increasing blood pressure.

Two hormones predominate in the stabilization or maintenance of fluid and electrolyte balance: antidiuretic hormone (ADH), sometimes called vasopressin, and aldosterone. These hormones act on the kidneys to increase water resorption and retain or excrete specific electrolytes ([Bunt, 1986](#)). By maintaining fluid and electrolyte balance, these hormones positively affect blood volume and blood pressure as well (see [Chapter 11](#)).

FOCUS ON APPLICATION

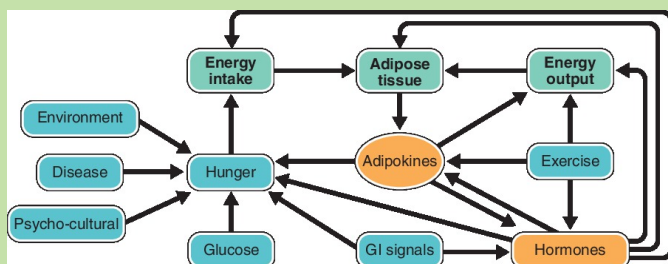
Multiple Factors Affect Energy Balance and Adiposity

Exercise professionals routinely prescribe exercise to increase caloric expenditure (energy output) in an attempt to maintain caloric balance or to create an energy deficit (calorie expenditure > calorie intake) to decrease adiposity. Much of [Chapter 8](#) in this text is devoted to such issues. This chapter focuses on hormonal responses to exercise, including the hormones that regulate hormonal adjustments to exercise.

Below is a schematic illustration of factors that influence energy intake and output and their relationship to adiposity.

Exercise professionals prescribing exercise for energy balance must consider many factors. The accompanying schematic reminds us:

1. A large number of factors affect energy balance.
2. The various factors interact extensively.
3. Hormones have a central role in the body's responses to exercise, in regulating hunger, and in influencing adipose tissue.
4. Adipose tissue releases adipokines that in turn affect other hormones, the exercise response, and hunger.



Source: Adapted by permission from Springer: McMurray, R. G., & A. C. Hackney: Interactions of metabolic hormones, adipose tissue and exercise. *Sports Medicine*. 35(5):393–412 (2005).

Hormonal Involvement in Muscle, Bone, and Adipose Tissue

Hormones involved in the structure and function of muscle, bone, and adipose cells are not primarily responsible for the immediate goals of supporting acute exercise (although some, like myokines, may be involved). Many are important during recovery and generally facilitate training adaptations, especially changes in body composition (Bunt, 1986; Kraemer, 1992b; Shai et al., 2015); therefore, they will be discussed briefly. Tissue repair mechanisms are activated during recovery after all exercise sessions. Hormones that impact muscle, bone, and adipose tissue include growth hormone, insulin-like growth factors (IGF), testosterone, estrogen, progesterone, calcitonin, parathyroid hormone (PTH), and leptin. The role of PTH, calcitonin, and estrogen in bone health is detailed in Chapter 16.

The impact of GH on protein synthesis in muscle is mediated through IGF. GH stimulates the release of IGF from the liver, and both muscle and connective tissue produce IGF. Specific effects of IGF include amino acid uptake, muscle synthesis, connective tissue (collagen) synthesis, bone and cartilage growth, and maintenance of fat-free muscle mass. Testosterone is an anabolic hormone responsible for the high ratio of muscle mass to fat mass that occurs in males at adolescence. Testosterone stimulates the release of GH and IGF, and influences neural factors contributing to anabolic processes to help regulate muscle synthesis and breakdown.

Adipose tissue has long been thought of as a mere storage depot for body fat, but we now know that adipose tissue is also an endocrine organ that plays an important role in regulating energy metabolism (Berggren et al., 2005; Fasshauer and Bluher, 2015; Galic et al., 2010; Greenberg and Obin, 2006; McMurray and Hackney, 2005; Ronti et al., 2006). As shown in **Figure 21.10**, adipose tissue secretes a number of cytokines, specifically termed adipokines because they are released from adipose tissue, although they are chemically identical to cytokines released from immune cells. To date, several hundred adipokines have been discovered (Blüher, 2016; Blüher and Mantzoros, 2015). **Adipokines** are bioactive peptides released from adipose tissue that act locally and systemically through autocrine, paracrine, and endocrine effects. Adipokines influence multiple organs/systems and have a myriad of physiological roles, including mediating appetite (satiety), energy balance, and eating

disorders; influencing insulin sensitivity and lipid metabolism; helping regulate cardiovascular and immune function; as well as providing feedback that affects adipose tissue secretion and endocrine function.

Adipokines Adipokines are bioactive peptides released from adipose tissue that influence multiple organs/systems and have a myriad of physiological roles.

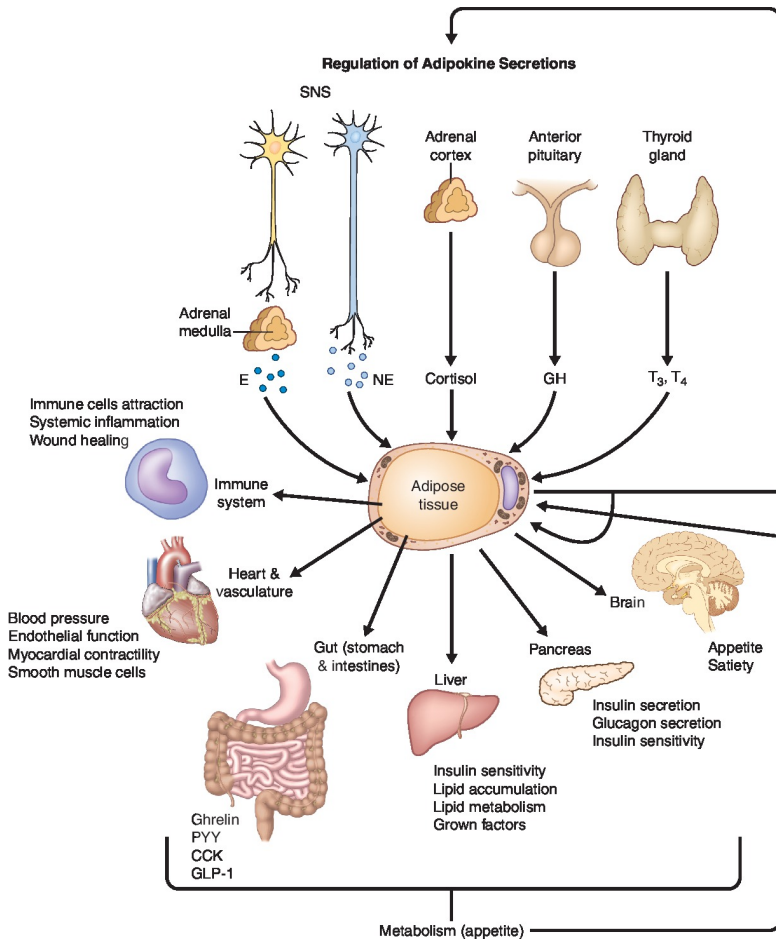


Figure 21.10 Adipose Tissue as an Endocrine Gland.

Adipose tissue secretes several peptides, collectively called

adipokines, which have widespread and coordinated functions throughout the body. Adipokines also help modulate a multitude of neurohormonal factors that, in turn, regulate the secretion of adipokines from adipose tissue.

The most prominent adipokine is the satiety hormone, leptin. Deficiencies in leptin or leptin receptors are associated with obesity (Tomas et al., 2004) (Chapter 8). Adiponectin is another adipokine and is associated with insulin resistance and the metabolic syndrome. Other adipokines are important mediators of inflammation and are discussed throughout this text in relation to vascular inflammation (Chapter 15), muscle damage (Chapter 19), and overtraining (Chapter 22). Adipokines interact extensively with the cardiovascular system (Farkhondeh et al., 2020) and play a role in the regulation of vascular function, blood pressure control, and hemostasis (Blüher and Mantzoros, 2015; Mishra et al., 2016). Adipokines also affect the GI system, which releases gut hormones (ghrelin [stomach] and peptide YY [PYY], cholecystokinin [CCK], glucagon-like peptide 1 [GLP1] [intestines]) that play key roles in regulating appetite (Schubert et al., 2014). An exciting new area of research is the role of beige and brown adipose tissue (BAT; see Chapters 7 and 8) in endocrine function (Villarroya et al., 2013, 2017). Historically, research has been focused on white adipose tissue (WAT), while recent research is now investigating the role of BAT in a healthy metabolic profile. Adipose tissue, including WAT, beige, and BAT, functions as an endocrine organ and plays an important role in integrating the functions of the neuroendocrine system, the immune system, and metabolism. These physiological links help explain the role of increased adiposity in the development of atherosclerosis, hemostatic imbalance, insulin resistance, and the development of the metabolic syndrome.

Hormonal Responses to Exercise

It should be fairly obvious that the action of most of the

aforementioned hormones increases during exercise (except for insulin). Without an increase in hormonal secretion, the enhanced functions just described would not occur. What is not so obvious is the pattern of response seen in blood concentrations of each of these hormones to exercises of different intensities, durations, and metabolic demands. Recall that changes in blood concentration of hormones may not simply indicate an increase in secretion, because hormonal levels are affected by changes in clearance rates, blood volume, receptor-binding turnover, and other factors (Bunt, 1986; Hackney and Lane, 2015; Kraemer, 1992a). The following section presents what is known about hormonal response patterns in relation to the six categories of exercise used throughout this text:

1. Short-term light to moderate submaximal aerobic exercise
2. Long-term moderate to heavy submaximal aerobic exercise
3. Incremental aerobic exercise to maximum
4. Static exercise
5. Dynamic resistance exercise
6. Very short-term high-intensity anaerobic exercise

In many instances, the hormonal responses in all six categories of exercise have not been identified. In general, much more is known about long-term moderate to heavy aerobic exercise and incremental exercise to maximum than other categories of exercise.

Although it might seem overwhelming to consider how different hormones respond to various categories of exercise, it is important to appreciate the differences in hormonal response based on the type of exercise performed. Indeed, many of the metabolic and cardiorespiratory responses and muscular and body composition adaptations described earlier in this text are determined in large part by these endocrine responses.

Metabolic and Cardiovascular Hormones

Epinephrine ***and*** ***Norepinephrine***

(Catecholamines)

Epinephrine (E) and norepinephrine (NE) are released from the adrenal medulla as a result of SNS stimulation. These hormones have widespread actions throughout the body and affect both the metabolic and cardiorespiratory responses to exercise. When interpreting the responses of E and NE to exercise, remember that NE is also released from sympathetic nerve endings. Because neural stimulation occurs more quickly than the endocrine response, the initial increase in NE in response to exercise is from the SNS (see **Figure 21.2**). Both E and NE are later released from the adrenal medulla. In this light, it should not be surprising that NE shows an elevation at lower workloads than E and that blood levels are generally higher for NE than E ([Galbo, 1983](#)).

The catecholamines are often called the “stress hormones,” and they have widespread effects throughout the body and play an important role in coordinating adjustments to exercise, particularly intense exercise. Cardiovascular adjustments such as an increased heart rate and force of contraction and vasoconstriction in nonworking muscles are accomplished by the actions of the SNS and augmented and reinforced by circulating E and NE. Catecholamines are critical to the control of circulation, blood clotting, and immune defense and thus warrant considerable attention.

Minimal increases in both E and NE occur during short-term light to moderate submaximal aerobic exercise (**Figure 21.11A and B**). During long-term moderate to heavy submaximal aerobic exercise, the increase depends on time and is gradual if energy is supplied aerobically up to the point of fatigue (**Figure 21.11C and D**). Incremental aerobic exercise to maximum elicits positive exponential increases in both E and NE (**Figure 21.11E and F**), clearly indicating that a lower limit of submaximal intensity must be exceeded before a response is achieved and that above that point the increase in hormonal level depends on intensity ([Acevedo et al., 2007](#)). NE starts to spill out of organs by sympathetic nerve stimulation at intensities close to 70% of

$\dot{V}O_2 \text{ max}$. E and NE from the adrenal medulla are secreted at intensities above 80–90% of $\dot{V}O_2 \text{ max}$ ([Borer, 2003](#)). The E and NE response to static exercise (**Figure 21.11G and H**) is

larger than during dynamic resistance exercise of equal heart rate or aerobic energy demand (oxygen consumption), and the rise in plasma E seems to be larger relative to NE than during aerobic exercise ([Galbo, 1983](#)). The response of E and NE to dynamic resistance exercise appears to be related to the force of muscle contraction, the amount of muscle tissue stimulated, and the amount of rest between repetitions (**Figure 21.11I and J**) ([Kraemer, 1988](#); [Kraemer and Ratamess, 2005](#)). Very short-term high-intensity anaerobic work, such as in the Wingate anaerobic test (WAT), results in a large increase in both E and NE ([Vincent et al., 2004](#)).

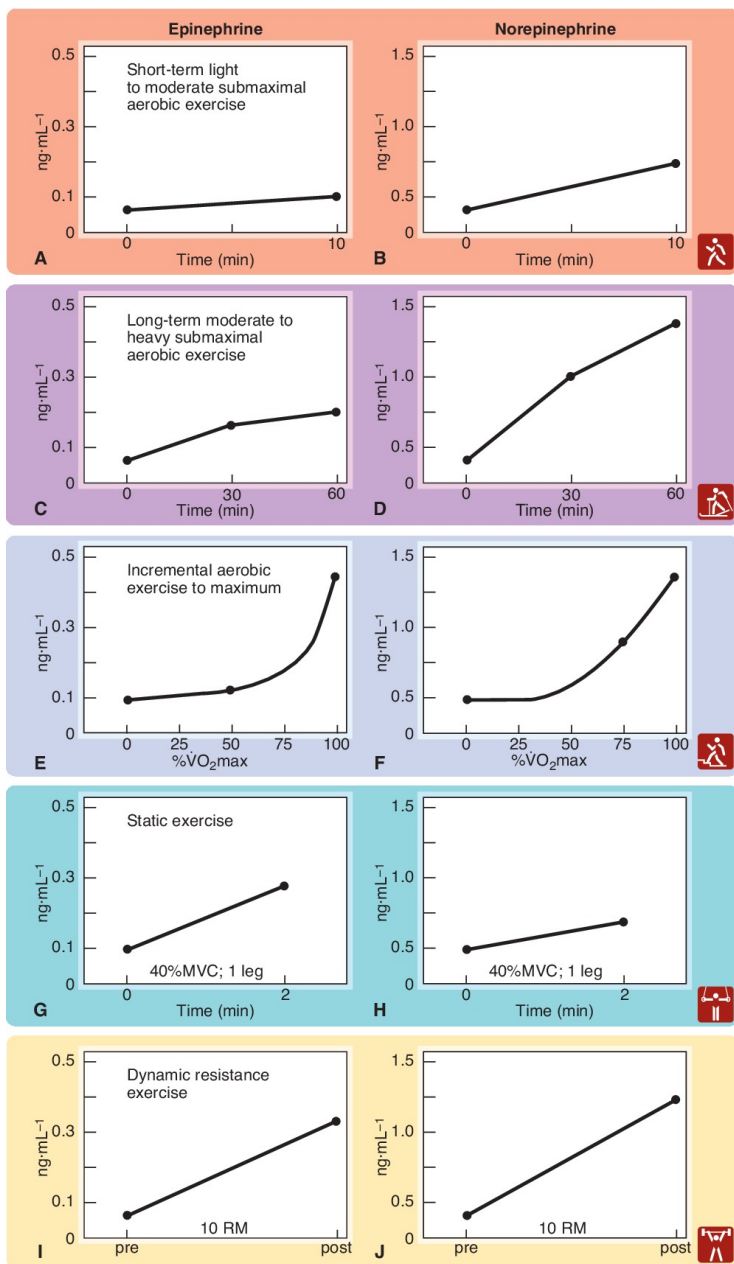


Figure 21.11 Epinephrine and Norepinephrine Responses to Various Categories of Exercise. A, B. Short-term light to moderate submaximal aerobic exercise. C, D.

Long-term moderate to heavy submaximal aerobic exercise. **E, F.** Incremental aerobic exercise to maximum. **G, H.** Static exercise. **I, J.** Dynamic resistance exercise. **Sources:** [Galbo \(1983\)](#); [Kraemer \(1988\)](#).

Fluid Balance Hormones

Exercise causes rapid shifts in body fluid compartments, including a rapid decrease in plasma volume. Plasma volume may decrease by approximately 15% in the first few minutes of exercise, largely because the increase in blood pressure leads to an increase in capillary filtration pressure that pushes plasma into the interstitial space. Additionally, sweat loss during prolonged exercise can cause further reductions in plasma volume. Protecting against excessive fluid loss is important to maintain an aqueous environment for metabolic reactions, to maintain thermal balance, and to support cardiovascular function. Several hormones function together to help maintain fluid balance in response to exercise stress.

Antidiuretic hormone, released from the posterior pituitary, increases water retention, whereas aldosterone, released from the adrenal gland, increases salt and water retention. Thus, these two hormones act to maintain plasma volume. Aldosterone is linked with angiotensin II and renin. Renin converts angiotensinogen to angiotensin I, which is subsequently converted to angiotensin II, which initiates a signal for the adrenal glands to release aldosterone. Angiotensin II is also a potent vasoconstrictor helping to maintain blood pressure during vasodilation in working muscles. Thus, renin, antidiuretic hormone, and aldosterone work in concert to help maintain fluid balance, and thereby cardiovascular function, during exercise. (See [Chapter 11](#) for a discussion of the hormonal control of blood volume.) Changes in renin, antidiuretic hormone, and aldosterone—known as fluid balance hormones—occur parallel to each other. They highly correlate with changes in NE and have similar time courses. **Figure 21.13E** depicts the positive exponential response of ADH to incremental exercise to maximum ([Wade, 2000](#)).

Metabolic Hormones

A number of overlapping hormonal responses help ensure that skeletal muscles can increase their metabolic activity during exercise. As discussed earlier, the catecholamines play an important role regulating metabolic responses to exercise (by increasing lipolysis, stimulating insulin, and suppressing glucagon). This section focuses on several other metabolic hormones directly involved in regulating the metabolic response to exercise.

The exercise responses of the metabolic hormones have been studied most extensively during long-term moderate to heavy submaximal aerobic exercise and incremental aerobic exercise to maximum. Therefore, what is known about the exercise response of these hormones applies primarily to these categories of exercise. Although plasma levels are presented for each hormone in the figures, keep in mind that changes in plasma levels may reflect not changes in secretion but rather changes in clearance rates. The responses might also reflect ANS changes.

Insulin and Glucagon

Insulin operates to store fuel, whereas glucagon's role is to mobilize fuel. Therefore, in general, the responses of these two hormones are close to mirror images of each other. The ratio of glucagon to insulin primarily controls fuel mobilization, however. Insulin has multiple actions, including increasing glucose and free fatty acid uptake and decreasing glycogenolysis and gluconeogenesis. In contrast, glucagon stimulates glycogenolysis and gluconeogenesis and is important for maintaining blood glucose levels during prolonged exercise.

During short-term light to moderate submaximal aerobic exercise, insulin declines a small amount initially before leveling off, and glucagon rises a small amount initially before leveling off (Galbo, 1983). Long-term moderate to heavy submaximal aerobic exercise causes an initial rapid increase in glucagon followed by a gradual increase (**Figure 21.12A**) and a complementary initial rapid drop in insulin followed by gradual decline (**Figure 21.12B**) (Galbo, 1983). During incremental exercise to maximum, glucagon increases in a positive exponential manner.

The increase in glucagon is proportionally greater as exercise intensity increases than is the decline in insulin (**Figure 21.13A and B**). Indeed, at high workloads ($>60\% \dot{V}O_2 \text{ max}$), plasma insulin levels begin to rise again, resulting overall in a truncated U-shaped curve that remains below resting levels. Neither static activity nor short-term high-intensity exercise (such as sprinting or dynamic resistance exercise) appears to affect either insulin or glucagon much, because fuel demands for these activities do not require extensive mobilization. However, some authors have reported decreased insulin levels following a bout of resistance exercise ([Raastad et al., 2000](#)).

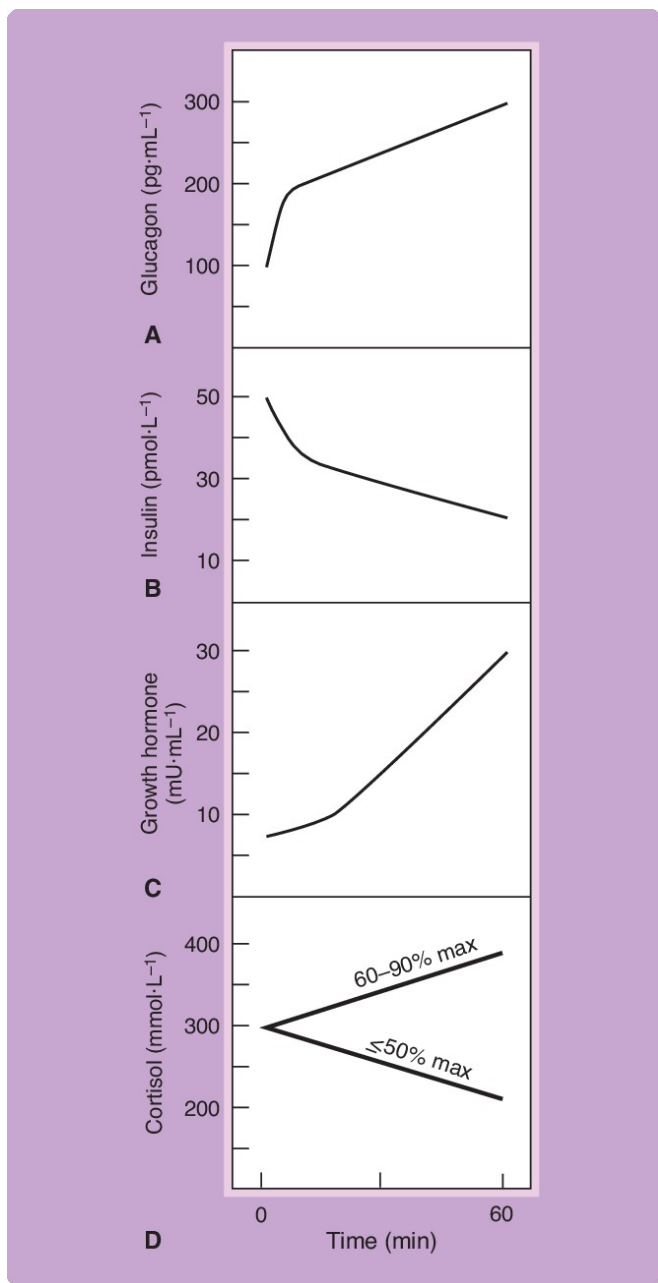


Figure 21.12 Hormonal Responses to Long-Term Moderate to Heavy Submaximal Aerobic Exercise.



A. Glucagon. **B.** Insulin. **C.** Growth hormone. **D.** Cortisol.

Sources: [Galbo \(1983\)](#); [Sutton et al. \(1990\)](#).

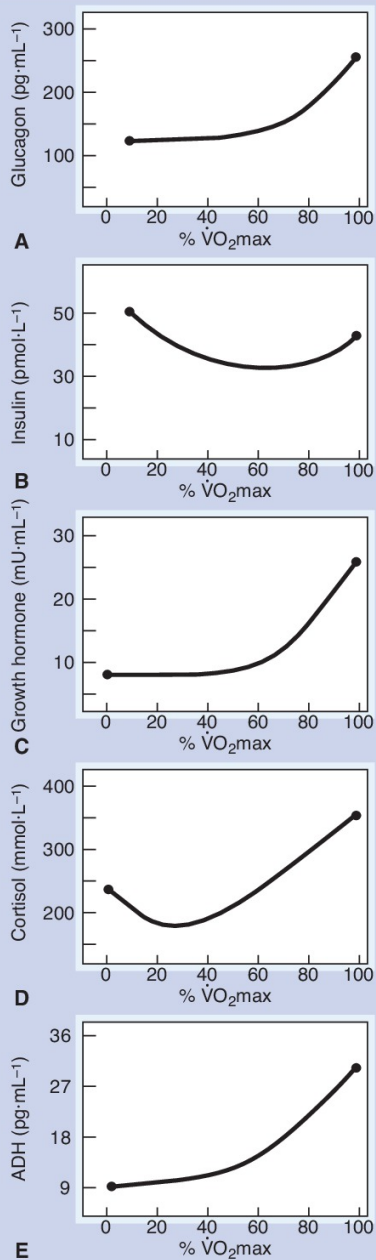


Figure 21.13 Hormonal Responses to Incremental



Aerobic Exercise to Maximum.

A. Glucagon. B. Insulin. C. Growth hormone. D. Cortisol. E. Antidiuretic hormone (ADH). **Sources:** [Galbo \(1983\)](#); [Sutton et al. \(1990\)](#); [Wade \(2000\)](#).

Growth Hormone

Exercise causes an increase in circulating GH that is influenced by intensity and duration, fitness level, age, gender, nutritional status, and body composition ([Nindl et al., 2014](#)). As a protein hormone, GH has a slow rate of response, secretion, and clearance ([Galbo, 1983](#)). Thus, there is a delay between the onset of exercise and changes in blood hormonal levels. The greater the intensity of the exercise, the shorter this delay is. Short-term light to moderate submaximal aerobic exercise and static exercise are too brief for any changes to become apparent. A short but high-intensity exercise causes a peak value in GH that may occur from 15–30 minutes into recovery.

Long-term moderate to heavy submaximal aerobic exercise leads to a gradual increase in GH over 30–60 minutes (**Figure 21.12C**) ([Galbo, 1983](#)). If the activity continues much longer, however, as in a marathon, GH concentrations return to near baseline levels. With incremental aerobic exercise to maximum (**Figure 21.13C**), GH concentration increases with increasing workloads in a positive exponential fashion after the initial delay.

GH release increases during and following resistance exercise. High total work and short rest periods are associated with a much larger increase in GH than low total work volume and long rest periods ([Kraemer, 1992a](#); [Thomas et al., 2013](#)). Similarly, exercise using large muscle mass results in greater elevations of GH than those using a small muscle mass. Research suggests that the metabolic properties of the muscle affect the GH response. That is, protocols that cause high lactate levels (e.g., programs that use moderate- to high-intensity and high-volume exercise that stress large muscle mass and that use relatively short rest intervals, thus stressing the fast glycolytic fibers) produce the most substantial increases in GH in both men and women

(Kraemer and Ratamess, 2005; Thomas et al., 2013). GH response to exercise can be influenced by a variety of factors such as oxygen demand and availability, afferent signals from muscle metabolic receptors, motor center activity, catecholamines, and changes in core temperature (Thomas et al., 2013). Evidence also suggests that GH response is affected by menstrual state (normal cycling or disordered menstrual cycle) (Nakamura et al., 2011). Although research has consistently shown an increase in GH with exercise, researchers have also investigated the amplitude and pulsatile release of GH during recovery from exercise as this appears to be related to desired exercise outcomes such as decrease in fat mass (Weltman et al., 2008; Wideman et al., 2006). See Focus on Research Box for analysis of a study investigating the secretory profile of GH following aerobic and resistance exercise.

The effect of exercise on IGF is less clear than the response of GH. IGF levels may rise immediately following acute exercise; however, these findings are inconsistent (Nemet et al., 2003). Research has reported both increases and decreases in IGF concentration despite increases in circulating GH (Kraemer et al., 1995; Nemet et al., 2003). A recent meta-analysis found that total IGF is increased following both a single bout of aerobic and resistance exercise, but free IGF was not significantly increased by either type of exercise (de Alcantara Borba et al., 2020).

Cortisol

Like GH, the steroid-based cortisol is a slow-acting hormone. In addition to multiple roles in metabolism, cortisol has numerous biological functions throughout the body, as outlined in **Table 21.4**. Cortisol is also considered a “stress hormone” because it mediates many responses to stress, particularly chronic or long-term stress. As such, cortisol is an important mediator of immune response to exercise (see **Chapter 22**).

TABLE 21.4 Multiple Effects of Cortisol on Various Bodily Functions

| Category of Function | Cortisol Activities |
|-----------------------|--|
| Metabolism | ↑ Protein degradation in muscle and connective tissue ↓ Protein synthesis in muscle and connective tissue ↓ Amino acid transport into muscle ↑ Amino acid transport into liver ↑ Gluconeogenesis ↑ Glycogen synthesis in liver ↓ Glucose uptake and utilization ↑ Lipolysis |
| Immunity | ↓ Inflammatory cytokines (IL-1 and IL-6) ↓ Capillary permeability ↓ Phagocytosis ↓ T lymphocytes |
| Circulation and blood | ↑ Vasoconstriction ↑ Blood volume (body fluid retention) ↑ Erythrocyte and leukocyte synthesis |
| Kidney function | ↑ Glomerular filtration rate ↑ Sodium retention |
| Brain functions | ↑ Mood (euphoria) ↓Mood (depression) ↓ Taste, hearing, and smell ↓ Food intake |

Source: Modified from Borer (2003).

FOCUS ON RESEARCH

Growth Hormone Secretory Profiles after Aerobic and Resistance Exercise

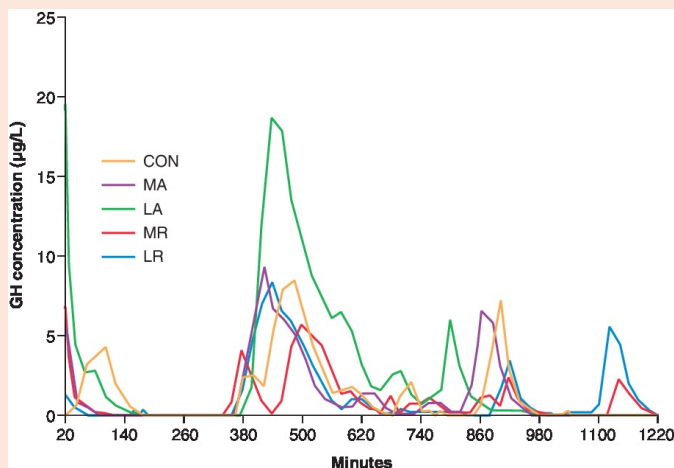
Growth hormone levels increase with exercise, and the increase in GH is thought to play an important role in the anabolic and lipolytic actions that accompany exercise. In fact, many people engage in exercise training programs in order to increase muscle mass and decrease fat mass, and these changes are mediated, at least partially through GH. Thus, understanding the GH response to different types of exercise is of considerable interest, and the effect of GH is not dependent solely on the blood concentrations. Rather, the structure of the GH molecule and the amplitude of its pulsatile release may be even more important than

circulating concentrations. Thus, Nindl and colleagues undertook a study to document GH secretion profiles during a 20-hour recovery period after aerobic and resistance exercise bouts. This study included eight healthy male volunteers who were recreationally trained and who participated in five independent conditions in random order:

1. Control
2. A moderate-duration aerobic exercise session (MA: 1 hour; 3 bouts of 15-minute cycling at 70% of VO_2 peak; 5- to 7-minute rest b/t bouts)
3. A long-duration aerobic exercise session (LA: 2 hours; 6 bouts of 15-minute cycling at 70% of VO_2 peak; 5- to 7-minute rest b/t bouts)
4. A moderate-duration resistance exercise session (MR: 1 hour; 25 sets 5 or 10 RM/set; 90- to 120-second rest intervals)
5. A long-duration resistance exercise program (LR: 2 hours 50 sets 5 or 10 RM/set; 90- to 120-second rest intervals)

The accompanying figure shows the GH concentration peak profiles for a representative volunteer immediately after and for 20 hours after each of the exercise conditions. Long-duration aerobic exercise had the greatest effect on GH concentration. Statistical analysis revealed that only long-duration aerobic exercise caused a significant amplification of GH release.

The authors speculated that the increased GH release following long-duration aerobic exercise is due to the higher energy demands of this exercise and the greater demand for postexercise fat mobilization.



Source: Reprinted with permission from Nindl, B. C., J. R. Pierce, K. R. Rarick, A. P. Tuckow, J. A. Alemany, M. A. Sharp, M. D. Kellogg, & J. F. Patton: Twenty-hour growth hormone secretory profiles after aerobic and resistance exercise. *Medicine & Science in Sports & Exercise*. 46(10):1917–1927 (2014). Copyright ©2014 The American College of Sports Medicine.

It is difficult to generalize a pattern of cortisol's exercise response because of the initial delay in exercise-enhanced concentrations and because in short-duration and/or low- to moderate-intensity activity, clearance of the hormone exceeds secretion. This means that although secretion may increase, blood concentrations actually decrease (Sutton et al., 1990). This outcome can be seen in **Figure 21.12D**, which depicts the response of cortisol to long-term moderate to heavy exercise.

Work intensity less than or equal to 50% of $\dot{V}O_2 \text{ max}$ results in a steady decrease in blood cortisol level, whereas intensity loads from approximately 60–90% of $\dot{V}O_2 \text{ max}$ cause a gradual rise in blood cortisol level over time, despite a constant workload. The truncated U pattern for incremental exercise to maximum seen in **Figure 21.13D** reinforces the importance of intensity in the cortisol response. Indeed, cortisol may not increase until anaerobic metabolism makes a significant contribution to the

total energy supply. Anaerobic exercise causes greater increases in cortisol than aerobic exercise, even at the same total work output, probably because of the greater intensity (Kraemer, 1988, 1992a). Further research suggests that greater intensity exercise, such as high-intensity interval training, causes a transient increase in cortisol and appears to promote anabolic processes (Wahl et al., 2013). Dynamic resistance exercise causes large increases both during and after high-intensity sessions, probably because of its large anaerobic component. Exercise involving large muscle groups results in greater elevations of cortisol than exercise involving small muscle groups (Smilios et al., 2003). Cortisol levels in the blood remain elevated up to several hours after exercise. Exercise programs causing the greatest change in GH (and lactate) also result in the most substantial changes in cortisol (Kraemer and Ratamess, 2005).

Thyroid and Parathyroid Hormone

The pattern of thyroid and parathyroid hormonal responses to the different categories of exercise is unclear (Galbo, 1983). Numerous studies have reported no change in blood concentrations of triiodothyronine (T₃) or thyroxine (T₄). However, it is possible that free T₃ and free T₄ change without resulting in changes in the total concentration of thyroid hormone; this lack of change does not mean an unchanged turnover (Berent and Wartofsky, 2000). Following long-term heavy aerobic exercise, free T₄ increases slightly, while free T₃ declines gradually over time, resulting in nonpathological hypothyroidism. It is believed that reduced secretion from the thyroid gland, reduced conversion of T₄ to T₃, and increased target tissue uptake may contribute to the exercise-induced hypothyroid state (Hackney et al., 2012).

Similarly, PTH concentrations have been shown to increase during endurance exercise, with low parathyroid concentrations present during recovery (Scott et al., 2014). The data are insufficient to draw conclusions regarding PTH responses to different exercise intensities. Additionally, limited research exists in relation to calcitonin hormone responses to exercise.

The Influence of Menstrual Cycle Hormonal Fluctuations on Physiological Responses to Exercise

Although this chapter focuses on the effects of exercise and exercise training on the neurohormonal system, fluctuations in estrogen and progesterone during the menstrual cycle can potentially cause changes in exercise performance or the physiological responses and adaptations to exercise/exercise training. Current literature suggests that fluctuations in female reproductive hormones do not affect muscle contractile characteristics or $\dot{V}O_2 \text{ max}$ (and its determinants). Similarly, neither maximal strength nor intense anaerobic activities appear affected by the menstrual cycle. During prolonged exercise in the heat, however, the exercise time to exhaustion decreases during the midluteal phase (when progesterone is increased and body temperature is elevated) (Janse de Jonge, 2003; Marsh and Jenkins, 2002).

There is some controversy concerning the lactate response to exercise during various phases of the menstrual cycle that may be the result of different analysis techniques.

Forsyth and Reilly (2005) studied 11 endurance-trained eumenorrheic (regularly cycling) female athletes not taking oral contraceptives at the midfollicular (6–10 days after menses) and midluteal (6–10 days after the LH surge) phases of the menstrual cycle. Menstrual phase was confirmed by blood hormonal analyses. Subjects were tested at 06:00 and 18:00 hours on a Concept II rowing machine using an incremental test to maximum. Lactate threshold (LT1) was determined in three ways: by a mathematical curve-fitting procedure, by the visual procedure shown in Chapter 3 (Figure 3.15), and at the fixed blood lactate concentration [La] of 4.0 mmol·L⁻¹ (LT2). Ventilatory threshold (VT1) was also determined. At rest and a fixed exercise intensity, [La]

was significantly lower in the midluteal phase than in the midfollicular phase. In the midluteal phase, LT at 4.0 mmol·L⁻¹ occurred at a significantly higher exercise intensity, heart rate, and oxygen consumption than in the midfollicular phase. Blood lactate concentrations at VT1 and LT1 (but not power output, heart rate, or ratings of perceived exertion) using the mathematical curve-fitting procedure were significantly lower in the midluteal phase, but no changes were observed in any variable when LT1 was determined by the visual curve-fitting technique. The curve-fitting techniques provide information regarding the balance between lactate production and clearance. Below the LT1 threshold, production and clearance are equal. The glycogen-sparing effect of estrogen in the luteal phase likely causes a decrease in lactate production above LT1. This impact is masked when a set concentration is used for the lactate threshold.

These findings suggest that the menstrual cycle phase must be carefully considered when a fixed value of blood lactate is used to establish responses to training or even training load. Workouts linked to absolute lactate values may need adjustment throughout the menstrual cycle. When lactate thresholds are of interest, testing should be performed during the same menstrual cycle phase for each testing session. Alternately, use of the visual curve-fitting techniques should eliminate the problem.

Sources: [Janse de Jonge \(2003\)](#); [Marsh and Jenkins \(2002\)](#); [Forsyth and Reilly \(2005\)](#).

Hormones Related to Muscle, Bone, and Adipose Tissue

Muscle

A single bout of resistance exercise leads to acute changes in the hormonal environment (an increase in catecholamines and IGF) that is linked to the cellular processes involved in protein

turnover and muscle growth (Kraemer and Ratamess, 2005). Muscle protein turnover is an important part of protein metabolism and is necessary for muscle breakdown (during exercise) and subsequent muscle repair (during recovery) (Crewther et al., 2006). High weight, low repetition resistance training sessions cause large acute increases in GH and the catabolic hormone, cortisol. On the other hand, high repetition, low weight resistance training sessions cause little or no change in GH and modest changes in cortisol.

Testosterone plays a key role in mediating tissue repair and muscle hypertrophy following resistance training and to a smaller extent following endurance training. In fact, testosterone is considered the major promoter of hypertrophy and subsequent increase in strength in men following resistance training (Vingren et al., 2010). An acute bout of high-intensity exercise leads to increased serum testosterone levels in men with the increase being greater following resistance exercise than high-intensity dynamic aerobic exercise (Kotwal et al., 2020). In general, testosterone levels in men increase acutely and then return to or below baseline within 30 minutes. The magnitude of the increase is influenced by the resistance exercise, with high-volume programs leading to greater increases (Vingren, 2010). Testosterone interacts with several other anabolic hormones, including GH and IGF, and nutritional state to promote protein synthesis following resistance exercise (Gharahdaghi et al., 2021). Protein synthesis involves multiple hormones and is dependent upon the nutritional status of the exerciser. Furthermore, there is substantial complexity due to redundancy in some of the hormonal actions, multiple receptors and binding proteins, and different pathways of action (Kraemer et al., 2017). The acute increase in testosterone following resistance exercise is seen in postpubertal boys but not in prepubertal boys or in girls. Aging men have lower basal testosterone levels and an attenuated response to a bout of resistance exercise (Paunsknis et al., 2018; Vingren et al., 2010). Women have much lower basal levels of testosterone than men, and most studies suggest that testosterone does not increase in women following resistance exercise (Kraemer et al., 1993; Vingren et al., 2010). The acute increase in testosterone in men is a consistent finding, but there remains a great deal about the effect of testosterone that remains unknown.

An increase in testosterone does not seem to be necessary for muscle growth or an increase in muscle size, as evidenced by increased muscle mass and size in women and children following resistance exercise, but testosterone does seem to optimize such results. Further, some studies have found elevated levels of testosterone among those who regularly perform resistance training, whereas other studies have not found elevated levels among resistance-trained men (Hooper et al., 2017).

Recently, research has documented that skeletal muscle also acts as an endocrine gland and releases “myokines” in response to muscle contractions. These myokines act as autocrines (have an impact on skeletal muscle itself), as paracrines, and as endocrines with an impact on the function of the liver, pancreas, cardiovascular system, and adipose tissue (white, beige, and brown). The most notable myokine, interleukin-6 (IL-6), serves to ensure fuel availability during exercise by aiding in the release of glucose from the liver and fatty acids from adipose tissue (Nimmo et al., 2013; Schnyder and Handschin, 2015).

Complete case studies 1 and 2 in the Check Your Comprehension boxes to indicate your understanding of the difference in hormonal responses to endurance and resistance exercise.

CHECK YOUR COMPREHENSION 1-CASE STUDY 1

Liz, a graduate student in kinesiology, takes a break from her research duties and goes out for a 60-minute bike ride. What hormones are involved in regulating the metabolic response to her exercise and what are the general goals of these hormones?

Check your answer in Appendix C.

CHECK YOUR COMPREHENSION 2-CASE STUDY 2

Lars, a graduate student in kinesiology, takes a break from his teaching assistantship and goes out to the weight room for a work out. What hormones are involved in mediating the

recovery from this exercise and what are the general goals of these hormones?

Check your answer in Appendix C.

Bone

Acute submaximal aerobic and resistance exercise results in elevated blood levels of estrogen and progesterone ([Chilibeck, 2000](#); [McMurray and Hackney, 2005](#)). Kemmier and colleagues ([Kemmier et al., 2003](#)) have shown that free testosterone, estradiol, and GH increase as a result of a single bout of high-impact exercise in postmenopausal females. IGF does not change, but the insulin-like growth factor-binding protein-3 increases biphasically (two peaks) during exercise and then 22 hours after exercise. This could be important for osteoporosis prevention.

Adipose Tissue

Testosterone may suppress leptin in an acute bout of exercise, but this is confounded by the duration and intensity of the exercise. In general, serum leptin concentrations are unchanged by short-duration (< ~40 minutes) light to moderate submaximal exercise. However, both very short-duration high-intensity anaerobic exercise (including resistance exercise) and long-duration moderate to heavy submaximal aerobic exercise appear to reduce serum leptin concentrations. These findings may also be related to changes in nutrient availability or flux at the adipocytes. The clinical relevance of the effects of exercise on circulating leptin concentration has not yet been established ([Hulver and Houmard, 2003](#); [Zafeiridis et al., 2003](#)). Low and moderate aerobic exercise do not cause a change in adiponectin, but levels (corrected for plasma volume changes) increase 30 minutes after heavy or maximal aerobic exercise ([Ferguson et al., 2004](#); [Jürimäe et al., 2005, 2006a, 2006b](#); [Kraemer and Castracane, 2007](#)).

Adipose tissue releases multiple inflammatory factors including IL-6. During prolonged exercise, there is a suppression of adipose-derived IL-6, but following exercise, there is a rise in

levels of IL-6. It is hypothesized that the rise in IL-6 may alter fat metabolism in recovery from exercise (Nimmo et al., 2013).

Hormonal Adaptations to Training

Training programs are not usually deliberately designed to bring about adaptations in the hormonal system. No research-based training principles have been established, and few systematic attempts have been made to vary intensity, duration, frequency, length of training period, and modality in large numbers of subjects. Some information has been derived from cross-sectional studies that compared untrained (or sedentary) individuals to trained (or fit) individuals. Some information has also been derived from testing individuals before and after a training period. It is important to understand that adaptations in one hormone may impact plasma levels of another, and changes in receptor sensitivity may not be reflected in circulating hormone levels. Furthermore, adaptations in SNS responses may cause changes in hormonal levels. As with hormonal responses to exercise, training adaptations in the metabolic, cardiorespiratory, and neuromuscular systems often result from adaptations in the endocrine system. In general, the hormonal system responds to an acute bout of exercise in the same way before and after a training program, but the hormonal response after training is typically attenuated at submaximal workloads (Hackney and Lane, 2015).

Adaptations Related to Metabolic Function

Most of what is known about hormonal training adaptations comes from studies comparing the response of individuals to a prolonged bout of exercise before and after exercise training (**Figure 21.14**). The major hormones involved in fuel mobilization (glucagon, insulin, NE, E, cortisol) all show a dampening effect as a result of exercise training; that is, the change from resting levels that occurs during exercise represents a smaller disruption of homeostasis than in the untrained state (Bunt, 1986). Given that endurance training decreases the SNS response to an absolute workload, epinephrine and NE are

expected to have muted responses (**Figure 21.14A**).

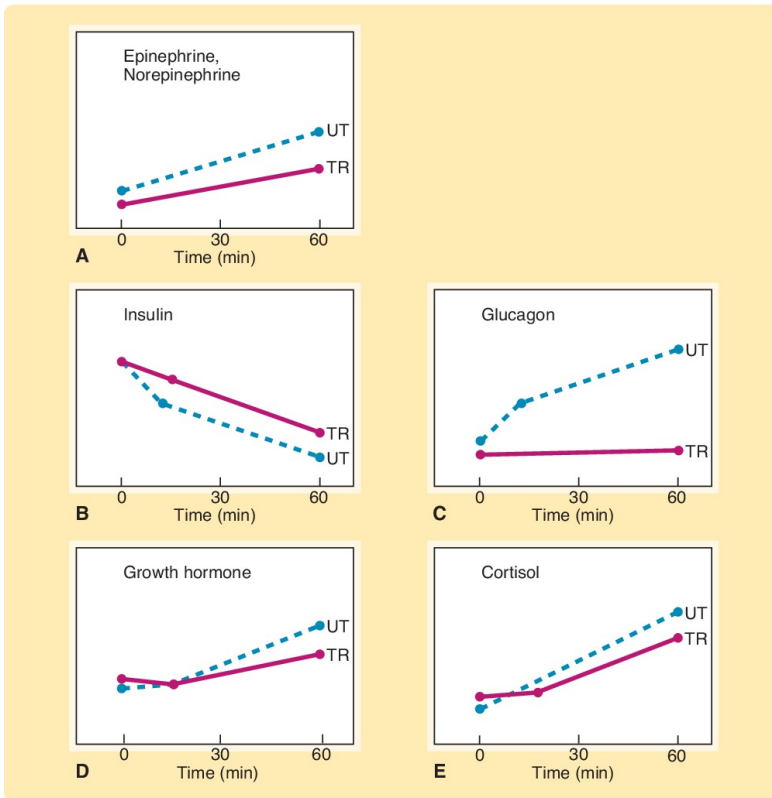


Figure 21.14 Training Adaptations Exhibited during Long-Term Submaximal Aerobic Exercise.

A. Epinephrine, norepinephrine. B. Insulin. C. Glucagon. D. Growth hormone. E. Cortisol. **Sources:** Bunt (1986); Coggan and Williams (1995); Kjaer and Lange (2000); Sutton et al. (1990).

The decline in insulin is less in the trained than in the untrained state (**Figure 21.14B**). The rise in glucagon is also less: so much less that 60 minutes of submaximal exercise may not be long enough or intense enough to cause a glucagon response in a trained individual (**Figure 21.14C**) (Coggan and Williams, 1995; Sutton et al., 1990). Increased insulin sensitivity compensates for

these changes. GH and cortisol have the same dampening effect, although both of these appear to have higher resting levels in aerobically trained than in untrained individuals (**Figure 21.14D and E**). Resistance-trained individuals appear to have unchanged resting GH concentrations (Kraemer, 1992a; Kraemer and Ratamess, 2005).

Adaptations Related to Cardiovascular Function

Neither of the hormones primarily responsible for fluid and electrolyte balance (ADH and aldosterone) show any clear training adaptation. However, plasma volume is higher in trained versus untrained individuals. The thyroid hormones adapt with an enhanced turnover rate, but too little is known about PTH to draw any conclusion.

At the end of incremental exercise to maximum, E, NE, and cortisol are increased posttraining. Higher values reflect the trained individual's ability to do more high-intensity work (Borer, 2003; Bunt, 1986; Kjaer and Lange, 2000; Sutton et al., 1990).

Adaptations Related to Muscle, Bone, and Adipose Tissue

Muscle

It has been hypothesized that IGF initially decreases as a result of training but that after an unknown length of time (probably longer than 5 weeks), resting levels of both GH and IGF increase, indicating an anabolic (tissue growth) internal environment (Eliakim et al., 2000).

The impact of chronic training on testosterone is controversial, possibly depending on the level of training. Moderate training results in an increase and extreme training in a decrease (Urhausen and Kindermann, 2000). Together, a training increase in testosterone and GH could contribute to increases in muscle mass (Kraemer, 1992b).

Bone

Moderate exercise training appears to result in elevated resting levels of estrogen and progesterone, whereas severe training results in decreased resting levels. Male and female athletes reportedly have lower PTH levels and higher bone mineral density than nonathletes ([Chilibeck, 2000](#)).

Adipose Tissue

Exercise training often leads to a decrease in overall body fat, improvement of mitochondrial function, improved fatty acid metabolism, and up-regulation of enzymes involved in the metabolism of polyunsaturated fatty acids. Exercise also affects adipokine release from adipose tissue and thus may mitigate inflammation and improve insulin sensitivity ([Mika et al., 2019](#)).

Exercise training affects hormones released from adipose tissue. Training suppresses leptin levels ([Gleim and Glace, 2000](#)) if fat mass is lost. Levels of leptin are generally low in athletes of both sexes as well as in recreational runners, consistent with low levels of body fat. Also, in the general population, activity levels and leptin have been shown to be negatively related in both sexes ([Popovic and Duntas, 2005](#)). Finally, some studies have reported decreased resting concentrations of leptin with endurance or resistance training ([McMurray and Hackney, 2005](#)). Acute low to moderate physical activity causes hormonal changes that facilitate lipolytic activity, but trained individuals may be more sensitive to hormonal signals for the mobilization of fat as a fuel source ([McMurray and Hackney, 2005](#)). Training increases resting adiponectin levels in previously sedentary individuals ([Blüher et al., 2006](#)) but not necessarily in already highly trained athletes. For example, elite rowers despite a volume-extended training season did not exhibit any resting adiponectin level changes ([Jürimäe et al., 2006a, 2006b](#)).

Hormonal Adaptations to Resistance Training

A hallmark of resistance training is an increase in muscle mass/hypertrophy. Hormones are thought to play an important role in

mediating this adaptation. **Figure 21.15** depicts a theoretical model by which hormones, and other factors, may respond to mechanical and/or metabolic stress and result in muscle hypertrophy. Multiple hormones are involved in various cellular responses that result in increased protein synthesis. This section will only address the primary hormonal adaptations that have been investigated following resistance training.

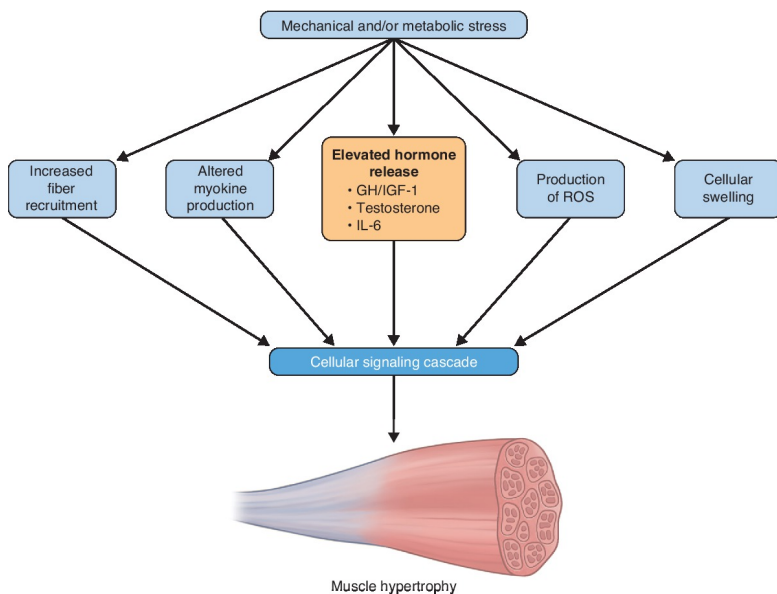


Figure 21.15 Theoretical Model of the Role of Hormones in Mediating Muscle Hypertrophy.

GH, growth hormone; IGF, insulin-like growth factor; IL-6, interleukin-6; ROS, reactive oxygen species.

Growth Hormone and IGF

It is widely thought that resistance programs designed to increase muscle mass result in increases in GH and IGF (Crewther et al., 2006). It is believed that exercise-induced elevations of GH and IGF are mediated by increased lactate and hydrogen ion concentrations in the blood whereby a reduction in pH influences the secretion of GH hormone (Schoenfeld, 2013). As seen in **Figure 21.16A and B**, a resistance training program results in a

small but significant decrease in resting GH and IGF concentrations ([Mitchell et al., 2013](#)). **Figure 21.17A and B** shows the overall response to a single bout of resistance training before and after a training program. The values represent the area under the curve for the response and are thus much larger than the resting values. The data indicate that a resistance training program results in a small but significant decrease in the amount of GH and IGF secreted during exercise. Some research also suggests that acute GH responses may differ depending on age ([Simon et al., 2015](#); [Walker et al., 2015](#)), but considerably more research is needed to understand the effect of age and gender on hormonal adaptation to resistance training.

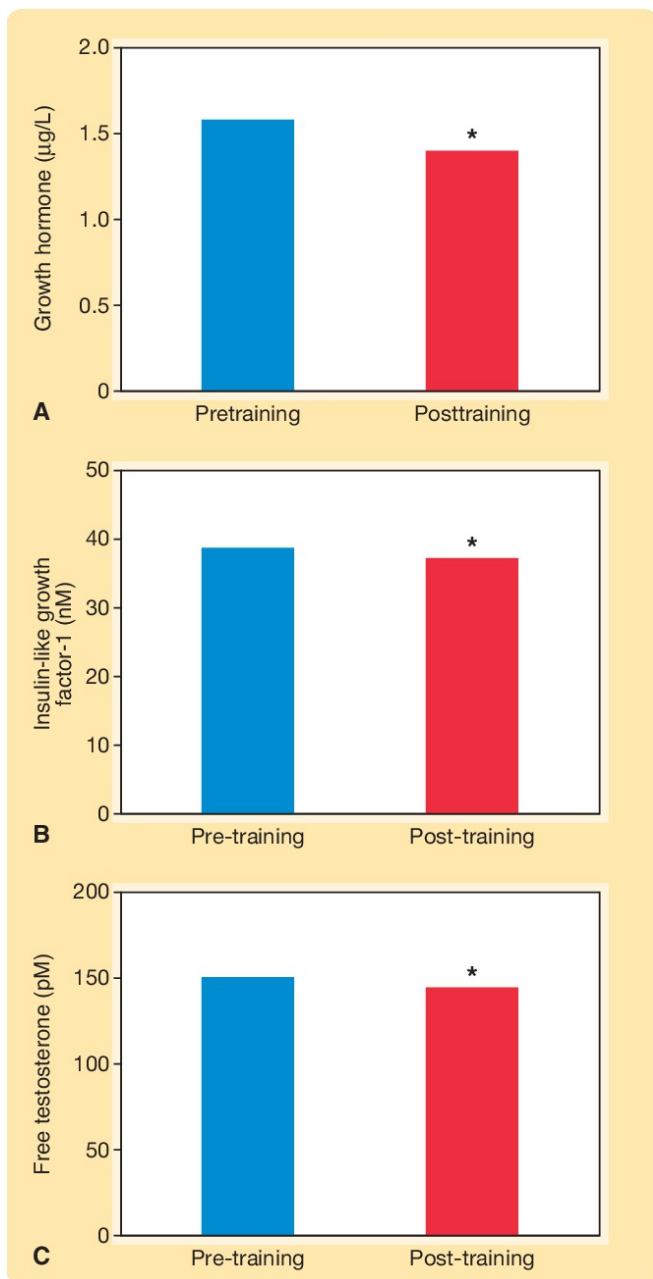


Figure 21.16 Effect of Resistance Training on Resting Levels of Growth Hormone (A), Insulin-Like Growth Factor (B), and Free Testosterone (C). * $p < 0.05$

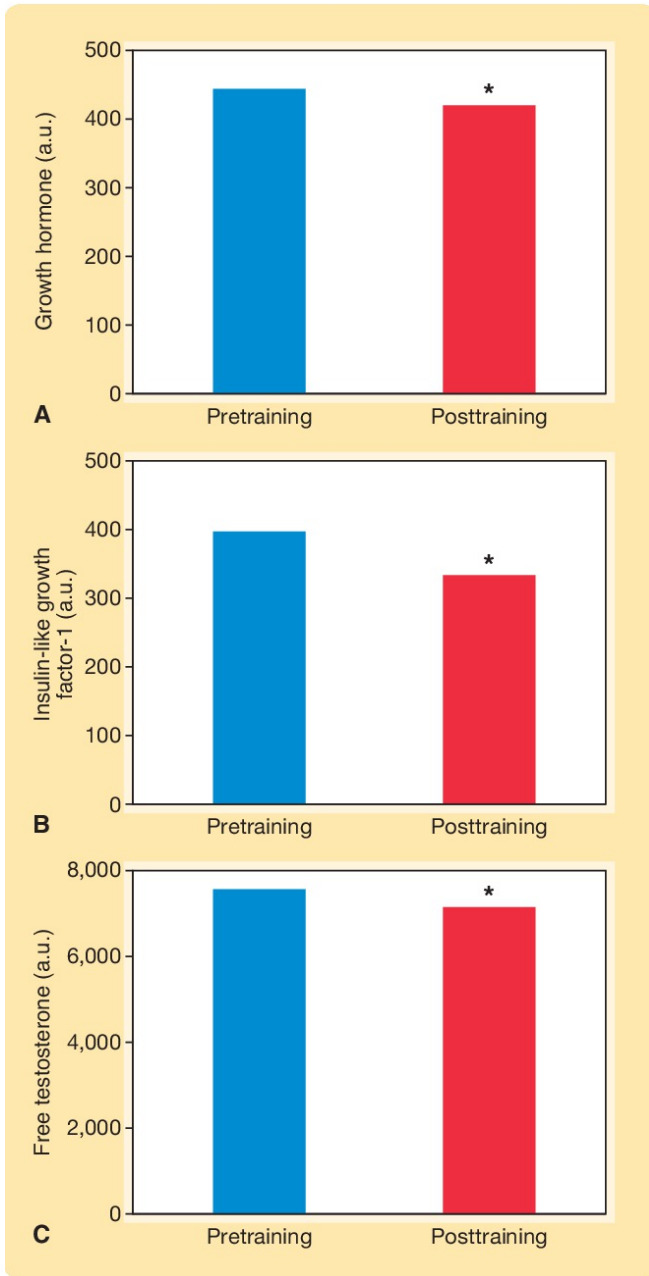


Figure 21.17 Effect of Resistance Training on Exercise Response of Growth Hormone (A), Insulin-Like Growth Factor-1 (B), and Free Testosterone (C). * $p < 0.05$

Testosterone

The effect of resistance training on testosterone appears to be dependent on factors such as age, gender, training status, and exercise modality (Kraemer and Ratamess, 2005; Schoenfeld, 2013). As seen in **Figure 21.16C**, research indicates that resistance training programs result in slightly lower resting concentrations of testosterone, although the significance of these small changes remains unclear (Mitchell et al., 2013). It is thought that testosterone only influences muscle hypertrophy in hypo- or hypertestosterone conditions, emphasizing the importance of other components within the signaling cascade. **Figure 21.17C** shows data that indicate that the free testosterone released during a single bout of resistance training is also significantly decreased following a resistance training program, but the magnitude of the decrease is small. The role of testosterone in metabolic adaptations requires further investigation to determine its effect within normal ranges (Mitchell et al., 2013).

IL-6

IL-6 is released from muscle cells and is implicated in structural adaptations following a resistance training program. However, research studies report inconsistent findings regarding IL-6 and its contribution to muscle hypertrophy. Some studies have shown increased IL-6 concentration in the blood immediately postexercise but no direct role of IL-6 in hypertrophic adaptations (Mitchell et al., 2013). Other research has found no difference in IL-6 as a result of training programs. It is possible that measuring IL-6 in the active muscle instead of blood concentrations would allow researchers to draw more meaningful conclusions regarding the influence of IL-6 on muscle size structure (Mitchell et al., 2013).

Summary

1. The nervous system and hormonal system both help maintain homeostasis and respond to the stress of exercise.

These systems interact and overlap in multiple ways, so much so that they are often referred to collectively as the neurohormonal system. Both systems communicate with target cells by chemical messengers.

2. A target cell is activated when the chemical message from the nervous system (neurotransmitter) or endocrine system (hormone) binds to receptors on or in the target cell. Receptor activation can cause one or more of the following: change in the electrical state of the cell, change in enzyme activity, change in secretory activity of the cell, muscle contraction, or protein synthesis.
3. The autonomic nervous system (ANS) carries information from the CNS to cardiac muscle, smooth muscle, and endocrine glands. The two branches of the ANS, the sympathetic and parasympathetic nervous system (PSNS), work in opposition to each other. The sympathetic nervous system (SNS) plays a critical role in directing the body's response to exercise, whereas the PSNS is particularly important during recovery from exercise.
4. The exercise response is mediated primarily through the SNS. Its primary functions during exercise are to enhance cardiorespiratory function, regulate blood flow and maintain blood pressure, maintain thermal balance, and increase fuel mobilization for the production of energy.
5. During exercise, several metabolic hormones (glucagon, insulin, growth hormone [GH], epinephrine [E], and norepinephrine [NE]) function together to mobilize fuel for the production of adenosine triphosphate (ATP) and to maintain blood glucose levels.
6. During exercise, several hormones help enhance cardiac function (E, NE), distribute blood to active tissue (E, NE), and maintain fluid and electrolyte balance (antidiuretic hormone [ADH], renin, and aldosterone).
7. Hormonal adaptations may result from aerobic exercise training, or individuals may become more sensitive to a lower level of hormone so that the same effect occurs following training even without a changed baseline hormonal level. The most common pattern is a blunted hormonal response to exercise following training (glucagon being an

important exception).

8. Resistance training is consistently associated with small but significant reductions in resting and exercise levels of GH, IGF, and testosterone.

Review Questions

1. Define homeostasis, and identify the role of the nervous system and hormonal system in maintaining homeostasis.
2. Why are the nervous system and the endocrine system often referred to collectively as the neuroendocrine system?
3. What role does the autonomic nervous system play in regulating the exercise response?
4. Describe receptor activation, and list five changes that may occur in a target cell as a result of receptor activation.
5. Discuss the primary role of exercise-induced hormonal changes relative to the cardiovascular system and the metabolic system.
6. Is adipose tissue an endocrine tissue? Explain. Is muscle an endocrine tissue? Explain.
7. Create a table that shows how the hormones involved in regulating metabolism and fluid balance respond to exercise.
8. How does the hormonal system adapt as a result of aerobic exercise training?
9. How does the hormonal system adapt as a result of resistance training?

For further review and study tools, visit [Lippincott Connect](#).

Literature Search

In this chapter, we discussed immunological changes resulting from exercise and exercise training. To explore this topic further,

do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search endocrine response to exercise training, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example:
 - ii. Hormonal response to resistance exercise
 - iii. Cortisol response to long-term strenuous exercise
 - iiii. Neural training adaptations as a result of high-volume endurance training programs in elderly women
 - iv. Effect of carbohydrate ingestion on endocrine response to endurance exercise
 - v. Continue your search for aspects of this topic that are of particular interest to you.

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22 The Immune System, Exercise, Training, and Illness



CHAPTER OUTLINE

Introduction

The Immune System

- Structure and Function of the Immune System

- Functional Organization of the Immune System

Exercise Immunology

The Immune Response to Exercise

- Medium-Duration (<45 Minutes), Moderate- to High-Intensity Aerobic Exercise

- Prolonged (1–3 Hours) Moderate- to High-Intensity Aerobic Exercise

Intense Interval Exercise

Cytokine Response to Exercise

Skeletal Muscle as an Endocrine Tissue That Releases
Regulatory Cytokines (Myokines)

Neuroendocrine Control of Immune Response to Exercise

Training Adaptation and Maladaptation

Hypothesized Causes and Mechanisms of Overtraining
Syndrome (OTS)

Markers and Monitoring of Training to Predict
Overtraining Syndrome (OTS)

Prevention and Treatment of Overtraining Syndrome (OTS)

Selected Interactions of Exercise and Immune Function

Exercise, the Immune System, and Upper Respiratory Tract
Infection

Exercise, the Immune System, and Cancer

Exercise, the Immune System, and AIDS

Summary

Review Questions

Literature Search

OBJECTIVES

After studying the chapter, you should be able to:

- Identify the primary cells of the innate and adaptive branches of the immune system, and indicate the mechanisms by which they lead to antigen destruction.
- Describe the sequence of events in inflammation.
- Differentiate between the immune responses to moderate aerobic exercise and exhaustive exercise.
- Describe the role of cytokines in regulating inflammation.
- Differentiate between overreaching and overtraining.
- Identify the causes of the overtraining syndrome, and describe actions designed to attempt to prevent it.
- Respond to this question: Does exercise increase or decrease the

likelihood of upper respiratory tract infection?

- Describe the interaction among exercise training, the immune system, upper respiratory tract infections (URTIs), cancer, and HIV/AIDS.

Introduction

Exercise training has many health benefits. Often, active individuals, perhaps yourself included, claim they feel better and are healthier than their sedentary friends. They claim these benefits not only just because they have altered their risk factors for major diseases but also because they have colds, flu, sore throats, and other common illnesses less often. On the other hand, one hears about Olympic and professional athletes competing despite an illness or, conversely, being unable to compete at all because of illness—not injury. Indeed, a common cause of poor physical performance at athletic events is acute respiratory infection. Acute respiratory conditions such as the common cold or influenza annually affect 90 out of 100 persons, causing substantial morbidity and economic cost. The relationship between exercise and immune function thus has important implications for public health and for athletes (Brolinson and Elliott, 2007; Gleeson and Pyne, 2016). In addition, many are interested in the role of exercise and exercise training in relation to diseases involving the immune system, such as cancer or HIV/AIDS. Does physical activity protect against certain cancers? Should individuals with disease be physically active?

Definitive answers to these questions are not yet possible. This chapter provides an overview of the functioning of the immune system, explores the acute immune response to exercise, considers adaptations in the immune system that occur as a result of exercise training, and, finally, addresses beneficial and detrimental influences of exercise on immune function and disease susceptibility.

The Immune System

Humans are constantly exposed to bacteria, viruses, and parasites capable of causing mild to serious disease. The fact that most of the time these foreign invaders do not overcome us is a testimony to the importance and efficiency of the body's defense mechanisms, comprised primarily of the immune system. The **immune system** is a complex and precisely ordered system of cells, hormones, and chemicals that regulate susceptibility to, severity of, and recovery from infection and illness (Nash, 1994). *Immunology* is the study of the body's physiological responses to destroy or neutralize foreign matter (Smith, 1995). The immune system operates with the nervous system and the endocrine system to maintain homeostasis. Although these systems operate independently, each with its own collection of highly specific cells and regulatory factors, they overlap considerably, and each system depends on the others for normal development and function (Pedersen et al., 2007). These three interrelated systems interact in complex, bidirectional, anatomical, and physiological ways (Miles, 2005) and are studied by the growing field known as psychoneuroimmunology or neuroimmunology.

Immune System A precisely ordered system of cells, hormones, and chemicals that regulate susceptibility to, severity of, and recovery from infection and illness.

In its organization and operation, the immune system parallels the nervous and endocrine systems. For example, all three systems consist of identifiable cells and chemical substances. All three systems react to stimuli; each has a network for communication within itself and with the other systems; and all three control and interact with other cells and organs. One major difference, however, is a gradient of mobility. The nervous system functions through fixed nerves and locally released neurotransmitters. The major endocrine glands are also fixed in place, but their hormones travel throughout the body, primarily via the bloodstream. The immune system consists primarily of free mobile cells that move within and outside the bloodstream, although some may be anatomically specific (Roitt et al., 1998).

As with the nervous and hormonal systems, one must

understand the parts and processes of the immune system to understand how it functions. **Figure 22.1** outlines the basics of the immune system. All immune responses involve recognizing a threat to the body and reacting to eradicate that threat while minimizing damage. The most common threat or stimulus to the immune system is an infectious microbe or pathogen; such microorganisms include bacteria, fungi, parasites, protozoa, and viruses. The SARS-CoV-2 virus that causes COVID-19 has become familiar to everyone. Some pathogens (all viruses, some bacteria, and small protozoan parasites) invade the body's cells and replicate there, while others (most bacteria and larger parasites) reside primarily in body fluids and extracellular spaces. The site of the infection and the specific pathogen determine how the immune system responds. The immune system can also be stimulated by cellular damage, such as muscle damage. In this case, the goal of the immune response, mediated largely through the process of inflammation, is to bring immune cells to the site of local tissue damage to clear cellular debris and set the stage for tissue repair. Recent data also suggest that immune mediators play a role in regulating metabolism.

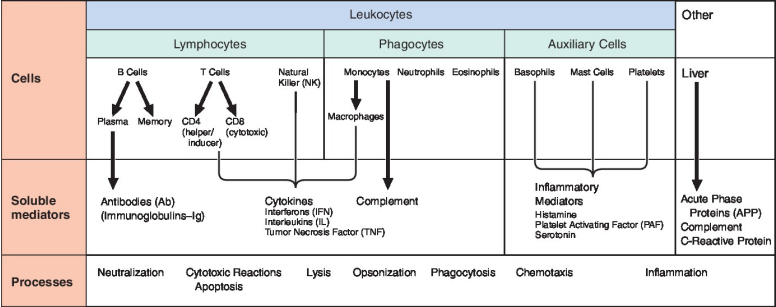


Figure 22.1 Components of the Immune System.

Source: Based on Roitt et al. (1998).

Structure and Function of the Immune System

Cells

The primary immune cells are *leukocytes* or white blood cells. Leukocytes are subdivided into *lymphocytes* and *phagocytes*, each of which has three major subdivisions (**Figure 22.1**).

B lymphocytes and T lymphocytes (B cells and T cells) specifically recognize individual pathogens. Any pathogen that can be specifically recognized by B cells and/or T cells is called an *antigen*. Both B cells and T cells are derived from bone marrow cells, but B cells mature in the bone marrow, whereas T cells mature in the thymus. Each B and T cell is genetically programmed to recognize only one particular antigen. Once an antigen is recognized, B cells proliferate into plasma cells that produce an antibody to act immediately and memory cells that will ultimately provide lasting immunity. Vaccinations confer immunity against diseases such as polio or measles by altering an antigen such that it becomes harmless but still brings about an antibody reaction.

Antigens recognized by T cells have been processed first in some way. Sensitized T cells enlarge and divide into two functionally separate classifications: CD8 cells, also called cytotoxic or killer cells, destroy infected cells directly. CD4 cells, which are helper or inducer cells, stimulate the action of cytotoxic cells and increase antibody production by B plasma cells. Through a variety of feedback mechanisms, both CD4 and CD8 cells can also act as suppressor T cells that inhibit cytotoxic T cells and antibody production, thus preventing excessive destruction (Roitt et al., 1998). Although they are classified as large granular lymphocytes, *natural killer (NK) cells* are different from other lymphocytes in that they act spontaneously against any target, apparently by recognizing surface changes on a variety of tumor cells and virally infected cells. As their name implies, these cells directly destroy infected host cells.

Phagocytes are leukocytes that bind to pathogenic microorganisms and antigens, internalize them, and then kill them. When they are in the bloodstream, mononuclear phagocytes are called *monocytes*; when they migrate into tissue, they evolve into *macrophages*. Macrophages often “process” an antigen and then present it to T lymphocytes. *Neutrophils* are the most abundant blood leukocytes. Large numbers are necessary because when neutrophils engulf and destroy foreign material,

they also die. *Eosinophils* are specialized to act against large extracellular parasites.

The role of the *auxiliary cells* is to release mediators to produce inflammation. *Mast cells* lie close to blood vessels; *basophils* and *platelets* circulate in the blood. The main purpose of inflammation is to attract leukocytes and their resultant soluble mediators to a site of infection or injury.

Soluble Mediators

Soluble mediators are intervening agents dissolved in a solution. These substances are produced primarily, but not exclusively, by immune cells that act either directly on the target pathogen or indirectly by signaling other immune cells to act or release additional mediators. Each immune cell produces and secretes only one particular set of mediators, although more than one cell type may produce the same classification of mediator. **Figure 22.1** shows representative soluble mediators but there are many others.

Antibodies produced by B plasma cells are also known as *immunoglobulins (Ig)*. Antibodies do not directly destroy antigens. Instead, they identify the invader by forming an antigen-antibody complex, and they then activate other soluble mediators and immune cells that perform the actual destruction. **Cytokines** are proteins or peptides that are released from immune cells and other tissues (notably skeletal muscle and adipose tissue, which are now recognized as endocrine glands). Cytokines are involved in communication between immune cells (especially lymphocytes and phagocytes) and other cells of the body. **Table 22.1** presents the major cytokines involved in the exercise immune response. Cytokines stimulate the proliferation of various immune cells and are important regulators of inflammation and the immune response. Among the principal types of cytokines are *interferons (IFNs)*, which are important in limiting the spread of certain viral infections; *interleukins (ILs)*, each of which acts on a specific group of immune cells to divide and differentiate; and *tumor necrosis factors (TNFs)*, which are particularly important in inflammation and cytotoxic reactions. Cytokines are thought to be important in the exercise immune response because certain ones (IL-1, IL-6, IFN γ , and TNF α) are proinflammatory factors

that probably play a role in coordinating the responses to muscle damage that may result from strenuous exercise. Other cytokines (IL-4, IL-10, and IL-6 in a dual role) are anti-inflammatory and suppress the activity of inflammatory cells, allowing normal structure and function to be restored. The release of these anti-inflammatory cytokines follows the proinflammatory response to vigorous physical activity (Moldoveanu et al., 2001). It is also becoming clear that cytokines released from muscle (myokines) play a role in regulating metabolism (Görgens et al., 2015).

Cytokines Proteins or peptides that are released from immune cells and other tissues (notably skeletal muscle and adipose tissue) and are involved in communication between immune cells (especially lymphocytes and phagocytes) and other cells of the body.

TABLE 22.1 Major Cytokines Involved in the Exercise Immune Response

| Cytokine | Released From | Primary Function |
|---|--|---|
| Interleukin: IL-1 α , IL-1 β | Macrophages | T-cell activation Macrophage function Proinflammatory function Fever |
| IL-2 | T cells | T-cell activation T-cell proliferation |
| IL-4 | T cells | Anti-inflammatory function |
| IL-6 | Activated T cells, macrophages, muscle | T- and B-cell growth Stimulates acute phase proteins Anti-inflammatory function Proinflammatory function |
| IL-10 | T cells, B cells, macrophages, mast cells | Anti-inflammatory and immunosuppressive functions |
| Interferon | | |
| IFN α | Leukocytes | Antiviral activity |
| IFN γ | T cells, NK cells | Stimulates cytotoxicity Macrophage activation Proinflammatory function |
| Tumor necrosis factor TNF α | Macrophages, NK cells | Stimulates cytotoxicity against tumor cells Proinflammatory function |

Complement is a group of serum proteins whose overall function is to control inflammation. Complement activation results in a series of reactions, including increased blood flow to

the site and increased permeability of capillaries to plasma molecules, which ultimately leads to destruction of the stimulating antigen (Mackinnon, 1999; Marieb and Hoehn, 2018).

Acute phase proteins (APPs) are produced in and secreted from the liver, as are some complement proteins (Mackinnon, 1992; Marieb and Hoehn, 2018). APPs are stimulated by proinflammatory cytokines and are so named because they increase rapidly during an infection. For example, chronically elevated levels of C-reactive protein reflect systemic infection and are associated with increased risk of type II diabetes and cardiovascular disease. APPs stimulate an increase in the number of leukocytes and play an important role in tissue repair following muscle cell damage.

Inflammatory mediators are molecules that, as the name implies, control the development of inflammation. For example, *histamine*, *platelet-activating factor (PAF)*, and *serotonin* (Figure 22.1) all bring about increased vascular permeability and smooth muscle contraction. Other inflammatory mediators not released from basophils, mast cells, or platelets include *fibrinopeptides* (the substance removed from fibrinogen during blood coagulation) and *fibrin breakdown products* plus *prostaglandins (PGE₂)*. These substances make the actions of other inflammatory mediators more effective.

Processes

The immune system can destroy or inactivate a pathogen in numerous ways. The defense mechanism(s) employed in response to an immune challenge vary according to the type of pathogen and its life cycle stage. In *cytotoxic reactions*, complete cells are killed, primarily by punching holes in their outer membranes. A target cell may be signaled to self-destruct, a process that is termed *apoptosis*. *Lysis* is a form of cytotoxic reaction in which a cell is killed by destruction of its cell membrane. *Neutralization* occurs when antibodies block the binding site on antigens so that they cannot bind to tissues and cause damage. *Opsonization* is the coating of the membrane of an antigen, making it easier for phagocytes to adhere to and engulf the antigen. *Phagocytosis* is the engulfing and digesting of a pathogen. Additionally, intracellular granules (small grain-like bodies) may be released in

a *respiratory oxidative burst*, which is a potent killer.

Functional Organization of the Immune System

As depicted in **Figure 22.2** functionally, the immune system can be divided into two separate but interrelated and overlapping branches: the innate (nonspecific) and the adaptive (specific) branches (Marieb and Hoehn, 2018; Smith, 1995). **Table 22.2** provides a glossary of the cells, soluble mediators, and processes that play an important role in the immune response.

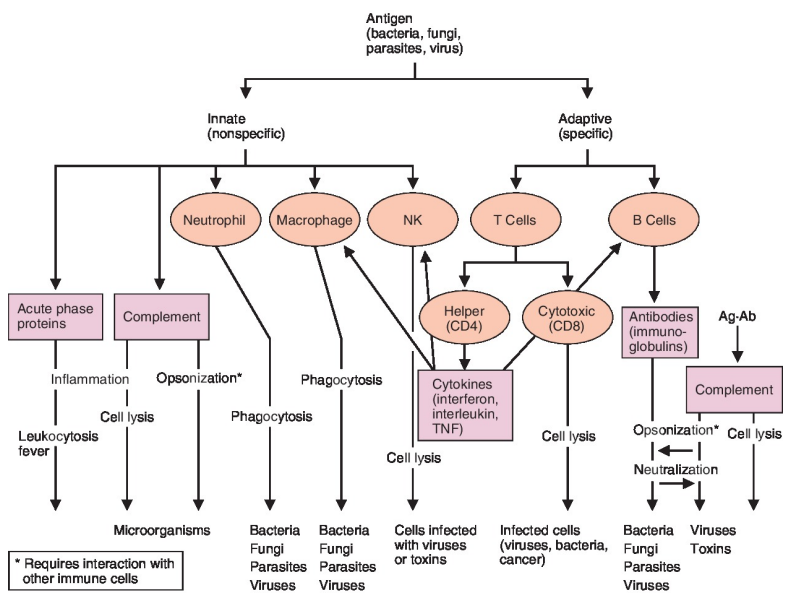


Figure 22.2 Overview of the Immune System.

The immune system is divided into the innate and adaptive branches and is composed of cells and chemical mediators that attack foreign antigens through several mechanisms. Immune cells are represented by *circles*, and soluble mediators (chemicals) are represented by *boxes*. The mechanisms of destruction are labeled within the *arrows* that point to the pathogen destroyed by the mechanism.

Sources: Modified from Marieb and Hoehn (2018); Smith

TABLE 22.2 Glossary of Cells, Molecules, and Processes Involved in the Immune Response

| Branch of Immune System | Processes | Cells | Molecules/Chemical Factors |
|-------------------------|---|---|---|
| Innate and adaptive | <p><i>Lysis</i> is the killing of a cell via destruction of the cell membrane.</p> <p><i>Phagocytosis</i> is the process of engulfing and digesting an antigen.</p> <p><i>Inflammation</i> is the process that prevents the spread of damaging agents, disposes of pathogens and cellular debris, and sets the stage for tissue repair.</p> | <p><i>Macrophages</i> are immune cells that (1) phagocytize pathogens, (2) present parts of the engulfed antigen on its plasma membrane to activate the T-cell response, and (3) secrete cytokines. They are important in both the innate and adaptive immune responses.</p> | <p><i>Antigens</i> are substances capable of provoking an immune response.</p> <p><i>Complement</i> is a group of ~20 plasma proteins. When activated, complement lyses microorganisms, enhances phagocytosis, and enhances the inflammatory response. Complement may be activated and function in either the innate or adaptive immune response.</p> <p><i>Cytokines</i> are chemicals released from sensitized T cells, NK cells, and activated macrophages to help regulate the immune response.</p> |
| Innate | | <p><i>Natural killer (NK)</i> cells are innate immune cells that destroy virus-infected and cancerous body cells by cell lysis.</p> <p><i>Neutrophils</i> are innate immune cells that phagocytize pathogens.</p> | <p><i>Acute phase proteins (APPs)</i> are blood proteins produced in the liver that function in the innate immune response. APPs are important in the response to infection and inflammation.</p> |
| Adaptive | <p><i>Neutralization</i> is a process that occurs when antibodies block the binding site on antigens so that they cannot bind to tissues and cause damage.</p> <p><i>Opsonization</i> is the process of coating the membrane of an antigen, making it easier for phagocytes to adhere to and engulf the antigen.</p> | <p><i>B cells</i> are lymphocytes that are part of the adaptive immune response and are responsible for the production of antibodies to a specific antigen.</p> <p><i>T cells</i> are lymphocytes that are responsible for cell-mediated responses of the adaptive immune system. Functionally, there are two classes of T cells: cytotoxic T cells (CD8 cells), which destroy virus-infected and cancer cells directly via cell lysis, and helper T cells (CD4 cells), regulatory cells that influence the activity of cytotoxic T cells, B cells, NK, and macrophages. Cytotoxic and helper T cells can act as suppressor cells once the infection is controlled.</p> | <p><i>Antibodies</i> are proteins produced by B cells to attack antigens.</p> |

The Innate Branch

The *innate branch* protects against foreign substances or cells without having to recognize them. It is nonselective and provides an initial line of defense against microbial invasion. The innate system consists of both a cellular component and physical barriers. Intact skin and mucous membranes are the major physical barriers. The primary cells of the innate system are the NK lymphocytes, neutrophils, and macrophages. Complement and acute phase proteins are the major soluble mediators. The innate system works through the processes of lysis, opsonization, and phagocytosis.

Inflammation

Central to the functioning of the innate branch of the immune system is the *inflammatory response* (**Figure 22.3**). The cells of the immune system are widely dispersed in the body. When a thermal or physical injury or an infection occurs, immune cells must concentrate at the site of the emergency. Muscle or tissue damage following strenuous exercise can be a potent signal for inflammation. As shown in **Figure 22.3**, immune cells converge through four processes:

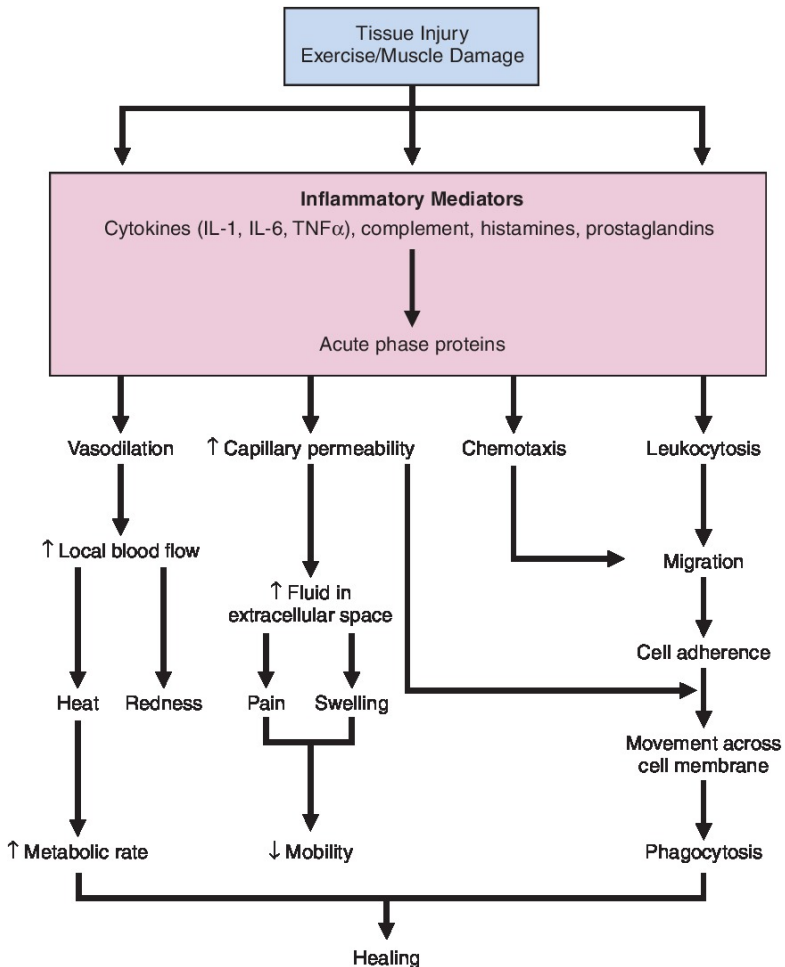


FIGURE 22.3 Sequence of Events in Inflammation.

Inflammation is characterized by heat, redness, pain, and swelling. These familiar responses occur because of the action of inflammatory mediators. Inflammation ensures immune cells are concentrated at the site of injury and that metabolic activity is increased to facilitate healing.

1. Vasodilation and an increased blood supply to the area
2. Increased capillary permeability
3. Chemotaxis
4. Leukocytosis (an increased number of leukocytes)

Increased blood flow and increased capillary permeability help bring leukocytes to the site of tissue damage or infection. *Chemotaxis* is the increased directional migration of immune cells. **Figure 22.4** depicts the sequence of events by which muscular damage can initiate chemotaxis and set the stage for repair of the damaged tissue. Chemotaxis occurs in response to the release of chemotactic factors, inflammatory mediators, and acute phase proteins. Neutrophils are the first cells to arrive at the injury site and do so within an hour. Neutrophils are followed by monocytes, which become macrophages once they enter the tissue. Macrophages dominate at the site of the injury 5–6 hours after the inflammatory response begins. On their arrival, these phagocytes adhere to the walls of the capillaries, a process facilitated by cell adhesion molecules (CAM). Eventually, the phagocytes push between the endothelial cells in the capillary and cross the membrane. They then squeeze through the cell membrane by a process called diapedesis and move to the actual site of the inflammation. Once there, both neutrophils and macrophages phagocytize the foreign antigens and/or cellular debris.

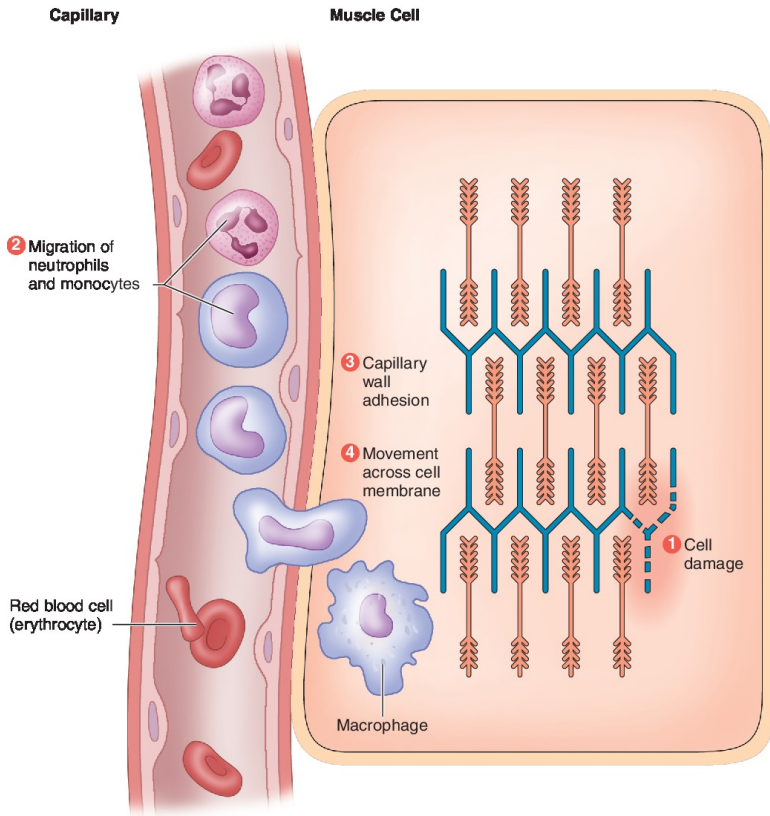


FIGURE 22.4 Events by Which Tissue Damage Leads to Increased Leukocytes in Tissues.

Muscle damage (1) produces a chemical signal that attracts neutrophils and monocytes to the area (2). The immune cells then adhere to the capillary wall (3) and move into the tissue area through the process of diapedesis (4).

Inflammation is characterized by redness, heat, swelling, and pain. The increased blood flow to the area resulting from vasodilation leads to the redness and heat. The increased capillary permeability allows fluid to seep into extracellular spaces, creating swelling that activates pain receptors. The pain and swelling can result in lack of mobility. Although immobility may be inconvenient, it forces the injured part to rest, which aids

in healing. Thus, inflammation can destroy foreign pathogens, prevent the spread of damaging agents, dispose of cellular debris due to tissue damage, and set the stage for tissue repair. Although part of the innate system, inflammation is also an important component of the adaptive immune response.

Complete the [Check Your Comprehension box 1](#) to assess your understanding of the inflammatory process.

CHECK YOUR COMPREHENSION 1

During soccer practice, Nick collided with another player, and both fell to the turf. After practice, Nick realized that he had scraped his knee on the turf (turf burn). The next day, Nick realized that the area around his turf burn was red, swollen, and painful. What caused these responses? How do these responses help promote tissue repair?

Check your answer in [Appendix C](#).

The Adaptive Branch

In contrast to the innate branch, in the *adaptive branch* of the immune system, immune cells recognize a foreign material and react specifically and selectively to destroy it. The adaptive branch is antigen specific, is systemic, and has memory. The adaptive branch includes humoral (from the Latin word humor, meaning “fluids”) and cell-mediated immunity. Antibodies produced from B cells provide *humoral immunity*. The antibodies circulate in the blood and lymph, where they bind to bacteria, toxins, and free viruses; inactivate them temporarily; and mark them for destruction by phagocytes or complements. *Cell-mediated immunity* is provided by T lymphocytes that directly attack and lyse cells infected by viruses, parasites, cancer cells, or grafts; release chemical mediators to enhance inflammation; and activate lymphocytes and macrophages.

The body’s ability to mount a specialized immune response involves specific proteins called *major histocompatibility complex (MHC)* that exist on the membranes of the body’s own cells and pathogens. MHCs are different in each individual except in

identical twins. With an organ transplant, therefore, the recipient's immune system must be suppressed because otherwise it would not recognize the donor MHC and would "attack" the transplanted cells, resulting in the "rejection" of the organ. As can be seen in **Figure 22.2**, the major cells of the adaptive branch are the B and T cells. The major soluble mediators are antibodies, cytokines, and complement. The processes of action include lysis, neutralization, opsonization, and, indirectly, phagocytosis. **Figure 22.5** shows how the innate and adaptive branches work together for a generalized immune response, emphasizing the role of macrophages in phagocytizing the foreign invader and activating the adaptive immune response by serving as antigen-presenting cells.

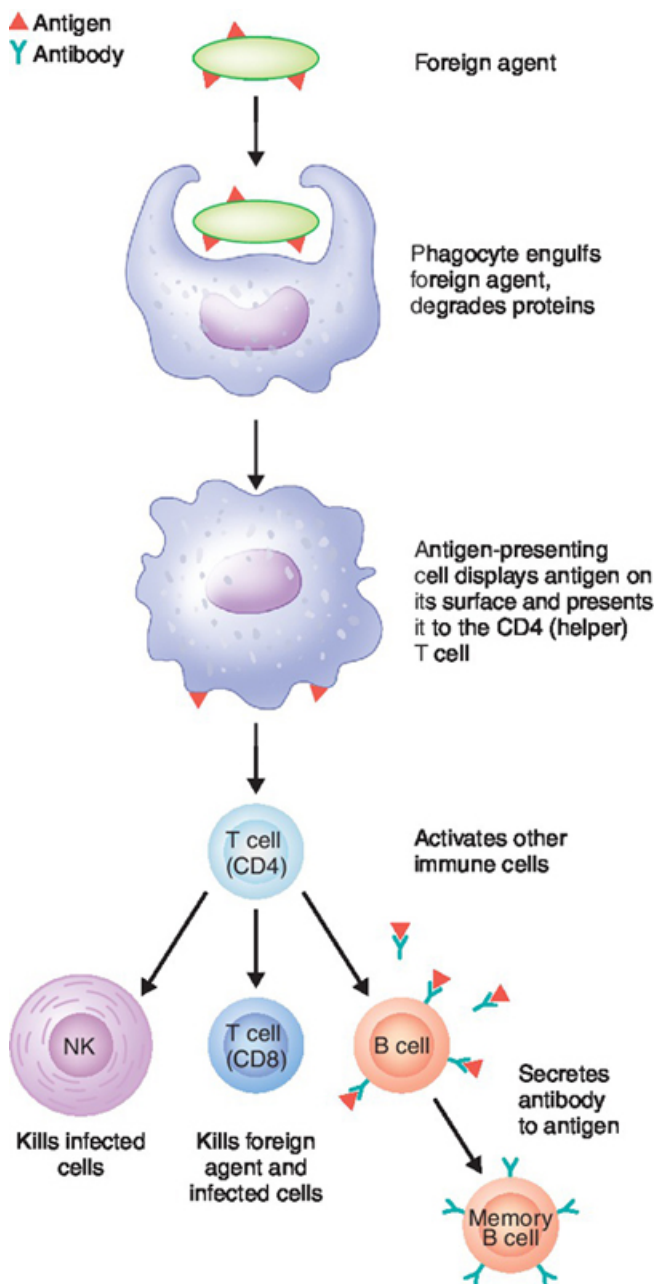


FIGURE 22.5 Central Role of Macrophages in the Innate and Adaptive Immune Responses.

Macrophages are phagocytes that ingest foreign agents as

part of the innate immune response. Once a macrophage has ingested a foreign invader, it activates the adaptive branch of the immune system by “presenting” portions of the foreign invader to adaptive cells.

The body’s many defenses against tissue injury and infection are amazing and, admittedly, can seem overwhelming. Remember that the functions of different structures and substances in the immune system overlap and are in some ways redundant. This ensures an effective immune response to most pathogens and is essential for health.

Exercise Immunology

There has been tremendous interest in the relationship between exercise and immune function and researchers have investigated this relationship for many decades. In fact, there is a recognized field of study, exercise immunology, that addresses a myriad of questions around this topic that can be categorized into four distinct time periods (Nieman and Wentz, 2019). Early studies (~1900–1979) in exercise immunology focused on acute changes in immune cell counts, whereas many studies conducted in the 1980s reported on the prevalence of acute respiratory illness following strenuous/exhaustive exercise. From 1990 to 2009, research in exercise immunology was focused on the inflammatory process, aging, and inflammation. Since 2010, there has been important work investigating how exercise affects protein and lipid metabolism, the microbiome of the gut, and clinical outcomes as a result of exercise training programs.

Figure 22.6 depicts a framework for studying exercise immunology and reveals some of the complexities involved in this endeavor (Woods et al., 1999). An acute bout of exercise may alter immune cell numbers or function directly or through any combination of the mechanisms listed below (Fleshner, 2005; Pedersen and Ullum, 1994; Steinacker et al., 2004; Woods et al., 1999):

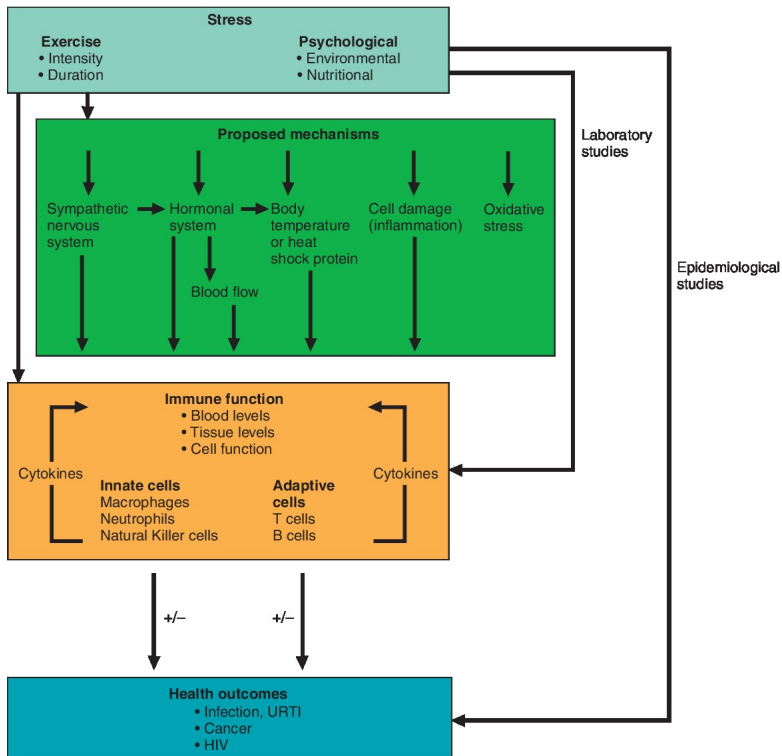


FIGURE 22.6 Relationships among Exercise, Immune Function, and Health.

Exercise is one of many factors that activate the immune system and it does so through multiple mechanisms. There are multiple immune responses to exercise, and they may have a positive or negative impact on health.

1. Directly stimulating immune function
2. Stimulating the sympathetic nervous system
3. Altering hormones (especially epinephrine, norepinephrine, cortisol, growth hormone, prolactin, and thyroxine)
4. Increasing body temperature
5. Inducing cell damage (and the release of acute phase proteins or heat shock proteins)
6. Increasing oxidative stress

The acute changes in immune factors that result from the stress of exercise are thought to lead to changes in health outcomes. There are a large number of health outcomes that may be affected by exercise or exercise training, including upper respiratory tract infections, cancer, and HIV.

The Immune Response to Exercise

The immune system responds to the acute stress of exercise. As with the effect of exercise on other body systems, the immune response to exercise depends largely on the intensity and duration of the exercise. Furthermore, because the physiological stress of exercise is additive with other stresses in one's life, including psychological, environmental, and nutritional stress, these factors must also be considered ([Steinacker et al., 2004](#); [Walsh and Oliver, 2016](#); [Walsh and Whitham, 2006](#)). Exercise appears to affect all of the cells of the immune system, but in different ways. Furthermore, each immune cell may perform several functions, and exercise may affect these functions differently.

Researchers investigating the effects of exercise on immune function typically have subjects engage in an acute bout of exercise in a laboratory setting. They may then report resulting changes in blood levels of an immune cell, tissue levels of an immune cell, or functioning of an immune cell. Such studies may compare the acute response of the immune system in individuals with different characteristics (age, fitness status, etc.).

Clearly, the type of exercise is a primary factor in determining the exercise response of the immune system. Most research on the effects of exercise on the immune system has investigated the effects of prolonged aerobic exercise, in large part because of the epidemiological data linking high-volume endurance training with a greater incidence of upper respiratory tract infection. Thus, the following sections deviate somewhat from the exercise categories used throughout this textbook being limited to two variations of long-term, moderate to heavy submaximal aerobic exercise and very short-term, high-intensity anaerobic exercise. Specifically, the immune response is detailed in relation to “medium-duration” (45 minutes), moderate- and high-intensity

aerobic exercise; “prolonged” (1–3 hours), moderate- and high-intensity exercise; and intense interval exercise.

Another important factor that must be considered when describing the effect of exercise on the immune system is the recovery period from exercise. A strenuous bout of exercise alters immune function for several hours or days. In fact, the suppression of several immune cells in the postexercise period may lead to a greater prevalence in upper respiratory tract infections frequently found among high-volume, endurance-trained athletes (Gleeson and Pyne, 2016; Nieman and Wentz, 2019; Tomasi et al., 1982).

Medium-Duration (<45 Minutes), Moderate- to High-Intensity Aerobic Exercise

Table 22.3 reports the percentage changes in immune cell numbers during or immediately after exercise and during recovery from medium-duration, moderate- and high-intensity exercise (Mackinnon, 1999). Exercise results in **leukocytosis**, an increased number of white blood cells. Leukocytosis is evident during most forms of physical activity and depends on the intensity and duration of the exercise. In most cases, the leukocytosis persists for at least 1–4 hours after exercise.

Leukocytosis An increase in circulating leukocytes (WBC).

TABLE 22.3 Exercise Response of Immune Cell Number to Medium-Duration (45 minutes), Moderate- and High-Intensity Aerobic Exercise

| Variable/Exercise Intensity | During or Immediately after Exercise | Recovery |
|-------------------------------|--------------------------------------|--|
| Total leukocytes | | |
| Moderate intensity | ↑ 0–40% | Unknown |
| High intensity | ↑ 50% | ↑ 50–100% 2 hr postexercise |
| Innate immune system | | |
| Neutrophils | | |
| Moderate intensity | ↑ 30–50% | Unknown |
| High intensity | ↑ 30–150% | Unknown up to 2 hr postexercise 25–100% 2–4 hr postexercise |
| Monocytes | | |
| Moderate intensity | No change | Unknown |
| High intensity | ↑ 0–20% | ↑ 0–50% 2 hr postexercise |
| Natural killer cells | | |
| Moderate intensity | ↑ 0–50% | Normal by 1 hr postexercise |
| High intensity | ↑ 100–200% | ↓ 40% 2–4 hr postexercise |
| Adaptive immune system | | |
| T cells | | |
| Moderate intensity | No change | Unknown |
| High intensity | ↑ 100% | ↓ 30% 1–2 hr postexercise |
| B cells | | |
| Moderate intensity | No change | Unknown |
| High intensity | No change | ↓ 0–25% 1–2 hr postexercise |
| Serum Ig | | |
| Moderate intensity | No change | No change |
| High intensity | No change | No change |
| Salivary IgA | | |
| Moderate intensity | No change | No change |
| High intensity | No change | No change |

Source: Based on data from [Mackinnon \(1999\)](#).

Neutrophils increase in number as a result of endurance exercise. The increases are greater after high-intensity exercise than moderate exercise. The increase in neutrophils is evident for 2–4 hours after exercise and is likely evident during moderate exercise, although research data are lacking. It appears that medium-duration aerobic exercise also enhances neutrophil function. Both the phagocytic activity and the oxidative burst activity of neutrophils are reportedly enhanced ([Woods et al., 1999](#)).

Circulating levels of monocytes are not altered by medium-duration moderate exercise but increase modestly during high-intensity exercise. This elevation persists for at least 2 hours after exercise. Recall that when monocytes leave the bloodstream, they are transformed to macrophages, which perform several roles in the immune response. Research suggests that macrophage functions are enhanced following medium-duration exercise. These functions include phagocytic activity, oxidative burst activity, and antitumor activity ([Nieman, 1997a](#); [Woods and Davis, 1994](#); [Woods et al., 1993, 1997, 1999](#)).

Circulating levels of natural killer (NK) cells increase during medium-duration bouts of both moderate- and high-intensity exercise, with the more intense exercise causing a larger increase

in NK cell numbers. Of greater importance, however, are the different responses during recovery. Following moderate-intensity exercise, blood levels of NK cells return to normal within 1 hour. Following high-intensity exercise, in contrast, NK cell levels drop approximately 40% below normal levels and remain depressed for 2–4 hours. Natural killer cell activity (NKCA) mimics the changes in NK cell numbers (Mackinnon, 1999; Woods et al., 1999). **Figure 22.7** depicts the changes in NKCA after 45 minutes

of intense (80% $\dot{V}O_2 \text{ max}$), 2.5–3 hours of running ($\sim 76\%$ $\dot{V}O_2 \text{ max}$), and during recovery. For now, concentrate on the 45-minute bout of exercise. Notice the large increase in NKCA immediately after exercise, followed by a reduction in NKCA below preexercise levels and a subsequent return to preexercise values by the 4th hour (Nieman et al., 1990, 1995a, 1995b; Woods et al., 1999).

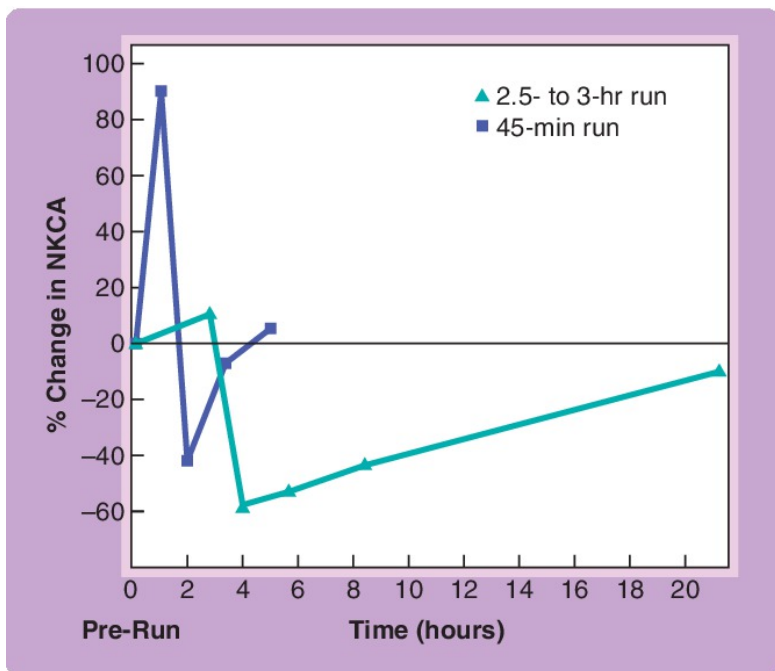




FIGURE 22.7 Natural Killer Cell Activity (NKCA) during and following Exercise of Different Durations.

NKCA is greater after 45 minutes of running ($\sim 80\%$

$\dot{V}O_{2\max}$) than after 2.5–3 hours of running ($\sim 76\%$

$\dot{V}O_{2\max}$). However, NKCA is suppressed longer after prolonged running (2.5–3 hours) than after 45 minutes of running. **Sources:** Reprinted with permission from Woods, J. A., J. M. Davis, J. A. Smith, & D. C. Nieman: Exercise and cellular innate immune function. *Medicine & Science in Sports & Exercise*. 31(1):57–66 (1999). Copyright ©1999 The American College of Sports Medicine. Based on data from Nieman et al. (1990); Nieman et al. (1995a); Nieman et al. (1995b).

T-cell numbers do not change during moderate-intensity, medium-duration exercise but increase markedly as a result of high-intensity exercise. During recovery from intense exercise, a suppression in T-cell numbers is evident for 1–2 hours of recovery. As with NK cells, this suppression in T cells after exercise may make one more susceptible to infection.

B-cell numbers do not appear to change immediately after medium-duration bouts of moderate- or high-intensity dynamic exercise. However, B-cell numbers decrease 1–2 hours following intense exercise. Daughter cells of the B cells, plasma cells, produce antibodies (immunoglobulins) that lead to destruction of antigens. Serum or salivary levels of immunoglobulins apparently do not change following aerobic exercise lasting less than 45 minutes (Mackinnon, 1999).

The net result of the changes associated with moderate to intense aerobic activity of less than 45 minutes (or 60 minutes based on some research) is an increased activity of antipathogen activity of macrophages, and increased circulation of

immunoglobulins, anti-inflammatory cytokines, neutrophils, NK cells, T cells, and B cells that play key roles in immune defense. It is thought that over time, these acute changes lead to enhanced immunosurveillance and lower inflammation (Nieman and Wentz, 2019).

Prolonged (1–3 Hours) Moderate- to High-Intensity Aerobic Exercise

Table 22.4 reports the changes in immune cell numbers during or immediately after exercise and during recovery from prolonged moderate- and high-intensity aerobic exercise (Mackinnon, 1999). Prolonged high-intensity exercise results in the greatest leukocytosis immediately after exercise and in recovery (Da Silva Neves et al., 2015).

TABLE 22.4 Exercise Response of Immune Cell Number to Prolonged (1–3 hr) Moderate- and High-Intensity Aerobic Exercise

| Variable/Exercise Intensity | During or Immediately after Exercise | Recovery |
|-------------------------------|--------------------------------------|--------------------------------|
| Total leukocytes | | |
| Moderate intensity | ↑ 25–50% | ↑ 25–65% 2 hr postexercise |
| High intensity | ↑ 200–300% | ↑ 200–300% 2–6 hr postexercise |
| Innate immune system | | |
| Neutrophils | | |
| Moderate intensity | ↑ 20–50% | ↑ 50–150% 2 hr postexercise |
| High intensity | ↑ 300% | ↑ 300–400% 2–6 hr postexercise |
| Monocytes | | |
| Moderate intensity | No change | No change |
| High intensity | ↑ 50–100% | ↑ 50–100% 2–3 hr postexercise |
| Natural killer cells | | |
| Moderate intensity | ↑ 70–100% | ↓ 0–50% 1–2 hr postexercise |
| High intensity | ↑ 100–200% | ↓ 30–60% 1–2 hr postexercise |
| Adaptive immune system | | |
| T cells | | |
| Moderate intensity | ↑ 20–30% | ↓ 20% 2 hr postexercise |
| High intensity | ↑ 30–60% | ↓ 30–40% 1–6 hr postexercise |
| B cells | | |
| Moderate intensity | No change | No change |
| High intensity | No change | No change |
| Serum Ig | | |
| Moderate intensity | No change | No change |
| High intensity | No change | No change |
| Salivary IgA | | |
| Moderate intensity | No change | No change |
| High intensity | ↓ 20–60% | ↓ 20–60% 1 hr postexercise |

Source: Based on data from [Mackinnon \(1999\)](#).

Neutrophils increase in number as a result of aerobic exercise. The increases are greater following high-intensity exercise. Prolonged high-intensity exercise causes a significantly greater increase in neutrophils than shorter exercise bouts of the same intensity. Large increases in neutrophil numbers are evident for 2–6 hours after high-intensity exercise. Despite the increase in neutrophil numbers after prolonged moderate- and high-intensity exercise, there is evidence that neutrophil function may not respond uniformly (Mackinnon, 1999). Prolonged moderate-exercise is associated with enhanced neutrophil function (phagocytic activity, oxidative burst activity, and antimicrobial activity). In contrast, prolonged high-intensity exercise is associated with a suppression of neutrophil function (Nieman, 1997a; Nieman and Wentz, 2019; Woods et al., 1999).

Circulating levels of monocytes are not altered by moderate-intensity aerobic exercise but increase substantially during intense aerobic exercise. The elevation persists for at least 2 hours after exercise. Furthermore, the increase in monocytes after prolonged high-intensity exercise is considerably greater than the increase in monocytes after medium-duration, high-intensity exercise, as is the postexercise elevation. Research suggests that macrophage function is enhanced following prolonged moderate-intensity aerobic exercise. However, high-intensity exercise is associated with impaired macrophage function (Nieman and Wentz, 2019; Woods et al., 1997, 1999).

Circulating levels of NK cells increase considerably following prolonged bouts of both moderate- and high-intensity aerobic exercise, with the intense exercise causing a larger increase in NK cell numbers. After prolonged moderate- or high-intensity exercise, NK cell numbers are reduced, more so after high-intensity exercise. As with medium-duration exercise, NKCA mimics the changes in NK cell numbers. Look again at **Figure 22.7**, this time paying attention to the change in NKCA after 2.5–

3 hours of intense ($\dot{V}O_{2\max}$ 76%) running and during recovery (Nieman et al., 1990, 1995a, 1995b; Woods et al., 1999). Notice the smaller increase in NKCA immediately postexercise for the prolonged run (2.5–3 hours) compared to the shorter run (45 minutes). However, during recovery from the prolonged run, NKCA is severe and persistently reduced below

preexercise levels. NKCA remained suppressed in excess of 20 hours following the prolonged, high-intensity run.

T-cell numbers increase after prolonged moderate-intensity and high-intensity exercise. During recovery from prolonged exercise (both moderate and high intensity), T-cell numbers are suppressed.

B-cell numbers do not appear to change immediately after prolonged bouts of moderate- or high-intensity exercise or during recovery from exercise. However, salivary levels of immunoglobulin A (IgA) are depressed after prolonged high-intensity exercise (Mackinnon, 1999; Novas et al., 2003; Tomasi et al., 1982). The depression that is seen in cell number and function is transient with values typically returning to baseline values within 24 hours. However, inadequate recovery periods may not allow for fully immune recovery and may play a role in overtraining syndrome (see discussion later in this chapter).

Intense Interval Exercise

Table 22.5 reports the changes in immune cell numbers during or immediately after exercise and during recovery from intense interval exercise (Mackinnon, 1999). Again, exercise results in leukocytosis for at least 2–6 hours after exercise.

TABLE 22.5 Exercise Response of Immune Cell Number to Intense Interval Exercise

| Variable | During Exercise or Immediately after Exercise | Recovery |
|-----------------------------------|---|----------------------------------|
| Total leukocyte number | ↑ 65–80% | ↑ 75% 2–6 hr postexercise |
| Innate immune system | | |
| Neutrophils | ↑ 25% | ↑ 60–100% 2–6 hr postexercise |
| Monocytes | ↑ 40–50% | ↑ 15–60% 2–6 hr postexercise |
| Natural killer cells | ↑ 100–200% | Normal by 1–2 hr postexercise |
| Adaptive immune system | | |
| T cells | ↑ 60–100% | ↓ 30–40% 1–2 hr postexercise |
| B cells | ↑ 0–7% | Normal by 1–6 hr postexercise |

Source: Based on data from [Mackinnon \(1999\)](#).

Neutrophils increase in number as a result of intense interval exercise and remain elevated for 2–6 hours afterward. Similarly, the circulating levels of monocytes increase following intense interval exercise and remain elevated for 2–6 hours. NK cell numbers increase markedly following intense interval exercise but return to preexercise levels within 2 hours.

T-cell numbers increase following intense interval exercise. During recovery, T-cell numbers are suppressed for 1–2 hours. B-cell numbers increase immediately after intense interval exercise but return to normal levels within 1–6 hours.

Cytokine Response to Exercise

Exercise is a potent activator of the immune system leading to

changes in many cytokine concentrations. Many of the cytokines are released from adipose tissue (adipokines) and muscle tissue (myokines). **Figure 22.8** is a schematic graph of the relative magnitude of changes in plasma cytokines during and after strenuous exercise. The most notable and best-characterized cytokine response to strenuous exercise is a large, rapid increase in IL-6. Plasma IL-6 concentrations increase exponentially, up to 100-fold, as exercise continues. This increase is related to exercise intensity, duration, muscle mass, and the individual's aerobic capacity (Gokhale et al., 2007; Miles, 2005; Raschke and Eckel, 2013; Steinacker and Lormes, 2004). Importantly, the increase in IL-6 does not require muscle damage, suggesting that cytokines likely play important regulatory roles in addition to mediating inflammation. Plasma levels of IL-6 typically return to baseline levels within a few hours after exercise if muscle damage has not occurred (Miles, 2005; Raschke and Eckel, 2013). Following the increase in IL-6, the concentration in other cytokines begins to increase. IL-1ra and IL-10 increase after IL-6, suggesting that IL-6 plays an important role in regulating the release of these anti-inflammatory cytokines. Importantly, the proinflammatory cytokines, TNF α and IL-1 β , which increase during infection, do not increase after exercise in the absence of muscle damage (Petersen and Pedersen, 2005). Taken together, these data suggest that while exercise may evoke both inflammatory and anti-inflammatory cytokines, the anti-inflammatory cytokine response predominates. It has been reported that physically active individuals have less systemic inflammation than their sedentary counterparts and cytokines are a potential mechanism for this observation (Hayashino et al., 2014).

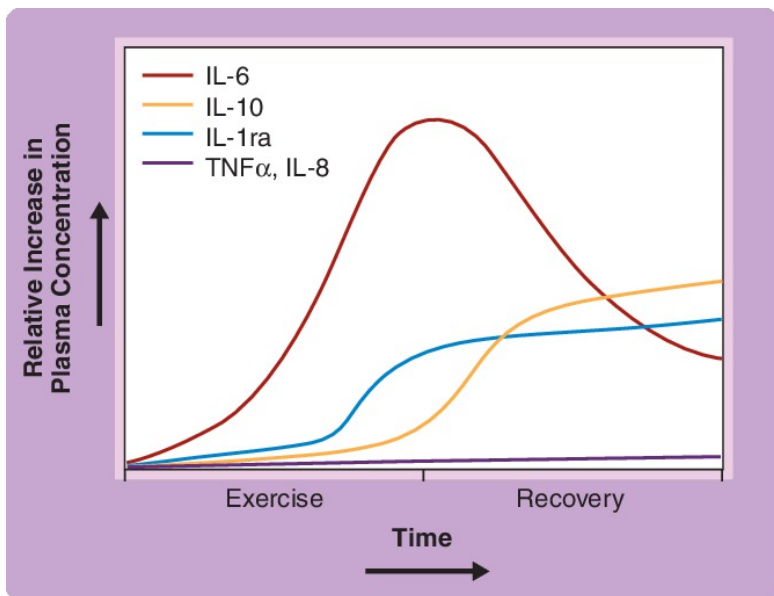


FIGURE 22.8 Cytokine Response to Strenuous Exercise.

Sources: Modified from Febbraio, M. A., & B. K. Pedersen: Muscle-derived interleukin-6: Mechanisms for activation and possible biological roles. *The Federation of American Societies for Experimental Biology*. 16(11):1335–1347 (2002). Copyright © FASEB. Reprinted by permission of John Wiley & Sons, Inc.; Petersen, A. M. W., & B. K. Pedersen: The anti-inflammatory effect of exercise. *Journal of Applied Physiology*. 98:1154–1162 (2005).

Skeletal Muscle as an Endocrine Tissue That Releases Regulatory Cytokines (Myokines)

An acute bout of exercise is known to have a profound effect on the immune system. As discussed earlier, much recent research has focused on the effect of exercise on cytokines and their role in regulating the immune response. Cytokines are released from contracting muscle as well as from immune cells (Pedersen et al., 2007; Steensberg et al., 2000). This discovery has important

implications because it provides a mechanistic link between muscle contraction and the regulation of the immune response to exercise. Furthermore, cytokines released from exercising skeletal muscles also play an important role in metabolism. Cytokines released from muscle tissue are termed myokines—parallel to the use of the term “adipokines” for cytokines released specifically from adipose tissue.

Interleukin-6 (IL-6) is the most abundant myokine released by muscle tissue. IL-6 is usually identified as a proinflammatory cytokine; however, it also plays an anti-inflammatory role (Petersen and Pedersen, 2005). During contraction, skeletal muscle fibers produce and release large amounts of IL-6 (up to a 100-fold increase). In fact, skeletal muscle production of IL-6 can account entirely for the exercise-induced increase in IL-6 (Steensberg et al., 2000). **Figure 22.9** is a schematic of how IL-6 may function as a myokine. IL-6 released from exercising skeletal muscle exerts its immunoregulatory role by its anti-inflammatory and proinflammatory actions. Anti-inflammatory effects include an increase in anti-inflammatory cytokines (IL-1ra, IL-10) and suppression of the proinflammatory cytokine TNF α . IL-6 also exerts a neuroendocrine influence by stimulating the hypothalamus and leading to the subsequent release of GH (from the anterior pituitary) and epinephrine and cortisol (from the adrenal glands). IL-6 also plays a metabolic role by increasing lipolysis and fat oxidation and increasing glucose uptake in skeletal muscle. Thus, IL-6 appears to play a central role in regulating a coordinated response to exercise that facilitates both the hormonal and immune responses necessary to balance inflammation and provide for the metabolic needs of working muscle (Pedersen et al., 2007).

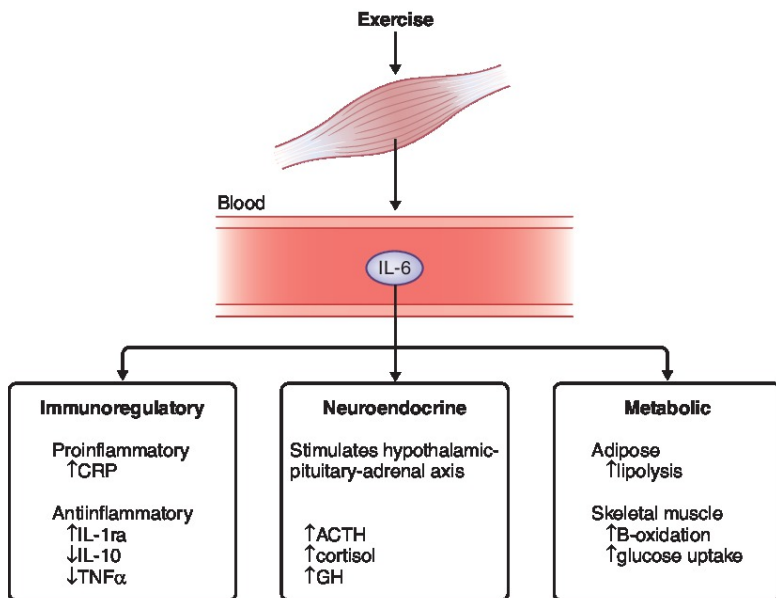


FIGURE 22.9 Regulatory Roles of Muscle-Derived Interleukin-6 (IL-6).

Interleukin-6 plays an important regulatory role in the immune, neuroendocrine, and metabolic responses to exercise.

CHECK YOUR COMPREHENSION 2

What tissue is primarily responsible for the increase in circulating IL-6 following exercise?

What are some of the functions of IL-6?

Check your answer in [Appendix C](#).

Complete the [Check Your Comprehension 2](#) queries to determine your understanding of IL-6.

While extensive research efforts have provided information about plasma levels of the proinflammatory and anti-inflammatory cytokines (see [Figure 22.8](#)) after strenuous exercise, these data must be interpreted cautiously. Cytokines

have different rates of production and clearance, and their localization and use are important for assessing their contribution to overall immune function (Mastro and Bonneau, 2005). It is, therefore, fair to say that while cytokines play an important role in neurohormonal regulation, hormonal regulation, and immune response to exercise, many questions still remain regarding the precise role of individual cytokines.

In summary, an acute bout of exercise has profound effects on many immune variables, including cell number and function and circulating levels of cytokines. In general, moderate exercise appears to cause transient increases in immune function, with immune cell number and function returning to baseline within a couple of hours. Strenuous exercise, however, causes greater disruption of the immune system and a decrease in some cells and cellular function (especially NK cells and T cells) that may last for many hours into recovery. This reduction in cell number and function is a theoretical link to the often described higher incidence of self-reported upper respiratory tract infections in high-volume, endurance-trained individuals.

Neuroendocrine Control of Immune Response to Exercise

As discussed in the previous chapter, exercise is a stressor that stimulates both neuroendocrine and immune responses. **Figure 22.10** builds on **Figure 21.2** and extends the basic stress response to depict the neuroendocrine influence on immune function. This figure emphasizes that the body's response to exercise stress is a system-wide effort that is largely coordinated by the integration of the immune system with the neuroendocrine system (Fragala et al., 2011). Much of the influence on the immune system depends on the hypothalamic-pituitary-adrenal axis, which results in the release of cortisol from the adrenal cortex, and the sympathetic-adrenal medulla axis, which results in the release of the catecholamines from the adrenal medulla. Sympathetic nerve stimulation also directly innervates lymphoid organs. Exercise also causes the release of cytokines from skeletal muscle and immune cells, which help regulate the immune response.

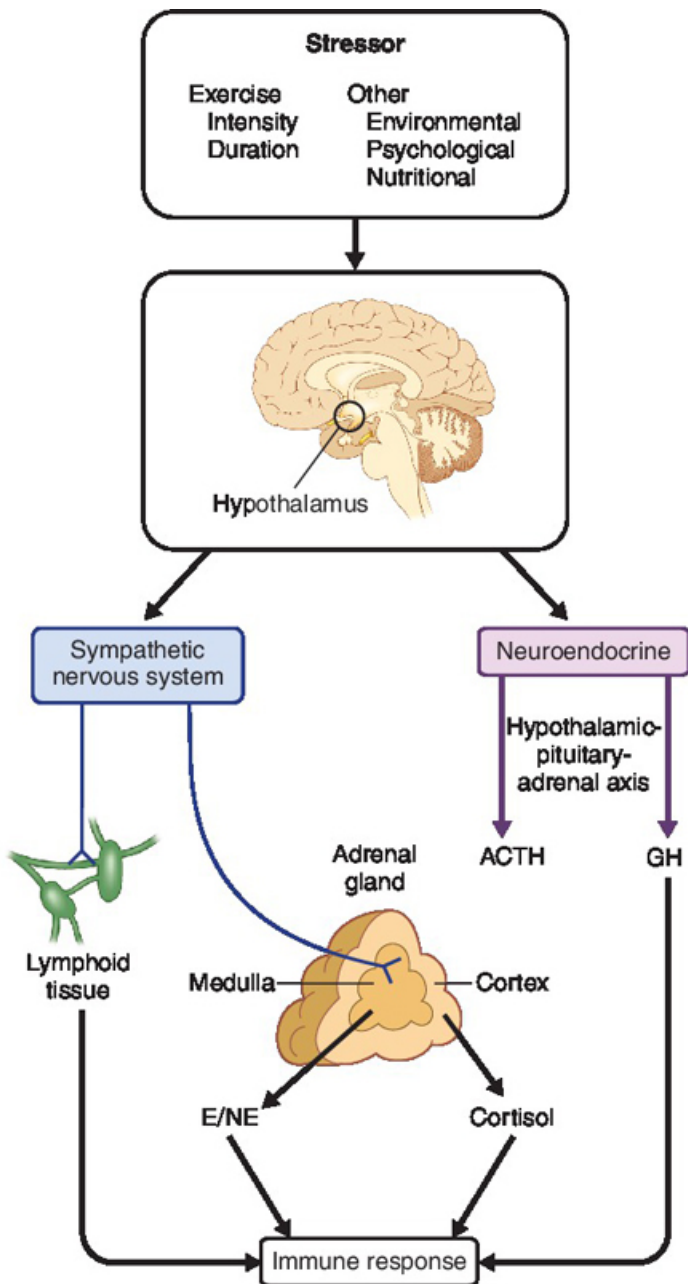


FIGURE 22.10 Neuroendocrine Regulation of Immune Function.

The immune response to exercise is mediated largely

through the nervous and endocrine systems.

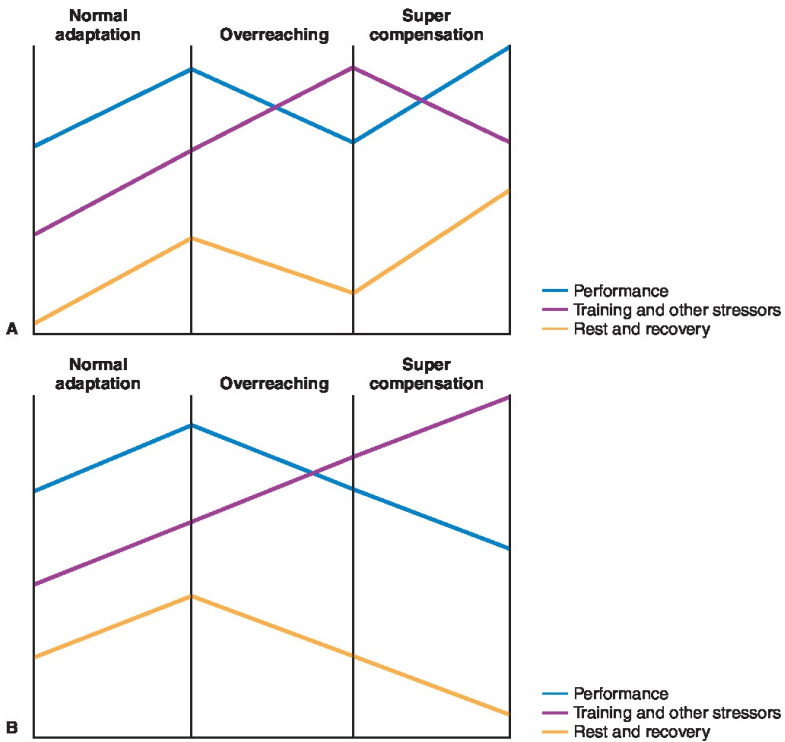


FIGURE 22.11 Exercise Training Adaptations (A) and Maladaptation (B).

Sources: Fry et al. (1992); Kreider et al. (1998); Kuipers (1998).

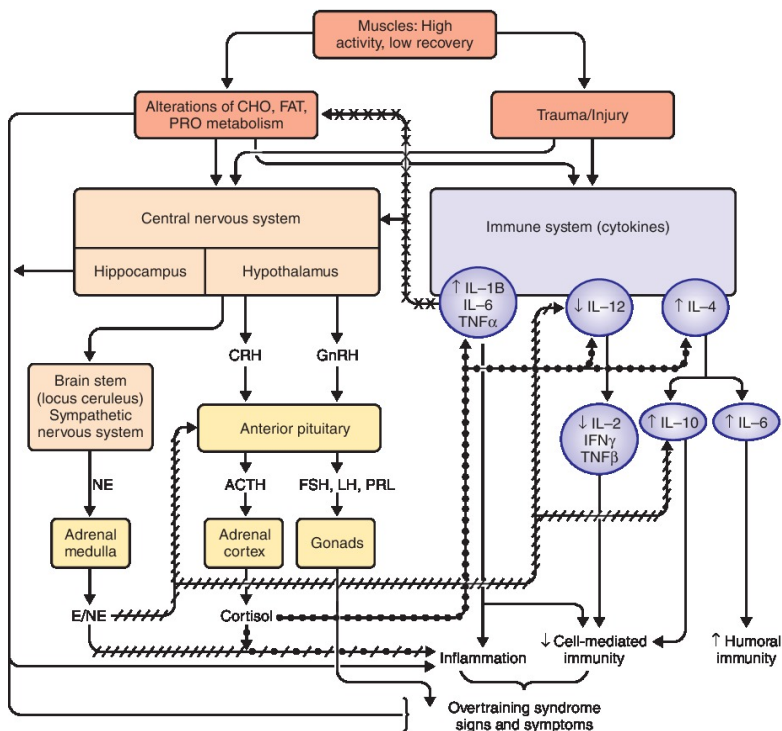


FIGURE 22.12 Hypothesized Mechanism of Overtraining Syndrome.

A high-volume training load performed at high intensity, applied in a monotonous manner, and without sufficient rest and recovery that alters metabolic processes and leads to an accumulation of muscle trauma appears to be the primary predisposing factor to the development of the overtraining syndrome. Alterations of carbohydrate (CHO), lipid (FAT), and/or protein (PRO) metabolism (represented on the left side of this flow chart) and microtrauma or tissue injury in muscles (presented on the right side of the flow chart) can individually or collectively impact both the central nervous system and the immune system. Full details describing this figure are included in the text. **Sources:** Budgett (1998); Chrousos and Gold (1992); Fry et al. (1992); Gastmann and Lehmann (1998); Keizer (1998); Kreider et al. (1998); Moldoveanu et al. (2001); Petibois et

al. (2003); Robson (2003); Smith (2000, 2003a, 2003b); Steinacker et al. (2004); Snyder (1998).

As stated previously, moderate aerobic exercise appears to enhance immune function, and the cells of the immune system return to resting levels soon after exercise. Severe, exhaustive aerobic exercise, in contrast, causes an enhanced immune function followed by a suppression of cell activity. These different responses appear to depend largely on the hormones epinephrine and cortisol. During exercise at greater than 60% $\dot{V}O_2$ max, epinephrine and cortisol levels in the blood begin to increase rapidly, reaching their highest level after maximal exercise. Epinephrine is associated with a substantial increase in the number of lymphocytes in the blood. Cortisol causes an increased number of neutrophils but a decreased number of lymphocytes. Immediately after exercise, the blood concentrations of epinephrine fall rapidly to preexercise levels, whereas cortisol levels remain elevated for 2 hours or more (see [Chapter 21](#)). Researchers, therefore, hypothesize that after intense exercise, epinephrine is responsible for the increase in circulating lymphocytes, and the longer-acting cortisol is responsible for the prolonged increase in neutrophils and the decrease in lymphocytes ([Nieman, 1994](#)).

The effect of exercise on specific immune cells or cytokines is interesting to many exercise scientists, but most individuals are more concerned about how exercise relates to health outcomes, such as the rate and severity of an infection. Since the 1980s, there has been research linking prolonged, intensive exercise to an increased risk of upper respiratory tract infections (URTIs). Some researchers conclude that the transient immune dysfunction after prolonged heavy exercise is associated with an increased risk of URTI ([Nieman and Wentz, 2019](#)). However, other researchers interpret the research findings to suggest that intensive exercise leads to a transient and time-dependent redistribution of immune cells to the peripheral tissue that results in a heightened state of immune surveillance and immune regulation, not to immune suppression ([Campbell and Turner, 2018](#)). These competing views are discussed later in the chapter in the section on Selected Interactions of Exercise and Immune

Function and will surely be the foundation for a great deal of future research.

Training Adaptation and Maladaptation

The goal of exercise training is to lead to improved physical performance and to improved health. Indeed, the vast majority of evidence presented in this book indicates that exercise training leads to positive adaptations. This is generally true for the immune system as well. However, the relationship of training to athletic performance involves a continuum that can be depicted as an inverted U (**Figure 1.10**) (Fry et al., 1991; Kuipers, 1998; Rowbottom et al., 1998). As described in Chapter 1, the goal of optimal periodized training is the attainment of peak fitness and/or performance. However, with too great or improperly applied training overload, maladaptation is possible. The over training syndrome (OTS) is thought to be caused by a combination of excessive training and insufficient recovery, and its pathology is complex, multifactorial, and still unclear (Cadelegiani, 2020).

Table 22.6 summarizes training adaptations in the immune system of moderately trained and highly trained or overtrained individuals. The resting immune system of moderately trained aerobic athletes is usually within normal parameters, although neutrophil function and serum immunoglobulins may be relatively low (Mackinnon, 2000; Nieman, 1994; Shephard et al., 1995). In general, moderate training appears to enhance the immune system, especially natural killer cell count and activity (Mackinnon, 2000; Matthews et al., 2002; Shephard et al., 1995; Woods et al., 1999). In contrast, overtraining is associated with a suppression of several immune variables. Most notable is a decrease of leukocyte numbers, a decrease in neutrophil function, a decrease in natural killer cell activity, and a reduction in lymphocytes (B cells, T cells, and NK cells) (Mackinnon, 1999, 2000; Nieman, 1997a; Woods et al., 1999). As seen in **Table 22.6**, training-induced adaptations also occur in several cytokines, acute phase proteins, and antibodies (immunoglobulins). Intense training or overtraining is associated with a reduction in acute phase proteins, serum Ig, salivary IgA,

and complement (Mackinnon, 1999; Nieman, 1997a; Papacosta and Nassis, 2011; Woods et al., 1999).

TABLE 22.6 Effects of Moderate and Intense Training/Overtraining on Immune Function and Resistance to Illness

| | Moderate Training | Intense Training/Overtraining |
|------------------------------|--|--|
| Cells | | |
| Leukocytes | No change in resting number | ↓ Resting number in circulation of athletes after intense exercise |
| Neutrophils | No change in resting number | No change in resting number (or decrease) ↓ Activation at rest and after exercise |
| NK cells | No change in resting number ↑ NKCA at rest | ↓ Resting number in circulation ↓ NKCA at rest |
| T cells | No change | No change or decrease |
| B cells | No change in resting number | No change in resting number |
| Chemical mediators | | |
| Cytokines | No change in resting concentration ↑ Resting IL-1 in plasma of athletes compared with nonathletes | No change in resting concentrations ↑ Resting IL-1 in plasma of athletes compared with nonathletes |
| APP | ↓ Acute phase protein release after exercise | ↓ Acute phase protein release after exercise |
| Antibodies (Ig) | No change in serum and secretory Ig levels ↑ Specific antibody response | ↓ Serum Ig levels in athletes after intensified training ↓ Salivary IgA levels in athletes after intensified training |
| Resistance to Illness | | |
| | ↑ Survival rate following viral infection ↑ Survival rate following bacterial infection ↓ Incidence of URTI ↓ Incidence of cancer | ↑ Incidence of URTI ↑ Rate of paralysis following polio infection |

Sources: Based on data from Mackinnon (1999); Mackinnon (2000); Nieman (1997a, 1997b).

One of the important research challenges in this area is to define more clearly what constitutes “moderate” training and “strenuous” training. Furthermore, there is a need to understand more fully whether strenuous training in itself results in immune suppression or whether immune suppression occurs only with more severe overtraining. Studies have documented decreases in some markers of immunity of 15–25% following heavy training programs, but whether or not that has functional consequences for health or illness remains unknown (Campbell and Turner, 2018; Walsh and Oliver, 2016). While many chapters in this text focus on the beneficial adaptations that result from exercise training, the following sections address the serious condition of maladaptation.

The first step toward maladaptation may be **overreaching (OR)**, a short-term decrement in performance capacity that

generally lasts only a few days to 2 weeks and from which the individual easily recovers. Overreaching can result from planned shock microcycles (see [Chapter 1](#)) or inadvertently from too much stress and too little planned recovery ([Carfagno and Hendrix, 2014](#); [Fry and Kraemer, 1997](#); [Fry et al., 1991](#); [Kuipers, 1998](#); [Meeusen et al., 2013](#)). If overreaching is planned and recovery is sufficient, positive adaptation and improved performance, sometimes called supercompensation, result ([Figure 22.11A](#)). This type of overreaching is called *functional overreaching (FOR)*. However, if recovery is insufficient and takes longer than desired, *nonfunctional overreaching (NFOR)* is said to occur. If NFOR is left unchecked or the individual or coach interprets the decrement in performance as an indication that more work must be done with less rest and recovery, NFOR may develop into overtraining ([Figure 22.11B](#)). Overtraining, more properly called the **overtraining syndrome (OTS)** (or staleness), is a state of chronic decrement in performance and ability to train, in which restoration may take several months, or even years. Indeed, the distinction between NFOR and OTS is based primarily on the amount of time needed for performance restoration (approximately several weeks to months for NFOR and several months to years with OTS). This is assuming that all other possible causes of prolonged underperformance have been eliminated ([Armstrong and vanHeest, 2002](#); [Bellinger 2020](#); [Bosquet et al., 2008](#); [Carfagno and Hendrix, 2014](#); [Meeusen et al., 2010, 2013](#); [Nederhof et al., 2008](#); [Schmikli et al., 2011](#)).

Overreaching (OR) A short-term decrement in performance capacity that generally lasts only a few days to 2 weeks and from which the individual easily recovers.

Overtraining Syndrome (OTS) A state of chronic decrement in performance and ability to train in which restoration may take several weeks, months, or even years.

The relationship between overreaching and the overtraining syndrome is often depicted as a continuum, as shown in [Figure 1.10](#) ([Carfagno and Hendrix, 2014](#); [Fry et al., 1991](#); [Meeusen et](#)

al., 2010, 2013). As the imbalance between training and recovery increases, the complexity of the symptoms also increases, and the athlete progresses from overreached to overtrained. Coaches and athletes often use shock microcycles to induce overreaching in the normal periodization of training, making it possible for scientists to follow and test athletes in such a situation. However, it is neither ethical nor reasonable to deliberately induce a state of OTS. Scientific data on OTS have, therefore, largely been derived retroactively from case studies of athletes with unexplained underperformance.

Data on meaningful numbers of subjects in both resistance and endurance activities followed from adaptation and performance improvement through overreaching to the overtraining syndrome are not available, thus it is difficult to identify markers of overtraining (Grandou et al, 2019). Nonetheless, what has become clear is that excessively high training volumes or intensities in resistance exercise presents different physiological and performance profiles when compared to overreaching or overtraining in endurance exercise. When excessive volumes of maximal resistance loads are used for training, maximal muscle strength is one of the last performance variables to be adversely affected. Conversely, high speed/sprint and power activities are the first types of performance to decrease with excessive resistance training (Meeusen et al., 2013). A very long list of signs and symptoms (over 125) has been observed in athletes exhibiting unexplained performance decrements (Fry et al., 1991; Urhausen and Kindermann, 2002). **Table 22.7** lists some, but not all, signs and symptoms that may be observed by athletes, coaches, and athletic trainers.

TABLE 22.7 Signs and Symptoms of the Overtraining Syndrome

| Type | Signs and Symptoms |
|----------------------------------|--|
| Performance related | <ul style="list-style-type: none"> • Consistent decrement in performance • Persistent fatigue and sluggishness that leads to several days of poor training • Prolonged recovery from training sessions or competitive events • Reappearance of already corrected errors • Increased occurrence of muscular accidents/injuries |
| Physiological | <ul style="list-style-type: none"> • Decreased maximal work capacities and markers • Increased disruption of homeostasis at submaximal workloads • Headaches or stomachaches out of proportion to life events • Insomnia • Persistent low-grade stiffness and soreness of the muscles and joints; feeling of "heavy" legs • Frequent URTIs: sore throats, colds, or cold sores • Constipation or diarrhea • Loss of appetite; loss of body weight or muscle mass, or both, when no conscious attempt is being made to diet or when weight loss is undesirable • An elevation of ~10% in the morning resting heart rate taken immediately on awakening |
| Psychological/ behavioral | <ul style="list-style-type: none"> • Feelings of depression • General apathy, especially toward previously enjoyed activities • Decreased self-esteem • Emotional instability or mood changes • Difficulty concentrating • Loss of competitive drive or desire • Perceived insufficient recovery |

The signs and symptoms of OTS appear to be outward manifestations of neuroendocrine imbalances, immune system

activation and/or suppression, or a reversal of normal physiological adaptations. They provide a starting point for research investigating the physiological mechanisms behind OTS as well as serving as a basis for diagnosing OTS.

Hypothesized Causes and Mechanisms of Overtraining Syndrome (OTS)

The precise cause(s) and mechanism(s) of OTS are unknown. A high-volume training load performed at high intensity, applied in a monotonous manner, and without sufficient rest and recovery that alters metabolic processes and leads to an accumulation of muscle trauma appears to be the primary predisposing factor (**Figure 22.12**) (Armstrong and vanHeest, 2002; Budgett, 1998; Fry et al., 1991; Meeusen et al., 2013; Petibois et al., 2002, 2003; Smith, 2000). Related stressors such as frequent competition, excessive travel, training and competing under inhospitable environmental conditions, and/or poor nutrition as well as unrelated stressors such as family, school, or work commitments accumulate and are likely peripheral causes. Thus, OTS may represent the sum of multiple life stressors and can be understood, at least partially with the context of Selye's General Adaptation Syndrome (see Chapter 1) (Meeusen et al., 2013).

Several hypotheses have been developed to explain the physiological mechanisms by which probable causative factors actually bring about OTS. Remember that a hypothesis by definition is “an assumption not proved by experiment or observation. It is assumed for the sake of testing its soundness or to facilitate investigation of a class of phenomena” (Thomas, 1985). Thus, while research data can account for some observed symptoms of OTS, these data are generally insufficient to justify calling any hypothesis a theory (a well-substantiated explanation that has been repeatedly tested and confirmed through observation and experimentation). In short, the underlying mechanism(s) remain(s) unclear (Armstrong and vanHeest, 2002; Carfagno and Hendrix, 2014).

The hypotheses to explain OTS generally address three major areas of physiological disruption: biochemical/metabolic, neuroendocrine, and immunological. **Figure 22.12** indicates how

these areas may interact and overlap in OTS. Refer to this figure throughout the following discussion.

Alterations of Carbohydrate, Lipid, and Protein Metabolism

Muscles obviously use fuel for contractions in exercise training. Indeed, a major objective of exercise training, especially endurance training, is to increase metabolism to support competition. OTS in endurance athletes thus may be mediated primarily through dysfunctions of carbohydrate (CHO), lipid (FAT), and/or protein (PRO) metabolism (Kreider et al., 1998; Petibois et al., 2002, 2003; Snyder, 1998). Glycogen depletion may become a reality with insufficient CHO ingestion after successive training bouts. Low glycogen can in itself lead to the defining signs of OTS, early fatigue, and poor performance (Snyder, 1998). Glycogen, of course, is the primary fuel for anaerobic metabolism. One of the most consistent findings in both endurance and strength athletes exhibiting OTS is a diminished maximal lactate concentration despite submaximal values remaining unchanged or being slightly reduced (Urhausen and Kindermann, 2002). Chronic energy deficiency (primarily glycogen depletion) amplifies the stress hormone and cytokine responses to exercise and may well be one of the “triggering” factors that can lead to the OTS (Meeusen et al., 2013). In fact, some authors have noted the similarities between training overload and Relative Energy Deficiency in Sport (RED-S) (see Chapter 6) and noted that both conditions involve the hypothalamic-pituitary pathways and low carbohydrate and energy availability (Stellingwerff et al., 2021). Furthermore, there is evidence of impaired muscle mitochondrial function that is associated with a decrease in glucose tolerance with excessive training (Flockhart et al., 2021).

The predominant backup fuel to CHO for exercise is, of course, FAT in the form of triglyceride-derived free fatty acids (FFA). While the utilization of higher amounts of FFA and the sparing of glycogen is a beneficial adaptation of training, this typically occurs in the presence of adequate glycogen stores. Without sufficient CHO stores and in the presence of the oxidative stress that accompanies endurance activity, alterations

occur in the triglyceride/fatty acid cycle, and polyunsaturated fatty acids (PUFAs) increase. These changes have been linked to the pathogenesis of inflammation and immunosuppression (Petibois et al., 2002, 2003; Steinacker et al., 2004). Additionally, leptin is released from adipose cells. Leptin provides feedback for satiety, may act as a metabolic hormone, and helps regulate hypothalamic-pituitary function. Movement of fatty acid from adipocytes may inhibit leptin secretion. Low plasma leptin levels activate the hypothalamus-pituitary-adrenal axis (HPAA) as well as sympathetic activity, and as a cytokine, leptin is closely linked to immune function (Saris, 2001; Steinacker et al., 2004).

These alterations in CHO (insufficient) and FAT metabolism (increased FFA utilization) also lead to shifts in PRO metabolism. Protein is not normally utilized extensively as fuel. However, under conditions of high-intensity, long-duration training or competition, especially when CHO stores are inadequate, PRO (particularly the branched chain amino acids [BCAA]) is utilized as fuel. Changes in amino acid levels have been theoretically linked to OTS symptoms.

Altered neuroendocrine function is predominantly mediated through the hypothalamus in Selye's classic stress response (Selye, 1956). The primary neural component is the brainstem (locus ceruleus)-sympathetic nervous system pathway. The primarily hormonal component is the hypothalamus-pituitary-adrenal axis (HPAA). The hypothalamus, as both a neural structure and an endocrine gland, can orchestrate the body's response by stimulating both the sympathetic nervous system and the endocrine glands. OR and OTS must be viewed on a continuum with a disturbance and adaptation and finally a maladaptation of the HPAA and all other hypothalamic axes (Meeusen et al., 2013). Importantly, several hormones, including epinephrine, cortisol, and testosterone, interact with the immune system. Increased levels of serotonin are associated with decreased motor excitability, decreased appetite, increased sleep, and altered neuroendocrine function—among the signs and symptoms often seen in OTS (Budgett, 1998; Gastmann and Lehmann, 1998; Kreider et al., 1998).

Muscle Trauma and Injury/Cytokine Theory

Microtrauma or tissue injury in muscles can impact both the central nervous system (CNS) and the immune system. Activation of the CNS by cytokines explains many of the signs and symptoms of OTS. Anti-inflammatory cytokines define the role of T cells. T cells divide into two distinct functional subsets: TH1, associated with cell-mediated immunity, and TH2, associated with humoral immunity. The up-regulation of TH1 cells primarily depends on IL-12; the up-regulation of TH2 cells primarily depends on IL-4. In addition, IL-4 and IL-10 coordinate to inhibit TH1 development. Thus, IL-10 suppresses cell-mediated immunity and macrophage function. The response to trauma/injury is a shift to a TH2 lymphocyte response, resulting in an up-regulation of humoral immunity and down-regulation of cell-mediated immunity. The individual may thus be more susceptible to URTI (Moldoveanu et al., 2001; Smith, 2003a). The binding of cytokines (particularly IL-6) in the hypothalamus activates the hypothalamic-pituitary-adrenal axis (HPAA) and the sympathetic nervous system, resulting in the release of E/NE and cortisol (Moldoveanu et al., 2001; Steinacker et al., 2004). These stress hormones bring about physical changes and are associated with mood changes such as depression and anxiety. The activation of the hippocampus may explain loss of attention and the return of previously corrected errors (Armstrong and vanHeest, 2002; Keizer, 1998; Smith, 2000, 2003b; Steinacker et al., 2004). Experimental evidence in support of the hypothesis that muscle damage and the resulting cytokine response play a key role in OTS is growing (Cheng et al., 2020; Main et al., 2010). Again, note the tremendous overlap among the neural, endocrine, and immune systems in this theory.

Markers and Monitoring of Training to Predict Overtraining Syndrome (OTS)

There has been extensive research aimed at trying to identify biomarkers that could be used to monitor overreaching or diagnosis overtraining, but the results remain inconclusive with wide variability reported among studies (Carrad et al., 2021). Despite numerous attempts to identify such a marker, the overwhelming consensus remains that no reliable and valid

variable or index is available to predict OTS or even to distinguish among well-trained, overreached, or overtrained athletes (Savioli et al., 2018). A review article that analyzed 39 publications reporting biomarkers and tools potentially diagnostic for OTS reported that many articles were of low quality. The range of the identified markers is consistent with the finding that OTS affects many body systems. Furthermore, the findings support the hypothesis that OTS might be a heterogeneous syndrome which expresses itself in different clinical features (Carrad et al., 2021). Although some individual studies have reported changes in exercise heart rate, or heart rate variability (HRV) as a convenient method for monitoring overreaching, a meta-analysis (Bosquet et al., 2008) has suggested that while such changes may be statistically valid, they have very limited practical value for athletes, coaches, and physicians as they typically fall within the day-to-day variability of heart rate.

Making matters still more difficult, there is no clear demarcation for when overload becomes overtraining load. The margin between adaptation/supercompensation and the functional impairment of OTS is fluid both among athletes and within individual athletes. Many studies have reported a decrease in clinical markers, including HRV, maximum lactate, IL-6, TNFalpha, leptin, adiponectin, GH, cortisol, and prolactin. However, none of these measures are consistently found to be decreased among those suffering from performance decreases associated with OTS (Cadegiani, 2020). Nonetheless, there is consensus that monitoring athletes is extremely important. The difficulty is how and what to monitor (Achten and Jeukendrup, 2003; Duclos, 2008; Hartmann and Mester, 2000; Hooper and Mackinnon, 1995; Lac and Maso, 2004; Meeusen et al., 2013; Urhausen and Kindermann, 2002). Training, performance, and mood state appear to be reasonable and practical places to begin.

Monitor Training and Recovery

All athletes should be encouraged to keep a daily training log. This should include body weight (with periodic evaluation of body composition), morning resting heart rate (often difficult because many athletes do not like doing this), training goal, training achieved, subjective ratings of perceived exertion during

training, fatigue ratings, any noticeable muscle/tendon/joint aches or pains, and any symptoms of URTI (Calder, 2004).

Athletes should be aware of and report (before beginning the day's workout) any meaningful changes (a rise $\geq 10\%$) in resting heart rate, any muscle/tendon/joint soreness, or URTI symptoms persisting from the day before. These could be indications of insufficient recovery. Therefore, it may be necessary to cut back the volume and intensity of the workout on this day to possibly avoid longer setbacks in the future (Hooper and Mackinnon, 1995). New wearable technology is making it easier to monitor resting heart rate and HRV, and some devices, such as the WHOOP (see Chapter 4), use algorithms to provide feedback on cardiac strain throughout the day and recovery achieved overnight strain. The use of HRV to monitor recovery, and thus help prevent OTS, is based on findings that the ANS is disrupted during OTS (Carrod et al., 2021).

At the end of each macrocycle (see Chapter 1), the training log should be carefully evaluated to determine the athlete's response to the training load. The training plan for the next cycle should be based on this evaluation (Smith, 2003a).

Monitor Performance

The most obvious item to monitor is, of course, the specific sports performance, including not only the outcome but also the effort involved and apparent recovery. Performance in training workouts should also be monitored. In some events, individualized ergometric tests can reproduce specific levels of performance in the exercise physiology laboratory or training room (Meeusen et al., 2013; Urhausen and Kindermann, 2002). Because athletes suffering from OTS often start a normal training workout, race, or other event but do not complete the performance as expected, the analysis should include splits in relation to a predetermined strategy. A two-bout exercise test protocol might be best for detecting differences in training status (Meeusen et al., 2013).

Meeusen et al. (2004) detected subtle differences in HPAA hormonal responses to two bouts of maximal exercise 4 hours apart between cyclists in a well-trained ($N = 7$) state and in an intentionally overreached state (same seven athletes) and in one

diagnosed OTS motocross athlete that would have been missed had only one exercise bout been used. Performance itself was also sensitive to the training status. Time to exhaustion in bout 2 decreased only 3% from bout 1 in the well-trained state, doubled to -6% in the overreached condition, and almost doubled again to -11% in the OTS athlete. Follow-up studies ([Meeusen et al., 2010](#); [Nederhof et al., 2008](#)) have supported the sensitivity of the hormonal response (particularly ACTH and prolactin) to a two-bout exercise test in being able to distinguish between NFOR and OTS athletes and NFOR, NFOR recovering, and normal athletes. Performance of an interval shuttle run test (alternately running for 30 seconds and walking for 15 seconds starting at speeds of 10 km·hr⁻¹) has also been shown to be sensitive to detecting NFOR ([Schmikli et al., 2011](#)).

Performance tests must be conducted after a recovery or regeneration cycle (see [Chapter 1](#)) when adaptation or maladaptation can accurately be evaluated and not confused with the normal fatigue of training ([Fry et al., 1992](#); [Rowbottom et al., 1998](#)). This analysis requires, of course, baseline data previously established for the athlete. The Focus on Application box describes a study that suggests that the 20-m shuttle test (20 MST or PACER) may be an easy way to monitor for OTS in team sports athletes.

FOCUS ON APPLICATION

Monitoring Overreaching

The authors were concerned about the lack of clear practical tests for nonfunctional overreaching, which is problematic both for scientists studying the issue and for coaches trying to prevent it. They designed their study to investigate changes in a variety of biochemical, immunological, physiological, and psychological markers for monitoring fatigue and recovery.

Eighteen Australian male rugby league players in a semiprofessional club volunteered and were divided into two

matched groups. All players completed 6 weeks of five to seven training sessions per week of progressive endurance development, resistance training, speed and agility training, and rugby drills. The intensive training (IT) group was intentionally overtrained, while the other group was normally trained (NT). The average training load for the two groups is presented in the accompanying graph, which shows the IT group did approximately 21% more training than the NT group.

Following 6 weeks of training, all players completed the same taper. This was a step-reduction taper consisting primarily of a reduction in training session duration while maintaining training intensity. The activities included three field sessions and two resistance training sessions.

All players were tested before the training protocol, after the 6 weeks of training (except for the 20 MST run each week), and at the completion of the 7-day taper. All measures were taken after 24 hours of rest after the last training session and at the same time of day. The only significantly different variables between the IT and NT groups after the 6-week overload period were the 20 MST, and glutamine/glutamate ratio. Because of its ease of administration, only the 20 MST results are discussed here. The NT group incrementally improved their 20 MST performance by approximately 1% per week through the first 5 weeks and then decreased slightly, for an overall improvement of $+3.3 \pm 7.7\%$ at week 6. In contrast, the IT group showed basically no change through week 3, approximately 1% decrease at week 4, and a decrease of $-9.2 \pm 3.8\%$ by week 6. After the taper, both groups showed an equal improvement of approximately +6% from the start of the training program, with no difference between groups. These results suggest that the 20 MST may be a useful measure for monitoring response to training and that a short taper is useful even for team sports players.



Source: Coutts et al. (2007).

Monitor Mood State

Subjective ratings of mood, fatigue, athletic and nonathletic stress, and muscle soreness have been suggested as the most cost-effective strategy for early detection of OTS and thus for monitoring training (Hooper and Mackinnon, 1995; Lehmann et al., 1993; Meeusen et al., 2013; Meyers and Whelan, 1998; Urhausen and Kindermann, 2002). The Profile of Mood States (POMS) (McNair et al., 1971) has been most extensively studied. This 65-item test provides subjective ratings of tension-anxiety, depression, anger, vigor, fatigue, confusion, and total mood. A shorter 24-item version is also available but is sometimes called the BRUMS, Brunel Mood Scale (Rohlfes et al., 2008; Terry et al., 2003). Although Profile of Mood States cannot diagnose OTS, it can document mood changes consistent with the condition (such as increased depression and tension-anxiety and decreased vigor and total mood score) in some athletes (Hawley and Schoene, 2003; Hooper and Mackinnon, 1995). The deterioration in mood states often coincides with an increased training load and usually precedes a decrement in performance (Urhausen and Kindermann, 2002). Athletes with signs of OTS typically exhibit both a greater increase and a different pattern in total mood disturbance than athletes undergoing the same training who remain symptom free. Male and female athletes respond similarly (Meeusen et al., 2013). Self-analysis questionnaires using well-being ratings can prove useful, including ratings of fatigue, stress level, muscle soreness (especially “heavy” legs), training

enjoyment, health concerns, irritability, self-confidence/self-esteem, attitude toward work/study/teammates, communication with teammates and coaches, and sleep quality/disorders. Although verbal reports are very important, all of these items should be recorded in a written training log. Subjective portions of this log require candor and complete honesty by the athlete and must be evaluated in light of training and performance. The usefulness of the log depends heavily on the coach's or athletic trainer's interpretation. The RESTQ-76 Sports questionnaire uses a self-report approach to evaluate physical, subjective, behavioral, and social aspects of stress and recovery (Kellman and Kallus, 2001). It is more specific in pinpointing disturbances related to or affecting training and is effective in identifying individuals at risk for OTS although it cannot provide the final diagnosis that someone is already overtrained (Carfagno and Hendrix, 2014). A 22-item questionnaire that includes six distinct symptom clusters (depression, vigor, physical symptoms, sleep disturbance, stress, and fatigue) covering three domains (mood, stress, and behavioral/physical symptoms) is informative for coaches and user-friendly for athletes but requires further validation (Main and Grove, 2009).

Prevention and Treatment of Overtraining Syndrome (OTS)

Prevention

Attempts to prevent the overtraining syndrome center on periodization of the exercise training with an emphasis on adequate recovery for adaptation and proper nutrition.

PERIODIZATION The key to preventing the OTS is careful periodization of training (see Chapter 1). It is difficult to attain the delicate balance between overload/progression (avoiding large immediate increases in overload) and sufficient rest/recovery, but this balance is essential for each individual (Meeusen et al., 2013; Smith, 2003a). Periodization allows the athlete to peak for specific competitions. Peak performance cannot be maintained indefinitely, so competitions must be

prioritized and periodization phases matched to these priorities. The more frequently peaks are attempted, the more vulnerable the athlete is to overtraining (Bompa, 2004; Smith, 2003a). Few studies have actually compared the effectiveness of periodization. However, Rhea and Alderman (2004) and Steinacker et al. (1998) have shown positive results for strength/power (weight lifting) and aerobic/anaerobic (rowing) programs using periodization.

The main protection against OTS is the inclusion of sufficient recovery after heavy physical training, including each daily training session and each competition. Athletes and coaches indoctrinated with the philosophy of “if some is good, more is better” often find it difficult to accept that “less can actually be more.” Possible components of daily recovery include hydration and proper nutritional refueling as soon as possible to deal with metabolic fatigue; light activity, stretching, and hydrotherapy (showering or pool/spa activities) to deal with neural fatigue; and unwinding with activities such as debriefing or listening to music to deal with psychological fatigue (Calder, 2004). This recovery should be monitored. Restful sleep of 8–9 hours is often prescribed; however, individuals have different requirements for sleep. What is essential is that the individual athlete sleeps for the amount of time that is required to feel wakeful during the day (Meeusen et al., 2013). Sufficient recovery should also be included within the training progression itself through the inclusion of easy days, days of active cross-training, rest days emphasizing stretching and relaxation techniques, and at least 1 day of complete rest per week (Kenttä and Hassmén, 1998; Meeusen et al., 2013).

NUTRITION Adequate nutrition, with the possible periodization of carbohydrate intake to match glycogen needs, is also critical. A key goal is to ensure sufficient glycogen stores to support high-intensity training or competition. Maintaining blood glucose is also important for attenuating increases in stress hormones (especially cortisol), diminishing the changes in immunity mediated through cytokines. Ingestion of carbohydrate during prolonged high-intensity exercise has been shown to blunt the inflammatory response (Coyle, 2004; Kreider et al., 1998; Venkatraman and Pendergast, 2002).

One to two hours of high-intensity exercise or as little as 20–

30 minutes of heavy interval training can result in glycogen depletion. As detailed in [Chapter 6](#), glycogen resynthesis occurs at about 5–6% per hour under optimal dietary conditions, thus requiring approximately 17–20 hours for complete recovery. Optimal conditions involve both the timing and the type of CHO ingestion. Consuming CHO immediately after exercise results in higher glycogen levels 4 hours after exercise than if CHO ingestion is delayed 2 hours. Thus, when the interval between exercise sessions is short (<8 hours), carbohydrate ingestion (50–100 g at the rate of 1.2 g·kg BW⁻¹·hr⁻¹) should begin as soon after the workout or competition as is practical (15–30 minutes); continue at that rate every 15–20 minutes until a larger meal of solid food (150–250 g of carbohydrate) is desired and possible; and be maintained for at least 4–6 hours ([Betts and Williams, 2010](#); [Cermak and van Loon, 2013](#); [Coyle and Coyle, 1993](#); [Jentjens and Jeukendrup, 2003](#)). When a longer recovery time is available, although the consumption of CHO immediately is not as important, it is recommended ([Ivy and Ferguson-Stegall, 2014](#)). As long as 7–10 g·kg BW⁻¹ of CHO is consumed over 24 hours at a rate of at least 50 g·hr⁻¹, muscle and liver glycogen will be replaced over this time ([Donaldson et al., 2010](#)). Carbohydrate supplementation can reduce postexercise stress hormone levels, inflammation, and oxidative stress. Furthermore, ingesting fruits high in carbohydrates and polyphenols can support performance and enhance oxidative and antiviral capacity ([Nieman and Mitmesser, 2017](#); [Spirandelli et al., 2020](#)). Adequate nutrition involves more than just carbohydrate ingestion, however. Athletes in hard training seem to maintain or decrease their spontaneous food intake, and a negative energy balance can occur ([Carfagno and Hendrix, 2014](#); [Meeusen et al., 2013](#)).

Fat and protein ingestion are critical. Low-fat diets (<20%) compromise endurance performance and lead to possible deficiencies in micronutrients. Both the quantity and type of fats alter immune functioning. Diets very low (<20%) or very high (>60%) in protein have negative effects on immune function as well. Diets unbalanced in fat and protein may be a factor in overtraining. Supplementation with amino acids, however, is not likely to reduce symptoms of fatigue or OR ([Meeusen et al., 2013](#)).

The Role of Nutritional Supplements in Lessening Immune Disruption with Strenuous Training

Walk into any nutritional supplement store (or even your local grocery store or drug store) and you will see numerous supplements claiming to boost immune function. Nutritional supplements to bolster immunity have long been used during periods of physiological stress, such as during surgery or during recovery from trauma. This has led many to speculate that nutritional agents may be useful in attenuating immune changes and inflammation following strenuous exercise and thus may lower the risk of upper respiratory tract infection. While there are theoretical reasons to believe that different supplements may be useful and some evidence in animal models to suggest the utility of supplementation, the majority of studies conducted with human athletes have been disappointing.

The accompany table reviews many proposed nutritional supplements and their rational for use along with current recommendations based on The International Society of Exercise and Immunology Position Statement on Immune Function and Exercise and review articles ([Bermon et al., 2017](#); Li et al., 2016; Neiman et al., 2017; Walsh, 2011a, 2011b). The reviews conducted for this table note the relative lack of well-designed, long-duration studies applying appropriate outcome measures on a large number of athletes. However, at this point, the scientific findings suggest that there are few nutritional supplements, other than carbohydrates, that are effective in dampening immune disruption with exercise or in reducing risk of upper respiratory tract infection in well-fed healthy athletes. However, there are cases where the findings are mixed, with some studies suggesting potential benefit while other studies did not find a benefit.

There are only two recommendations based on current scientific studies: carbohydrate supplementation and diets high in fruit and vegetable intake. There is strong evidence that carbohydrate ingestion before and/or during prolonged exercise attenuates the increase in blood neutrophil and monocyte counts, stress hormones, and anti-inflammatory cytokine activity. Thus, carbohydrate ingestion during heavy exercise may provide a partial countermeasure to inflammatory responses associated with exercise. Fruits and vegetables are rich in vitamins and minerals and contain flavonoids, including quercetin. Quercetin, a dietary polyphenol found in tea, some fruits (e.g., apples, grapes, citrus fruits, raspberries, cranberries), and green leafy vegetables, has been investigated as a potential supplement. Fruits and vegetables augment oxidative capacity, enhance antiviral defenses, and dampen inflammation—and they enhance vascular health.

While the idea of a nutritional supplement to boost immune function is attractive, and a lucrative business, the available scientific evidence suggests that athletes should eat a sound healthy diet that includes plenty of fruits and vegetables, ensure they have sufficient carbohydrates immediately before or after exercise, drink plenty of water to stay properly hydrated, and perhaps recover with a cup of green tea.

| Potential Immunonutritional Supplement | Proposed Rationale | Recommendation Based on Current Evidence |
|--|--|---|
| Macronutrients | | |
| Carbohydrate | Maintains blood glucose during exercise, lowers stress hormones, and partially counters inflammation and immune changes | Recommended; 30–70 g·hr ⁻¹ of heavy exertion |
| High fruits and vegetable intake with extracts rich in polyphenols | Augmented oxidative capacity; enhanced antiviral defenses; aid inherent defenses against inflammation and oxidative stress | Recommended |
| Branched chain amino acids (BCAAs) | BCAAs (valine, isoleucine, and leucine) are the major nitrogen source for glutamine synthesis in muscle | Not recommended; lack of quality data |
| Vitamins/Minerals | | |
| Vitamin E | Quenches exercise-induced reactive oxygen species (ROS) and augments immunity | Not recommended; may be pro-oxidative; and proinflammatory at high doses. |
| Vitamin C | Quenches ROS and augments immunity | Not recommended; not consistently different from placebo |
| Vitamin D | Plays a key role in both innate and adaptive immunity | Mixed results |
| Multiple vitamins and minerals | Work together to quench ROS and reduce inflammation; cofactors for immune response | Not recommended; not different from placebo; balanced diet is sufficient (may be useful if diet is not sufficient). Concerns over blocking ROS signaling for training adaptations |
| Advanced Supplements | | |
| Glutamine | Important immune cell energy substrate that is lowered with prolonged exercise | Not recommended; body stores exceed exercise-lowering effects |
| Bovine colostrums | Mix of immune, growth, and hormonal factors improves immune function and the neuroendocrine system and lowers illness risk | Mixed results |
| Probiotics | Improve intestinal microbial flora and thereby enhance gut and systemic immune function | Mixed results |
| N-3 PUFA (fish oil) | Exerts anti-inflammatory effects postexercise | Mixed results |
| β-Glucan | Receptors found on immune cells, and animal data show supplementation improves innate immunity and reduces infection rates | Mixed results |
| Herbal supplements (e.g., ginseng, Echinacea) | Contain bioactive molecules that augment immunity and counter infection | Not recommended; humans studies do not show consistent support within an athletic context |
| Quercetin | Anti-inflammatory, antioxidative, and antipathogenic effects | Recommended, especially when obtained by eating a diet high in fruits and vegetables |

Sources: [Bermon et al. \(2017\)](#); [Li et al. \(2016\)](#); [Neiman et al. \(2017\)](#); [Walsh et al. \(2011a, 2011b\)](#).

Hydration, the last critical component, should include both water and sports drinks. Thirst is not a reliable indication of the amount of fluid needed. [Chapters 6 and 14](#) discuss fluid needs for adequate hydration and rehydration.

The quest to find the foods or nutritional supplements to support the immune system during strenuous training has motivated scientists, athletes, and commercial entities for many years. Although supplements are aggressively marketed to help with performance and to promote recovery, scientific data suggest that a sound diet should form the basis of sport nutrition. The accompanying Focus on Application box reviews the current recommendations regarding nutritional supplements to lessen immune disruption with strenuous training and to help prevent OTS.

Treatment

If training adjustments are insufficient and OTS develops, treatment is needed. The first step in treatment is a medical examination to rule out possible illnesses or injuries as the underlying cause of a performance decrement. A nutritional analysis may be valuable as well given the overlap between OTS and RED-S. Once these possible causes have been ruled out, the individual must be guided through a recovery program. This must be done carefully, because active—and especially competitive—individuals often resist recommendations to cut down or cease training. Rest may be a naughty four-letter word to an athlete, but rest is the only established treatment. Reduced training may be sufficient for recovery in some cases of OR, but recovery from OTS requires rest (Meneses-Echavez et al., 2015; Mujika and Padilla, 2000).

Armstrong and VanHeest (2002) point out that OTS and clinical (major) depression involve very similar signs and symptoms, brain structures, neurotransmitters, endocrine pathways, and immune responses and thus probably have similar causes. Given these communalities, they and others, including the American College of Sports Medicine (ACSM, 2000), suggest treating overtrained athletes with antidepressant medications and professional psychological counseling. Research supporting the use of specific antidepressants is needed.

Just as there is no definitive marker for overtraining, so too is there no definitive marker to indicate recovery. Resumption of training must be individualized on the basis of signs and symptoms and be gradual. The best “treatment,” of course, is prevention (ACSM, 2000; Flynn, 1998; Meeusen et al., 2013).

Complete the [Check Your Comprehension 3—Case Study 1](#) to evaluate your understanding of how to evaluate recovery from training in an attempt to avoid the OTS.

CHECK YOUR COMPREHENSION 3—CASE STUDY 1

The table below includes data for two individuals, Candace and Maya, who are undergoing late general preparation/early sport-specific training for basketball. Both players are in their junior year and have been regular starters for the team. The coach has suggested that players use the WHOOP to monitor their

recovery. The data given below summarize their training during the week, and the RPE, HRV, and total recovery score of both athletes. The recovery score is a proprietary measure that integrates information from HRV, HR, sleep, and respiration to indicate the "percent of recovery."

Review the data in the table below. If this pattern were to continue, which player is most likely to suffer training maladaptation? Support your answer.

What workout adjustment(s) would you make to this individual's training routine to reduce the likelihood of maladaptation? Would you make any adjustments to the other individual's training routine? If so, what?.

Check your answers in [Appendix C](#).

| Day | Training Session | Duration (min) | RPE: CR10 Scale | HRV | %Recovery |
|---------|-----------------------|----------------|-----------------|-----|-----------|
| Candace | | | | | |
| M | Resistance | 90 | 5 | 89 | 84 |
| T | Speed, agility, power | 45 | 7 | 100 | 82 |
| W | Endurance running | 60 | 4 | 96 | 86 |
| TH | Speed, agility, power | 45 | 7.5 | 104 | 74 |
| F | Resistance | 90 | 5 | 91 | 68 |
| S | Speed, agility, power | 45 | 6.5 | 108 | 84 |
| SU | Endurance | 60 | 4 | 88 | 76 |
| Maya | | | | | |
| M | Resistance | 90 | 4 | 68 | 72 |
| T | Speed, agility, power | 45 | 7.5 | 70 | 46 |
| W | Endurance running | 60 | 5 | 48 | 68 |
| TH | Speed, agility, power | 45 | 7.5 | 62 | 52 |
| F | Resistance | 90 | 3.5 | 44 | 38 |
| S | Speed, agility, power | 45 | 7.5 | 42 | 42 |
| SU | Endurance | 60 | 5 | 38 | 34 |

Selected Interactions of Exercise and Immune Function

Exercise, the Immune System, and Upper Respiratory Tract Infection

Many people believe that individuals who exercise regularly are less likely to acquire a cold or the flu, whereas others believe that highly trained, competitive athletes are more susceptible to upper respiratory tract infections (URTI-). This topic has been the focus of considerable research over the past 50 years, and there is still uncertainty around the results. In fact, even the causes of URTI are still poorly understood, although it is generally accepted that

it can be related to innate and genetic susceptibility and to several environmental factors connected with training load and nutrition (Cicchella et al., 2021). It is important to note that the relationship between exercise and URTI is strongly influenced by the exercise intensity and overall training load (Gleeson, 2007; Mackinnon, 1999; Matthews et al., 2002; Nieman, 2000, 2012; Nieman et al., 1990, 1993; Trochimak and Hübner-Wozniak, 2012).

Figure 22.6 shows a schematic model explaining the theoretical relationship between exercise and health outcomes, including the incidence of infection. This model suggests that exercise is a stressor that can lead to immunomodulation (either immunoenhancement or immunosuppression) via various mechanisms and that immune enhancement or suppression can lead to a decreased or increased risk of infections, respectively.

As discussed earlier, an acute bout of exercise leads to changes in immune cell number and cell function. **Figure 22.13** presents the “open window” hypothesis, which suggests that the suppression of immune function (based on NK cell activity) is greater and more prolonged after severe exercise than after moderate exercise (Nieman and Wentz, 2019; Pedersen and Ullum, 1994). Notice the similarities between the data presented in **Figure 22.7** and the theoretical curve in **Figure 22.13**. The decrease in the number of the T cells and NK cells and the decrease in NK cell activity during recovery from strenuous exercise form the basis of the Open Window hypothesis, or a period of increased susceptibility (the “open window” represented by the rectangular shaded area in **Figure 22.13**) for infection.

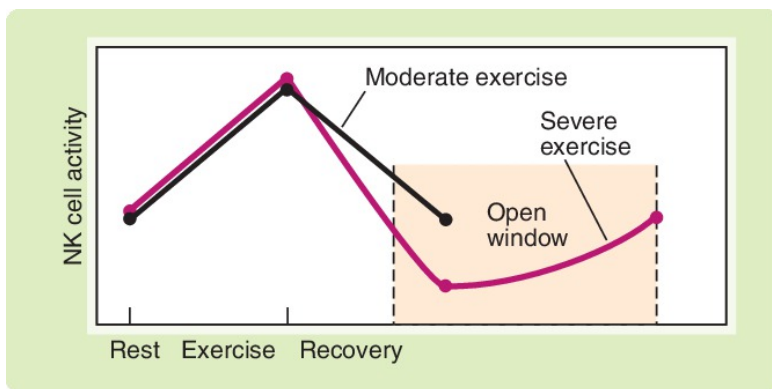


FIGURE 22.13 The “Open Window” Hypothesis.

The open window (*shaded rectangular area*) is a period after severe exercise when NK cell activity decreases. It is proposed that during this period, microbial agents can establish an infection. **Source:** Adapted with permission from Pedersen, B. K., & H. Ullum: NK cell response to physical activity: Possible mechanisms of action. *Medicine and Science in Sports and Exercise*. 26(2):140–146 (1994). Copyright ©1994 The American College of Sports Medicine.

Although there is a strong consensus that immune cell numbers are decreased, and some measures of immune function are suppressed, for at least several hours during recovery from prolonged, intense endurance exercise, the Open Window hypothesis is not universally accepted. Some researchers suggest that changes in immune cell number and function measured in recovery may have little or no effect on health outcomes, such as infections ([Rowbottom and Green, 2000](#)). Other researchers interpret the data to suggest that the decrease in immune cells in the blood may reflect their movement into peripheral tissue where they can actually provide increased immune surveillance and even immunoenhancement ([Campbell and Turner, 2018](#); [Edwards et al., 2007](#)).

Scientific data on whether strenuous/severe exercise or high training loads lead to greater prevalence of upper respiratory tract infections is equivocal. Initial epidemiological studies supported the theory that prolonged, strenuous exercise, such as

marathon running, led to a higher incidence of URTI (Nieman, 1997b). On the other hand, moderate exercise training has consistently been associated with a lower self-reported incidence of URTI (Matthews et al., 2002). Based on these findings, a J-shaped model of the relationship between exercise workload and risk of URTI (see **Figure 22.14**) has been proposed (Nieman and Wentz, 2019; Trochimak and Hübner-Wozniak, 2012; Woods et al., 1999). This model suggests that a moderate level of exercise is beneficial but that very high levels of training may be detrimental. While this theory continues to be held by many exercise immunologists, not all studies have found that intense training is associated with an increased risk of URTI (Gleeson, 1996; Hemilä et al., 2003; Pyne et al., 1995) and some researchers have pointed out inadequacies in the research linking strenuous exercise to URTI (Campbell and Turner, 2018). An important limitation of most studies that have shown an increased risk of URTI among athletes engaged in strenuous athletic events (such as marathons, ultramarathons) is that they relied on self-reported symptoms of URTI rather than laboratory-based analyses of infection. Importantly, a study that used nasopharyngeal and throat swabs in athletes who reported URTI symptoms over a 5-month period found that only 30% of them had a positive laboratory diagnosis. Thus, the URTI *symptoms* reported in studies cited earlier in this paragraph may have been due to causes other than infections, such as allergy or asthma, mucosal inflammation, airway irritation due to increased respiratory rate, or exposure to cold air. Furthermore, there are research studies that have found that higher physical activity levels are associated with a lower incidence of URTI (Fondell et al., 2011). Thus, although the theory that intense training may lead to an increase in incidence of URTI has substantial support, much more research is needed before the actual relationships between strenuous exercise and immune function and clinical outcomes are fully known.

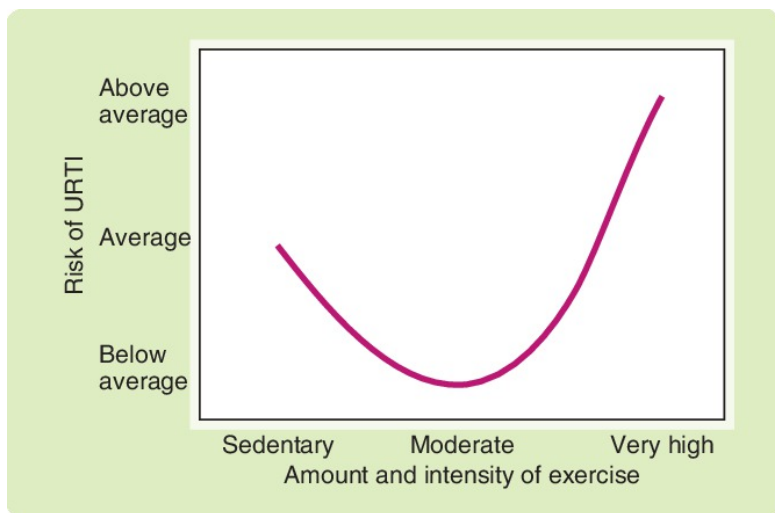


FIGURE 22.14 J-Shaped Model of Relationship between Exercise Intensity and URTI.

Source: Adapted with permission from Pedersen, B. K., & H. Ullum: NK cell response to physical activity: Possible mechanisms of action. *Medicine and Science in Sports and Exercise*. 26(2):140–146 (1994). Copyright ©1994 The American College of Sports Medicine.

Research indicates that moderate exercise enhances immune function and may play an important role in reducing an individual's susceptibility to infection. Exercise is immunomodulatory; whether exercise is immunosuppressive or immunopotentiating likely depends on the overall magnitude of the stress, including not only the physiological stress of exercise but also environmental, psychological, and nutritional stress. The immune response to exercise is mediated largely through the neuroendocrine system. Currently, evidence is insufficient to recommend any given laboratory test of immune function to ascertain whether an individual is at high risk for an infectious episode linked with overtraining or psychological stress. See the Focus on Research box for a look at how investigators are exploring this possibility.

Effect of Football Training on Upper Respiratory Tract Infection and Salivary IgA

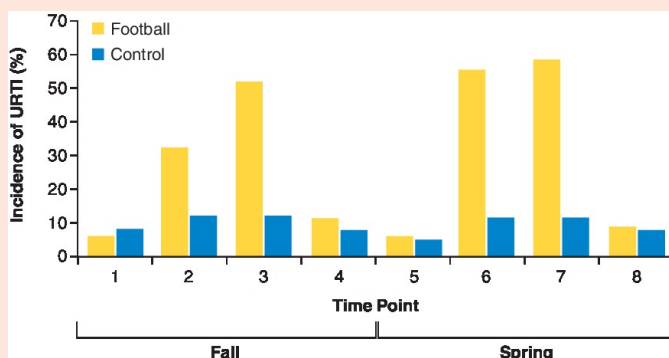
Coaches and athletes are concerned about URTI infections because they impair performance and may limit the ability to train. Given the detrimental effects, many are interested in finding immune markers to predict who is at risk for a URTI. Salivary immunoglobulin A (s-IgA) is a potential marker to predict URTI because the oral mucosa is the first line of defense against pathogens that may enter the body through this route.

Researchers investigated s-IgA and incidence of URTI in collegiate football players and normally active controls over 12 months. Data collected at eight specific times representing different training phases included both s-IgA data and self-reported data about symptoms of URTI.

As seen in the graph accompanying, the incidence of URTI increased dramatically following the first 6 weeks of training in both the fall and spring season (times 2 and 6, respectively) and during the fall and spring season (times 3 and 7). Analysis of the s-IgA data revealed that both the concentration of s-IgA and the secretion rate of s-IgA decreased in the football players during the same period. A regression analysis of these data revealed that the rate of s-IgA secretion was the most predictive of URTI.

These results have several important implications for those who work with athletes. Following intensive preseason training and during the in-season training phase, the incidence of URTI may be very high (>50%) among athletes—much higher than among normally active controls. The increased incidence of URTI is associated with suppression of s-IgA. The exact cause for this higher incidence of URTI is not certain but likely includes the stress of intensive training and competition along with more frequent exposure to the infections of other players because

football players spend so much time in close contact. One interesting finding was that s-IgA secretion rates below 40 mg·min⁻¹ were predictive of URTI, suggesting that in the future coaches and trainers may be able to measure this variable to predict who is at greatest risk for developing a URTI and who would, therefore, benefit from reduced training.



Source: Fahlman, M. M., & H.-J. Engles: Mucosal IgA and URTI in American college football players: A year longitudinal study. *Medicine & Science in Sports & Exercise*. 37(3): 374–380 (2005).

Precautions to Lessen Risk of Upper Respiratory Tract Infection (URTI)

Because competitive endurance athletes often do not have the option of training moderately, the precautions listed below can help lessen their risk of URTI (Cicchella et al., 2021; Gunzer et al., 2012; He et al., 2013; Robson-Ansley et al., 2012; Walsh et al., 2011a, 2011b):

1. Eat a well-balanced diet. Improper nutrition can compound the negative influence of heavy exertion on the immune system. Both carbohydrate and optimal vitamin D status appear to be particularly important.
2. Minimize other life stresses. Psychological stress may be

additive.

3. Avoid overtraining and chronic fatigue. Get plenty of rest, and space vigorous workout and race events as far apart as possible.
4. Consume plenty of carbohydrates before and/or during prolonged exercise.
5. Practice good sleep hygiene.
6. Get a flu shot. Flu shots are especially important for athletes competing in the winter.
7. Avoid excessive muscular soreness. Soreness may activate immune system involvement, and interfere with its ability to ward off viruses.
8. Try to avoid being around sick people.
9. Be tested for allergies and take competition approved allergy medications if needed.

Guidelines for Postponing Training Due to Upper Respiratory Tract Infection (URTI)

One of the challenges facing exercise enthusiasts, athletes, and occupational workers is knowing when exercise can be safely and effectively performed during an illness. The following guidelines suggest when exercise training is and is not appropriate.

1. Maintain training if the URTI is only minor and a systemic infection is lacking (no fever, aching muscles, extreme fatigue, or swollen glands); or just stop for a couple of days. Consider using decongestants during the day and antihistamines at night.
2. If the URTI causes positive signs of systemic infection (fever, aching muscles, extreme fatigue, swollen glands), stop training for 2–4 weeks. If training is not stopped, viral cardiomyopathy or severe viral infection may result.
3. If symptoms occur above the neck (runny or stuffy nose, scratchy throat), begin sessions with a short or light activity. If symptoms worsen, stop; if symptoms lessen, continue. If symptoms are below the neck (muscle ache, vomiting, diarrhea, fever), stop training until the symptoms go away.

Return to Play Guidance Following COVID-19 Infection

One of the challenges facing athletes, coaches, and trainers is when an athlete should return to exercise training and competition following an injury or illness. Athletic trainers are often charged with making such decisions, but following a serious injury or illness, it is often a team physician or treating physician who makes such a decision.

The COVID-19 pandemic presented a unique challenge because of the scale of infections, the wide range of clinical disease expression, and the broad scope of clinical severity. COVID-19 affects many systems, including the cardiac, pulmonary, musculoskeletal, nervous, hematological, and gastrointestinal systems. And, individuals with a COVID-19 infection can be asymptomatic, or have mild-moderate illness, severe illness or even die. For survivors, knowing when and how to return to exercise is a challenge for individuals responsible for sports teams and for individuals seeking to maintain fitness. This was especially true during the early stages of the pandemic when little was known about the consequences of the infection.

Researchers used past experience and available evidence to provide practitioners with actionable guidelines:

- Athletes with severe illness or with complications from COVID-19 should have a medical assessment involving blood tests, cardiac monitoring and imaging, and pulmonary function assessment before beginning a graduated return to play protocol.
- Those with mild to moderate COVID-19 infection should meet the following guidelines before initiating a graduated return to play protocol:

- Follow local guidelines such as masking and

social distancing.

- Rest at least 10 days from onset of symptoms.
 - Rest at least 7 days after being symptom free.
 - Be able to compete activities of daily living (e.g., walk 500 m) without excessive fatigue or shortness of breath.
 - Be off of all treatments.
- The graduated return to play protocol progressed through 6 progressive stages before an athlete returns to competition. The successive steps included:
 - Gradually increasing the intensity and duration of the activity
 - Monitoring subjective symptoms, HR, and RPE

Experience during the COVID-19 pandemic reinforced the need for guidelines to protect athletes and fitness participants by providing guidance on when individuals should return to exercise based on the specific illness and severity of the illness. The attentive exercise physiology student will also recognize how these guidelines relied on sound exercise physiology principles of gradually increasing the overload (time, intensity, and frequency) to guide a return to exercise program. In fact, the guidelines for COVID-19 are similar to guidelines put forth for any type of transition period when an athlete has had to stop training for a period of time.

Sources: [Caterisano et al. \(2019\)](#); [Elliott et al. \(2020\)](#); [Metzl et al. \(2020\)](#).

Exercise, the Immune System, and Cancer

There is consistent and compelling evidence that physical activity plays a role in preventing many types of cancer and improves longevity among cancer survivors ([Patel et al., 2019](#)). **Figure 22.15** presents hazard ratios for 13 different cancer types in which higher amounts of leisure-time activity (90th percentile [in many studies equivalent to the recommended 150 min·wk⁻¹] vs. 10th percentile) were shown to reduce the risk for developing

these cancers (Moore et al., 2016). The easiest way to interpret the hazard ratios is to subtract the ratio from 1.00 and multiply that value by 100. For example, esophageal cancer has a hazard ratio of 0.58. $1.00 - 0.58 = 0.42 \times 100 = 42\%$. This means that those participating in high levels of leisure-time physical activity have a 42% reduced risk of developing esophageal cancer. The other 12 cancer risks vary from a reduction of 27–10%. For 7 of the cancers, the risk reduction was 20% or more. These results were based on data from 12 prospective U.S. and European studies with self-reported moderate to vigorous physical activity. A total of 1.44 million participants aged 19–98 years (57% female) were involved. Twenty-six different cancers were assessed. In addition to the 13 where risk was lower with higher physical activity, 9 showed no significant effect of activity level and 2 (malignant melanoma and prostate cancer) showed a higher risk with higher activity. Greater sun exposure from exercising outdoors is likely the cause of the increased risk for melanoma. The investigators could not determine any biological rationale for the increase in prostate cancer but postulated that screening bias (more active people are also more likely to see a doctor for routine screenings) might have had an impact. The associations were generally similar between overweight/obese and normal-weight individuals. Smoking modified the associations for lung cancer but not other smoking-related cancers. The authors concluded that these findings support promoting physical activity as a key component of population-wide cancer prevention and control efforts. Note: this does not imply that physical activity is an absolute protection against even some cancers. It simply means a lower risk. How does cancer develop and how can physical activity/exercise training lower the risk of this happening? Obviously, the immune system is involved.

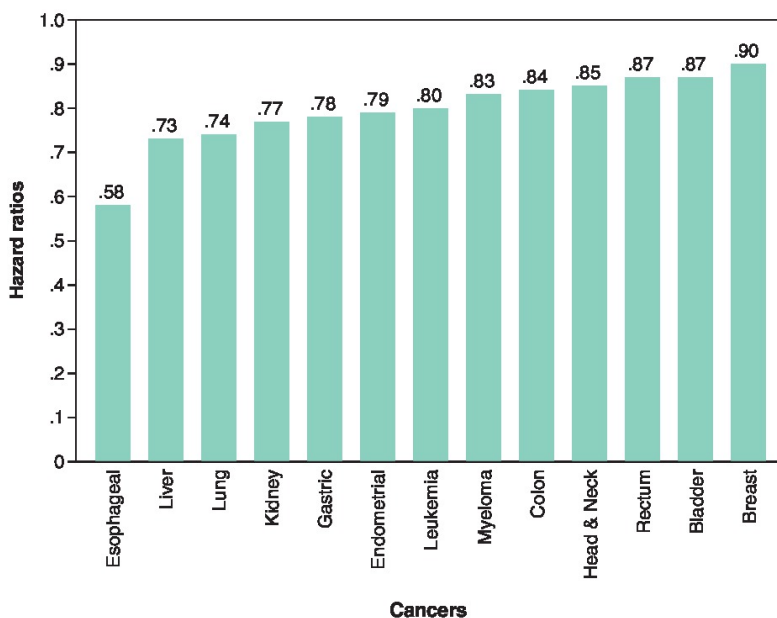


FIGURE 22.15 Hazard Ratios for Higher versus Lower Leisure-Time Physical Activity by Cancer Type Showing Significant Risk Reductions.

Source: Based on data from [Moore et al. \(2016\)](#).

The duel between the immune system and cancer develops in three phases: (1) elimination, (2) equilibrium, and (3) escape. Phase 1 represents immune surveillance and action. Tumor cells are detected by cells of both the innate (especially NK cells) and adaptive immune system (especially T cells). Some of the cancer cells are eliminated. In phase 2, tumor cells are contained but not eradicated; growth is abated, but the tumor is not destroyed. Mutations begin to occur by the tumor cells, and these become resistant to being killed. Unstable malignant cells escape antigen-specific T-cell recognition by antigenic loss or up-regulation of immunosuppressive mediators. Finally, in phase 3, as a result of these immunoevasive mechanisms and mutations, the malignant cells escape containment and uncontrolled tumor growth occurs ([Bigley et al., 2013](#); [Koelwyn et al., 2015](#)).

The interaction between the growth of cancer cells, the overwhelming of the immune system, and chronic exercise

training can probably best be explained by adding the additional factor of age into the explanation. While individuals of all ages contract cancer, the incidence of the disease increases with age. Over 60% of new cancer cases and 70% of cancer deaths occur in individuals aged ≥ 65 years. Cancer is the second leading cause of death for all ages (birth to >80 years), but the leading cause for individuals aged 40–80 years. From birth to age 49 years, the probability of developing invasive cancer is 3.4% or 1 in 29. This rises to 34.6% (1 in 3) over the age of 70 years (Siegel et al., 2016). With age comes immunosenescence. **Figure 22.16** diagrams several critical factors in these age-related immune system changes and the impact of exercise on them.

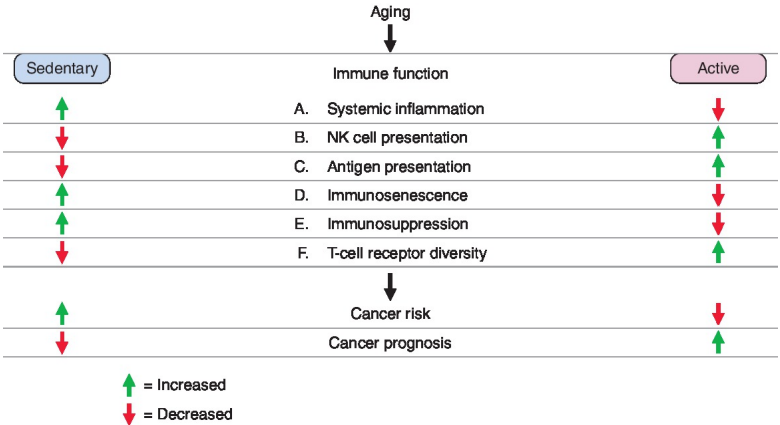


FIGURE 22.16 The Interaction of Age, Immune Function, and Activity Status.

Aging causes detrimental changes in the immune system leading to an increased risk of a variety of cancers and decreased probability for a good prognosis. Chronic activity/exercise training can improve these detrimental changes, decrease the risk of contracting cancer, and improve the prognosis for a good outcome. **Sources:** Based on information from Archer et al. (2011); Bigley et al. (2013); Koelwyn et al. (2015).

Aging increases proinflammatory monocytes, which lead to elevated levels of local and circulating proinflammatory cytokines

(**Figure 22.16A**). This chronic, low-grade inflammation creates a tumor-promoting environment. Although extreme exercise such as marathon running or a full triathlon can be proinflammatory, in most instances, moderate endurance and resistance exercise is anti-inflammatory (Koelwyn et al., 2015; Lancaster and Febbraio, 2014). Aging is associated with decreased NK cell expression in activating receptors and function (**Figure 22.16B**). Chronic aerobic and resistance exercise have shown promise as a means of increasing NK cell toxicity in older individuals. Antigen presentation is impaired in the elderly (**Figure 22.16C**). Monocytes become macrophages. Macrophages are important in antigen presentation to activate T cells. Exercise enhances the cytotoxic, antigen-presenting, and tumor-suppressive functions of monocytes and macrophages. Maintenance of equilibrium (phase 2 above) requires vigorous T-cell responses. T cells recognize, regulate, and eradicate cancer cells (Koelwyn et al., 2015). Unfortunately, aging is associated with an accumulation of functionally exhausted, senescent suppressor CD8⁺ T cells that are incapable of replicating sufficiently (**Figure 22.16D**). To compound matters, senescent T cells have also been shown to suppress other immune activities (**Figure 22.16E**). In addition, a T-cell pool with a large receptor diversity (TCR diversity) is needed to fight all of the novel mutations that premalignant cells undergo in an effort to escape immunological containment. Older individuals have marked age-driven reductions in TCR diversity in both the CD4⁺ and CD8⁺ T-cell subsets (**Figure 22.16F**). Increasing scores for cardiovascular/aerobic fitness have been shown to be associated with increased proportions of naive (new) CD8⁺ T cells and decreased proportions of senescent/exhausted CD4⁺ and CD8⁺ T cells. Exercise training in previously sedentary older individuals has been shown to enhance T-cell proliferation (Bigley et al., 2013). Taken together, these changes offer possible, although not definitive, evidence for the value of exercise in the fight against cancer in older individuals. Of course, the benefits of acute and chronic exercise on the immune system not tied completely to senescence would operate in younger individuals as well.

In addition to aging, body composition is an important contributor to inflammation. When an individual becomes obese, the immune cell profile changes. Specifically, the proportion of

anti-inflammatory macrophages, eosinophils, and CD4+ T cells decreases, and the proportions of proinflammatory activated macrophages, neutrophils, B cells, and CD8+ T cells increase. These changes initiate a state of local inflammation. As stated previously, chronic inflammation (whether from chronic infections, improper immune function, autoimmune disease, aging, or obesity) is associated with increased cancer risk. Exercise/exercise training has the potential (especially in conjunction with dietary control) to limit body fat accumulation and favorably alter lipid profiles to one that is associated with reduced inflammation. In addition, exercising muscles release myokines (specific cytokines released in response to muscle contraction), including IL-6. IL-6, in turn, stimulates the production of IL-1 receptor antagonists blocking the proinflammatory actions of IL-1 α and IL-1 β . This action contributes to the anti-inflammatory effects of exercise and the possible mediation of cancer ([Brandt and Pedersen, 2010](#); [Koelwyn et al., 2015](#); [Lancaster and Febbraio, 2014](#)).

Current evidence suggests that exercise/exercise training is a promising strategy to alter numerous components of the immune system and, in turn, the development and progress of cancer. Although there remain many important knowledge gaps, there is strong agreement that exercise training is an important component in the treatment and prognosis for cancer survivors ([Koelwyn et al., 2015](#); [Patel et al., 2019](#)).

By definition, cancer survivorship occurs from the time of diagnosis until the end of life. The continuum of cancer survivorship includes acute treatment and recovery, long-term disease-free or stable disease, and living with advanced cancer ([Rock et al., 2012](#); [Schmitz et al., 2010](#)). All major cancer treatments (surgery, chemotherapy, radiation, hormonal) have side effects that impact normal physiological functioning of the cardiorespiratory, neuromuscular-endocrine, immune, and gastrointestinal systems ([Schmitz et al., 2010](#)). Evidence strongly suggests that not only is exercise safe and feasible during cancer treatment, but that it can improve these impacted physiological functions and various aspects of quality of life. Generically then, the typical adaptations that occur with both aerobic and resistance exercise training in health-related physical fitness components (cardiorespiratory, body composition, and muscular

power/strength/endurance) are beneficial to most cancer survivors including those with advanced disease (Rock et al., 2012; Salakari et al., 2015). Specifically, two major symptoms that are associated with cancer treatment are tumor-induced cachexia (a multifactorial wasting syndrome including loss of muscle mass) and cancer-related fatigue. Recent literature indicates a role of chronic exercise in modulating cachexia (Bloch et al., 2013; Lira et al., 2015). Several systematic literature reviews and meta-analyses (Dennett et al., 2016; Meneses-Echavez et al., 2015; Tian et al., 2016) have shown that aerobic and resistance exercise training (with or without stretching) has a beneficial effect on the management of cancer-related fatigue both during secondary treatment (e.g., radiation after surgery) and after all treatment.

There is a consensus among the American Cancer Society (Rock et al., 2012), the American College of Sports Medicine (ACSM, 2022; Schmitz et al., 2010), and the U.S. Department of Health and Human Services (Physical Activities Guidelines Advisory Committee, 2008) that individuals with cancer should

1. Avoid inactivity and return to normal daily activities as soon as possible following diagnosis.
2. Engage in aerobic exercise, 150–300 min·wk⁻¹ if moderate intensity, or 75–150 min·wk⁻¹ if vigorous intensity.
3. Include resistance training exercises for all major muscle groups at least 2 d·wk⁻¹.
4. Integrate balance and flexibility exercises (stretching, Tai-chi, or yoga) on days that aerobic and resistance exercises are performed.

Obviously, exercise prescriptions should be individualized according to a cancer survivor's pretreatment fitness level, any additional preexisting medical complications, cancer site-specific considerations, response to treatment, and any persistent negative effects of treatment. Individuals unable to meet the goals listed above should be encouraged to be as physically active as the condition allows, keeping in mind that "some exercise is always better than no exercise."

More recommendations for specific cancers are listed in the

ACSM's Guidelines for Exercise Testing and Prescription (ACSM, 2022). In 2009, the American College of Sports Medicine in collaboration with the American Cancer Society developed a Certified Cancer Exercise Trainer program (<http://certification.acsm.org/acsm-cancer-exercise> trainer). A wide array of health care professionals as well as athletic trainers and other certified ACSM fitness providers may qualify to take the program.

Exercise, the Immune System, and AIDS

Acquired immune deficiency syndrome (AIDS) is a disease caused by the human immunodeficiency virus (HIV) and characterized by severe CD4 cell depletion. This virus infects the CD4-bearing T cells (helper T cells), which orchestrate much of the immune response of the body. As the infection progresses, there is greater depletion of the CD4 cells, leading ultimately to immunodysregulation (Brenner et al., 1994). HIV infection progresses through three stages (Centers for Disease Control and Prevention, 2016a, 2016b):

1. *Acute HIV infection*: Within 2–4 weeks after infection with HIV, the individual may/may not experience a flu-like illness (fever, headache, rash) that may last for a few weeks. During this stage, the virus infects relatively few CD4-bearing cells but multiplies and spreads throughout the body rapidly. It is not clinically detectable except by blood testing. These individuals are viral carriers and infectious despite the absence of outward signs of infection.
2. *Clinical latency (HIV inactivity or dormancy)*: This stage is sometimes called *asymptomatic HIV infection* or *chronic HIV infection*. Infected individuals may/may not have any symptoms, and even if they are not taking any medications, this period can last up to 10 years. Individuals taking highly active antiretroviral therapy (HAART) medications can live in this stage for several decades. HIV continues to multiply but at very low levels. HIV-positive individuals in this stage can infect others although those consistently taking HAART medications are less likely to do so. At the end of this phase, the viral count starts to go up and the CD4 cell count to go down. Symptoms are likely to occur.

3. **AIDS:** Severe CD4 cell depletion occurs along with major complications resulting from opportunistic infection (e.g., pneumonia or tuberculosis) or cancer malignancies. At this stage of the disease, individuals can be very infectious.

Fortunately, HIV infection rates are decreasing. Over the past decade, the number of individuals living with HIV has increased, while the annual number of new HIV infections has declined. In 2019, the estimated number of HIV infections in the United States was 34,800 (CDC.gov).

The use of highly active antiretroviral therapy (HAART) has dramatically changed the treatment of HIV and AIDS, significantly lessening the mortality and opportunistic infections. As a result of this new treatment, HIV infection is now treated as a chronic illness, although it remains a life-threatening disease (Quiles and Garber, 2014). The downside is that HAART treatment results in serious adverse effects, including headache, fatigue, nausea, vomiting, disorders of glucose metabolism, lipodystrophy syndrome (abnormal fat redistribution and metabolic disturbances), damage to the liver, bone metabolism abnormalities, and increase in cardiovascular disease. Exercise is a possible management strategy for many of these problems, but it is often difficult to maintain an exercise program in face of these challenges. Systematic reviews and meta-analyses have shown that aerobic and resistance training confers benefit to individuals with HIV and AIDS either singly or in combination, with beneficial effects for cardiorespiratory fitness, improved lipid and glucose levels, increased strength and flexibility, and decreased accumulation of fat in the trunk area of HIV-positive individuals. Importantly, the training has been found to be both safe and effective even during HAART treatment and does not appear to have any adverse effects on the treatment effectiveness (Gomes-Neto et al., 2013, 2015; O'Brien et al., 2004; Quiles and Garber, 2014; Shephard, 2015).

Exercise prescription principles for healthy adults can generally apply to persons with HIV with necessary modifications according to their individual health status. The exercise training program should include cardiorespiratory activity ≥ 5 d·wk⁻¹ if moderate intensity or ≥ 3 d·wk⁻¹ if vigorous intensity. Two to three days per week of resistance training for the major muscles

groups and flexibility exercises for the major muscle-tendon units are also recommended (Quiles and Garber, 2014).

Summary

1. The immune system can be functionally divided into the innate and the adaptive branches. The innate branch protects against foreign substances or cells without having to recognize them. In the adaptive branch of the immune system, immune cells recognize a foreign material and react specifically and selectively to destroy it.
2. Moderate aerobic exercise leads to an increase in the number and activity of neutrophils; an increase in the number, percentage, and activity of NK cells; an increase in phagocytic activity and secretion of cytokines from macrophages; an increase in the number of acute phase proteins; and an increase in the number of B and T cells.
3. Exhaustive aerobic exercise is associated with a reduction in NK cell number and activity and a decrease in lymphocytes and neutrophils.
4. The depressed levels of NK cells following strenuous exercise are a likely explanation of the vulnerability to acute infection associated with high-level competition and chronic overtraining.
5. The neuroendocrine system and the immune system overlap considerably. Hormonal control of the immune response is mediated primarily by the action of epinephrine and cortisol.
6. A sustained mismatch between exercise training stress and inadequate recovery can lead to training maladaptation. Maladaptation occurs over a continuum. The least serious is functional overreaching (FOR). FOR refers to short-term performance decrement (days to weeks) and is often done intentionally during a shock microcycle of periodization. Nonfunctional overreaching (NFOR) is more severe with a performance decrement that lasts from weeks to months and usually is accompanied by severe signs and symptoms. The overtraining syndrome (OTS) is the most serious stage of the

continuum with the greatest disruption and a performance recovery that takes months to years.

7. Although no single marker has been identified to predict NFOR or OTS, it is important to monitor training load, performance, and mood state and ensure adequate rest/recovery and nutrition in an attempt to prevent maladaptation. The primary treatment is rest.
8. Severe exercise training is associated with immunosuppression and an increased risk of URTI.
9. Physical activity is associated with lower prevalence and mortality rates for at least thirteen cancers including breast, bladder, and colon. Exercise training is important for individuals at all stages of cancer survivorship regardless of the type of cancer involved.
10. Exercise training appears to be beneficial to individuals infected with HIV.

Review Questions

1. Graphically present the two branches of the immune system, emphasizing how they work together.
2. Identify the primary cells of the innate and adaptive branches of the immune system, and indicate the mechanisms by which each leads to antigen destruction.
3. Describe the sequence of events in inflammation.
4. Describe the immune response to moderate aerobic exercise and to a severe exercise bout.
5. Differentiate between the training adaptations of the immune system to a moderate training program and to overtraining.
6. Describe the continuum for training maladaptation. Discuss the current hypotheses that explain overtraining. What factors should be monitored to attempt to prevent nonfunctional overreaching and the overtraining syndrome?
7. Describe the proposed relationship between exercise and the incidence of upper respiratory tract infection (URTI), and

- discuss evidence that exercise may not be related to URTI.
8. Describe the relationship between physical activity levels, immune function, and age.
 9. What is the role of exercise training for cancer survivors?
 10. Describe the role and benefits of physical activity for an individual infected with HIV.

Literature Search

In this chapter, we discussed immune function and its relationship to exercise and exercise training, and how this impacts several health conditions. To explore this topic further, do a literature search using a search engine such as PubMed, Google Scholar, or Web of Science.

- a. Search exercise and immune function, this will yield a huge selection of articles.
- b. Refine your search using key terms that may reflect your interest in this area. For example,
 - i. Endurance exercise and immune function.
 - ii. Endurance exercise and training maladaptation.
 - iii. Exhaustive exercise and overreaching.
 - iv. Resistance exercise and immune function in individuals with diabetes mellitus.
 - v. Cytokine response to high intensity exercise.
 - vi. Exercise training and risk of upper respiratory tract infection.
 - vii. Exercise and cancer risk.
 - viii. Nutritional strategies to decrease risk of OTS.
 - ix. Continue your search for aspects of this topic that are of particular interest to you.

For further review and study tools, visit Lippincott Connect.

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Appendix A

Units of Measure, the Metric System, and Conversions between the English and Metric Systems of Measurement

TABLE A.1 SI Units (Système International) (Metric)

| Physical Quantity | Unit | Symbol |
|------------------------|---------------------------|-----------------------|
| Mass | kilogram | kg |
| Distance | meter | m |
| Volume (liquid or gas) | liter | L |
| Time | second | sec |
| Force | newton | N |
| Work | joule | J |
| Power | watt | W |
| Angle | radian | rad |
| Linear velocity | meters per second | m·sec ⁻¹ |
| Angular velocity | radians per second | rad·sec ⁻¹ |
| Temperature | degrees | ° |
| Torque | newton-meter | N·m |
| Acceleration | meters per second squared | m·sec ⁻² |
| Amount of substance | mole | mol |

TABLE A.2 SI Units (Système International) (Metric)

| Prefix* | Meaning | Scientific Notation | Symbol |
|----------------------|-----------------------------|---------------------|--------|
| Makes Smaller | | | |
| deci- | one tenth of (0.1) | 10^{-1} | d |
| centi- | one hundredth of (0.01) | 10^{-2} | c |
| milli- | one thousandth of (0.001) | 10^{-3} | m |
| micro- | one millionth of (0.000001) | 10^{-6} | μ |
| Makes Larger | | | |
| kilo- | a thousand times (1,000) | 10^3 | k |

* Most commonly used in this text.

TABLE A.3 Conversions Between the English and Metric Systems of Measurement

| Measurement | Unit and Abbreviation | Metric Equivalent | English-to-Metric Conversion Factor | Metric-to-English Conversion Factor |
|----------------------------|--|--|--|--|
| Length | 1 kilometer (km) | =1,000 meters | 1 mile = 1.61 km | 1 km = 0.62 mile |
| | 1 meter (m) | =100 centimeters | 1 yard = 0.914 m | 1 m = 1.09 yards |
| | 1 centimeter (cm) | =1,000 millimeters = 0.01 meter | 1 foot = 0.305 m 1 foot = 30.5 cm | 1 m = 3.28 feet 1 m = 39.37 inches |
| | 1 millimeter (mm) | =0.001 meter | 1 inch = 2.54 cm | 1 cm = 0.394 inch |
| | 1 micrometer (μ m) | =0.000001 meter | | |
| Mass | 1 kilogram (kg) | =1,000 grams | 1 pound = 0.454 kg | 1 kg = 2.205 pounds |
| | 1 gram (g) | =1,000 milligrams | 1 ounce = 28.35 g | 1 g = 0.035 ounce |
| | 1 milligram (mg) | =0.001 gram | | |
| Volume (liquids and gases) | 1 liter (L) | =1,000 milliliters | 1 quart = 0.946 L 1 quart = 946 mL 1 gallon = 3.785 L | 1 L = 0.264 gallon 1 L = 1.057 quarts |
| | 1 milliliter (mL) | =0.001 liter | | 1 mL = 0.034 fluid ounce |
| | 1 microliter (μ L) | =1 cubic centimeter = 0.000001 liter | 1 pint = 473 mL 1 fluid ounce = 29.57 mL | |
| | 1 square meter (m ²) | =10,000 square centimeters | 1 square yard = 0.836 m ² | 1 m ² = 1.196 square yards |
| | 1 square centimeter (cm ²) | =100 square millimeters | 1 square inch = 6.452 cm ² | 1 cm ² = 0.155 square inch |
| Temperature | Degree Celsius ($^{\circ}$ C) | | $^{\circ}$ C = 5/9 ($^{\circ}$ F – 32) | $^{\circ}$ F = [(9/5) $^{\circ}$ C] + 32 |
| Force | 1 newton (N) | =0.1019 kilopond (kp) | 1 ft-lb-sec ⁻¹ = 0.138 N | |
| Linear velocity | 1 (m-sec ⁻¹) | | 1 mi-hr ⁻¹ = 26.8 m-min ⁻¹ | |
| Angular velocity | 1 radian per second (rad-sec ⁻¹) | | | 1 rad-sec ⁻¹ = 57.3 degrees-sec ⁻¹ |
| Work and energy | 1 joule (J) | =1 N-m | | 1 J = 0.738 ft-lb |
| | 1 kcal | =426.85 kpm = 4.18 kJ | | 1 J = 0.239 cal |
| | 1 kpm | =1 kpm = 0.00234 kcal | | |
| | 1 watt (W) | 1 W = 1 joule per sec (J-sec ⁻¹) 1 W = 6.12 kpm-min ⁻¹ | 1 horsepower (hp) = 745.7 W | 1 W = 0.0013 hp |
| Pressure | 1 newton per square meter (N-m ⁻²) | | 1 mmHg = 133.32 N-m ⁻² 1 atmosphere = 760 mmHg | 760 mmHg = 29.92 inches |

Appendix B

Metabolic Calculations

This appendix describes three methods used to calculate oxygen consumption ($\dot{V}O_{2\text{cons}}$). The first series of equations involves direct calculations using data obtained from open-circuit spirometry. This technique also allows for the calculation of carbon dioxide produced ($\dot{V}O_{2\text{prod}}$), which the other two, because they are indirect techniques, do not. The second series of equations calculates submaximal oxygen consumption values for selected activities (walking, running, cycle ergometer riding, and stair stepping) using known rates of work. The third set of calculations estimates maximal oxygen consumption ($\dot{V}O_{2\text{max}}$) from selected field tests (the PACER, or 20-m shuttle test, and the Rockport Fitness Walking Test [RFWT]).

The Calculation of Oxygen Consumed and Carbon Dioxide Produced from Data Obtained by Open-Circuit Spirometry

Basic Formulas

Theoretically, the amount of oxygen consumed is simply equal to the amount of oxygen in the inspired air minus the amount of oxygen in the expired air. All values are expressed in $\text{mL}\cdot\text{min}^{-1}$ or $\text{L}\cdot\text{min}^{-1}$:

oxygen consumption ($\text{L} \cdot \text{min}^{-1}$) = amount of
oxygen inspired ($\text{L} \cdot \text{min}^{-1}$) – amount of
oxygen expired ($\text{L} \cdot \text{min}^{-1}$)

$$\text{B.1} \quad \dot{V}\text{O}_2\text{cons} = \dot{V}_I\text{O}_2 - \dot{V}_E\text{O}_2$$

In practice, there is no way to obtain $\dot{V}_I\text{O}_2$ or $\dot{V}_E\text{O}_2$ directly, so a working formula is used. The working formula is based on the fact that the amount of a gas depends on the fraction (F) of the gas and the volume of the air containing that gas.

oxygen consumption ($\text{L} \cdot \text{min}^{-1}$) = [fraction of
oxygen in inspired air \times volume of inspired air
($\text{L} \cdot \text{min}^{-1}$)] – [fraction of oxygen in expired air
 \times volume of expired air ($\text{L} \cdot \text{min}^{-1}$)]

$$\text{B.2} \quad \dot{V}\text{O}_2\text{cons} = (F_I\text{O}_2 \times \dot{V}_I) - (F_E\text{O}_2 \times \dot{V}_E)$$

Although the term *fraction* and the symbol F are always used in this equation, these values really represent the percentages of oxygen in inspired or expired air, and they are expressed mathematically as decimals. Thus, $F_I\text{O}_2$ is a constant 20.93%, or 0.2093. The inspired ventilation values either can be directly measured by a spirometer or pneumoscan, as described in Chapters 4 and 9, or can be calculated.

The $F_E\text{O}_2$ is measured by an oxygen analyzer, as described in Chapter 4. As with the inspired ventilation, expired ventilation can be either directly measured by a spirometer or pneumoscan or can be calculated. Either \dot{V}_I or \dot{V}_E must be directly measured. When the value of one of them is known, the other can be calculated.

Ventilation Conversions

As stated in Chapter 9, the volume of air (either inspired or expired) is collected under conditions known as ambient temperature and pressure saturated, or ATPS. To do metabolic calculations, air volumes must first be converted to standard

temperature and pressure (STPD) values. This conversion is necessary so that the number of gas molecules in any given volume is equal. The equations vary slightly according to whether the measured volume is inspired or expired.

The conversion process is based on the impact of temperature, pressure, and water vapor molecules on volume. The effect of temperature on volume is described by Charles' law. *Charles' law* states that the volume of a gas is directly related to temperature assuming a constant pressure, that is,

$$\frac{T_1}{T_2} = \frac{V_1}{V_2}$$

Therefore, if the initial temperature (T_1) is increased (T_2), the initial volume (V_1) will also be increased (V_2). Conversely, if T_1 is decreased at T_2 , then V_1 will also be decreased at V_2 . In metabolic calculations, V_2 is the value that is unknown. Therefore, the working formula that takes into account the impact of temperature on volume becomes

$$V_2 \times T_1 = V_1 \times T_2 \quad \text{or} \quad V_2 = \frac{V_1 \times T_2}{T_1}$$

usually expressed as

$$V_2 = V_1 \left(\frac{T_2}{T_1} \right)$$

The effect of pressure on volume is primarily described by

Boyle's law. *Boyle's law* states that the volume of a gas is inversely related to pressure assuming a constant temperature, that is,

$$\frac{P_1}{P_2} = \frac{V_2}{V_1}$$

Therefore, if the initial pressure (P_1) is increased (P_2), the initial volume (V_1) will be decreased (V_2). Conversely, if P_1 is decreased at P_2 , then V_1 will be increased at V_2 . Because V_2 is again the value that is unknown, this formula rearranges to

$$V_2 \times P_2 = V_1 \times P_1 \quad \text{or} \quad V_2 = \frac{V_1 \times P_1}{P_2}$$

usually expressed as

$$V_2 = V_1 \left(\frac{P_1}{P_2} \right)$$

Water vapor molecules evaporate into the air (or into other gases) and account for part of the pressure exerted by the gas. The amount of water vapor is related exponentially to temperature. If the temperature is constant and a gas goes from being saturated with water vapor (S) to dry (D), the volume of the gas decreases. Conversely, if a gas goes from D to S, the volume increases. Tables, such as the abbreviated version in

Table B.1, are available to determine the water vapor pressure (PH₂O) at measured ambient temperatures.

TABLE B.1 Water Vapor Pressure (PH₂O) at Selected Ambient Temperatures

| Ambient Temperature (°C) | Ambient Temperature (°F) | PH ₂ O (mmHg) |
|--------------------------|--------------------------|--------------------------|
| 20 | 68 | 17.5 |
| 21 | 70 | 18.7 |
| 22 | 72 | 19.8 |
| 23 | 73 | 21.1 |
| 24 | 75 | 22.4 |
| 25 | 77 | 23.8 |

In practice, the formulas just described are generally combined so that the effects of temperature and volume are calculated concurrently, as indicated in the following formula:

$$V_2 = V_1 \left(\frac{T_2}{T_1} \right) \left(\frac{P_1}{P_2} \right)$$

B.3

The order of the temperature and pressure components in this equation may be reversed. If necessary, pressure is adjusted for water vapor.

Table B.2 presents the factors that are used in converting ventilatory volumes from ATPS to BTPS or STPD using expired and inspired ventilation volumes. BTPS ventilations are not used

in the calculation of $\dot{V}O_2$ consumed and $\dot{V}CO_2$ produced, but they are presented for completeness and because BTPS values are used in determining ventilatory thresholds ([Chapter 10](#)) and lung volumes ([Chapter 9](#)).

TABLE B.2 Ventilation Conversion Factors

| Volume | Pressure | Temperature |
|------------------|--|-------------------------|
| \dot{V}_E ATPS | $P_B - PH_2O$ at $T^\circ C$ | $273^\circ + T^\circ C$ |
| \dot{V}_I ATPS | $P_B - [RH \times PH_2O$ at $T^\circ C]$ | $273^\circ + T^\circ C$ |
| \dot{V}_E STPD | 760 | 273° |
| \dot{V}_E BTPS | $P_B - 47$ | $273^\circ + 37^\circ$ |

P_B , measured barometric pressure in mmHg; PH_2O , water vapor pressure in mmHg; $T^\circ C$, measured temperature in degrees Celsius; RH, relative humidity as a fraction; $273^\circ K = 0^\circ C$, standard temperature; $37^\circ C$, normal body temperature; 47 mmHg, water vapor pressure at $37^\circ C$.

The Conversion of \dot{V}_E from ATPS to BTPS

When any ventilation volume is converted, it is first important to identify P_1 , P_2 , T_1 , and T_2 . Thus, in converting from \dot{V}_E ATPS to \dot{V}_E BTPS, P_1 is the ambient pressure; P_2 is the body pressure, which equals barometric pressure; T_1 is the ambient temperature; and T_2 is the body temperature. Second, the factor representing each of these components should then be identified from **Table B.2**: that is, $P_1 = P_B - PH_2O$ at $T^\circ C$; $P_2 = P_B - 47$; $T_1 = 273 + T^\circ C$; and $T_2 = 273 + 37^\circ C$. This 273 is $273^\circ K$, to which either the measured temperature ($T^\circ C$) or the normal body temperature ($37^\circ C$) is added. Ambient pressure is the measured barometric pressure at the data collection site corrected for the water vapor pressure at the ambient temperature. **Table B.1** is used to determine the water vapor pressure. Body pressure is the measured barometric pressure corrected for water vapor pressure at body temperature. Normal body temperature is assumed to be $37^\circ C$. Water vapor pressure (PH_2O) at $37^\circ C$ is 47 mmHg.

Based on [Equation B.3](#), the conversion formula becomes

minute ventilation(L · min⁻¹)BTPS = minute
ventilation(L · min⁻¹)ATPS × temperature
correction(°Kelvin) × pressure correction(mmHg)
or

$$\text{B.4} \quad \dot{V}_E \text{BTPS} = \dot{V}_E \text{ATPS} \left(\frac{273^\circ + 37^\circ \text{C}}{273^\circ + T^\circ \text{C}} \right) \left(\frac{P_B - \text{PH}_2\text{O at } T^\circ \text{C}}{P_B - 47} \right)$$

For example, given the following information, we can correct \dot{V}_E from ATPS to BTPS conditions:

$$\dot{V}_E \text{ATPS} = 12 \text{ L} \cdot \text{min}^{-1}$$

$$T \text{ATPS} = 22^\circ \text{C}$$

$$P_B = 745 \text{ mmHg}$$

$$\dot{V}_E \text{BTPS} =$$

$$12 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^\circ + 37^\circ \text{C}}{273^\circ + 22^\circ \text{C}} \right) \left(\frac{745 \text{ mmHg} - 19.8 \text{ mmHg}}{745 - 47 \text{ mmHg}} \right)$$

$$\dot{V}_E \text{BTPS} = 13.1 \text{ L} \cdot \text{min}^{-1}$$

Under typical ambient conditions (without extremely high heat or altitude), BTPS values will be larger numerically than ATPS values because body temperature is usually higher than ambient temperature (which increases volume). Also, the body pressure adjusted for water vapor pressure at body temperature is typically lower than the ambient pressure adjusted for water vapor pressure at ambient temperature (which also increases volume).

The Conversion of \dot{V}_E from ATPS to STPD

In converting from $\dot{V}_E \text{ATPS}$ to $\dot{V}_E \text{STPD}$, we identify P_1 as ambient pressure, P_2 as standard pressure, T_1 as ambient temperature, and T_2 as standard temperature. Identifying each of these components from **Table B.2** results in the following: $P_1 = P_B - \text{PH}_2\text{O at } T^\circ \text{C}$, $P_2 = 760$, $T_1 = 273^\circ + T^\circ \text{C}$, and $T_2 = 273^\circ \text{C}$. The body (barometric) pressure must be adjusted for the water vapor pressure at the ambient temperature so that the conversion goes from saturated or wet air to unsaturated or dry

conditions. The formula for converting $\dot{V}_E \text{ ATPS}$ to $\dot{V}_E \text{ STPD}$ accounts for changes in temperature (T) and pressure (P) as follows:

minute ventilation ($\text{L} \cdot \text{min}^{-1}$) STPD = minute ventilation ($\text{L} \cdot \text{min}^{-1}$) ATPS \times temperature correction ($^{\circ}\text{Kelvin}$) \times pressure correction (mmHg)
or

$$\text{B.5} \quad \dot{V}_E \text{ STPD} = \dot{V}_E \text{ ATPS} \left(\frac{273^{\circ}}{273^{\circ} + T^{\circ}\text{C}} \right) \left(\frac{P_B - \text{PH}_2\text{O at } T^{\circ}\text{C}}{760} \right)$$

In this equation, the 273 represents the standard temperature of 0°C , which equals 273°K . The $T^{\circ}\text{C}$ represents the ambient temperature in degrees Celsius. PH_2O represents the water vapor correction from **Table B.1** to the body or barometric pressure (P_B), and 760 mmHg is standard pressure. Thus, given the

following information, we can correct \dot{V}_E from ATPS to STPD conditions.

$$\dot{V}_E \text{ ATPS} = 12 \text{ L} \cdot \text{min}^{-1}$$

$$T \text{ ATPS} = 22^{\circ}\text{C}$$

$$P_B = 745 \text{ mmHg}$$

$$\dot{V}_E \text{ STPD} =$$

$$12 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^{\circ}}{273^{\circ} + 22^{\circ}\text{C}} \right) \left(\frac{745 \text{ mmHg} - 19.8 \text{ mmHg}}{760 \text{ mmHg}} \right)$$

$$\dot{V}_E \text{ STPD} = 10.6 \text{ L} \cdot \text{min}^{-1}$$

The Conversion of \dot{V}_I from ATPS to STPD

The conversion of \dot{V}_I from ATPS to $\dot{V}_I \text{ STPD}$ is the same as from $\dot{V}_E \text{ ATPS}$ to $\dot{V}_E \text{ STPD}$, with one small addition: in most laboratory settings, the inspired air is not totally saturated; that is, the relative humidity is not 100%. The equation must therefore be modified to adjust for the measured relative humidity (RH), expressed as a decimal fraction at the ambient

temperature. Thus, the conversion equation becomes

$$\begin{aligned} \dot{V}_{\text{STPD}} = \\ \text{B.6} \quad \dot{V}_{\text{ATPS}} \left(\frac{273^\circ}{273^\circ + T^\circ\text{C}} \right) \left(\frac{P_B - [\text{RH} \times \text{PH}_2\text{O at } T^\circ\text{C}]}{760} \right) \end{aligned}$$

Again, **Table B.1** presents the water vapor pressures (PH₂O) at temperatures generally encountered in a laboratory (20–25°C or 67–77°F). Thus, under the conditions given in the previous example but with an RH of 46%, the calculations become

$$\begin{aligned} \dot{V}_{\text{STPD}} &= 12 \text{ L} \cdot \text{min}^{-1} \\ &\left(\frac{273^\circ}{273^\circ + 22^\circ} \right) \left(\frac{745 \text{ mmHg} - [0.46 \times 19.8 \text{ mmHg}]}{760 \text{ mmHg}} \right) \\ \dot{V}_{\text{STPD}} &= 10.8 \text{ L} \cdot \text{min}^{-1} \end{aligned}$$

Under typical ambient conditions (without extremely high heat or altitude), STPD values, whether derived from expired or inspired ventilation volumes, are smaller numerically than ATPS values. The reason is that standard temperature is usually lower than ambient temperature (which decreases volume), and standard pressure is usually higher than ambient pressure (which decreases volume). The number of gas molecules depends on the volume they occupy. Therefore, STPD volumes are used when it is important to know the number of gas molecules, as in the calculation of O₂ consumed and CO₂ produced. The number of gas molecules and the volume they occupy under STPD conditions are constant and independent of the particular gas involved.

The Conversion of \dot{V}_I from BTPS to STPD and Vice Versa

Occasionally, it is necessary to convert between BTPS and STPD, often based on which values any particular software-driven printout is programmed to provide. When BTPS is converted to STPD, P₁ is body or barometric pressure, P₂ is standard pressure,

T1 is body temperature, and T2 is standard temperature. From **Table B.2**, these factors are identified as $P_1 = P_B - 47$, $P_2 = 760$, $T_1 = 273^\circ + 37^\circ\text{C}$, and $T_2 = 273^\circ$. Substituting these into the generic **Equation B.3**,

$$V_2 = V_1 \left(\frac{T_2}{T_1} \right) \left(\frac{P_1}{P_2} \right)$$

we get

$$\begin{aligned} \dot{V}_E \text{STPD} = \\ \text{B.7} \quad \dot{V}_E \text{BTPS} \left(\frac{273^\circ}{273^\circ + 37^\circ\text{C}} \right) \left(\frac{P_B - 47 \text{ mmHg}}{760 \text{ mmHg}} \right) \end{aligned}$$

Using the values from our examples,

$$\begin{aligned} \dot{V}_E \text{ BTPS} &= 13.1 \text{ L} \cdot \text{min}^{-1} \\ P_B \text{ STPD} &= 745 \text{ mmHg} \end{aligned}$$

we can calculate $\dot{V}_E \text{STPD}$ as

$$\begin{aligned} \dot{V}_E \text{STPD} &= \\ 13.1 \text{ L} \cdot \text{min}^{-1} &\left(\frac{273^\circ}{273^\circ + 37^\circ\text{C}} \right) \left(\frac{745 \text{ mmHg} - 47 \text{ mmHg}}{760 \text{ mmHg}} \right) \\ \dot{V}_E \text{STPD} &= 10.6 \text{ L} \cdot \text{min}^{-1} \end{aligned}$$

Conversely, when STPD is converted to BTPS, P_1 is standard pressure (760 mmHg), P_2 is body pressure ($P_B - 47$ mmHg), T_1 is standard temperature (0°C or 273°K), and T_2 is body

temperature (assumed to be $273^{\circ} + 37^{\circ}\text{C}$). Substituting these values into the generic equation, we get

$$\text{B.8} \quad \dot{V}_{\text{E}}\text{BTPS} = \dot{V}_{\text{E}}\text{STPD} \left(\frac{273^{\circ} + 37^{\circ}\text{C}}{273^{\circ}} \right) \left(\frac{760 \text{ mmHg}}{P_{\text{B}} - 47 \text{ mmHg}} \right)$$

Again, using the values from our example,

$$\begin{aligned} \dot{V}_{\text{E}}\text{STPD} &= 10.6 \text{ L} \cdot \text{min}^{-1} \\ P_{\text{B}} &= 745 \text{ mmHg} \\ \dot{V}_{\text{E}}\text{BTPS} &= 10.6 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^{\circ} + 37^{\circ}\text{C}}{273^{\circ}} \right) \left(\frac{760 \text{ mmHg}}{745 \text{ mmHg} - 47 \text{ mmHg}} \right) \\ \dot{V}_{\text{E}}\text{BTPS} &= 13.1 \text{ L} \cdot \text{min}^{-1} \end{aligned}$$

Calculation of the Unknown Ventilation Value

The Calculation of \dot{V}_{E} from \dot{V}_{I} and \dot{V}_{I} from \dot{V}_{E}

Open-circuit metabolic systems measure either inspired or expired air, but not both. Typically, the volume of expired air does not equal the volume of inspired air. The reason is that the number of oxygen molecules used from the inspired air is not replaced by the same number of carbon dioxide molecules, except when an individual is burning pure carbohydrate and has an RER of exactly 1.0. When fewer CO_2 molecules replace the O_2 molecules, the RER value is less than 1.0, and \dot{V}_{E} will be smaller than \dot{V}_{I} . If the RER value is greater than 1.0, \dot{V}_{E} will be larger than \dot{V}_{I} . This occurs when both metabolic and nonmetabolic CO_2 molecules (primarily from the buffering of lactic acid) are

produced.

In order to solve Equation B.2 (i.e., calculate $\dot{V}O_2 \text{ cons}$), we must have values for both \dot{V}_E and \dot{V}_I . Fortunately, there is a way to convert \dot{V}_E to \dot{V}_I or \dot{V}_I to \dot{V}_E mathematically. This is called the *Haldane transformation* and is based on the fact that nitrogen (N₂) is an inert gas that does not participate in human metabolism, nor does it easily combine with any blood constituents. The number of particles of nitrogen does not change, although the concentration or fraction of the nitrogen will change because the fractions of oxygen and carbon dioxide are changing. Thus, the amount (again defined as the fraction of the gas times the volume of air containing that gas) of gaseous nitrogen expired is exactly equal to the amount of gaseous nitrogen inspired. That is,

fraction of inspired nitrogen \times the inspired
minute ventilation = fraction of expired
nitrogen \times the expired minute ventilation
or

$$\text{B.9} \quad F_{I\text{N}_2} \times \dot{V}_I = F_{E\text{N}_2} \times \dot{V}_E$$

Rearranging the equation, it becomes

$$\dot{V}_I = \frac{F_{E\text{N}_2} \times \dot{V}_E}{F_{I\text{N}_2}} \quad \text{or} \quad \dot{V}_E = \frac{F_{I\text{N}_2} \times \dot{V}_I}{F_{E\text{N}_2}}$$

$$\text{B.10} \quad \dot{V}_I = \dot{V}_E \left(\frac{F_{E\text{N}_2}}{F_{I\text{N}_2}} \right) \quad \text{and} \quad \dot{V}_E = \dot{V}_I \left(\frac{F_{I\text{N}_2}}{F_{E\text{N}_2}} \right)$$

As usual in these formulas, the measurements are percentages, but they are expressed as decimal fractions. Both $F_{I\text{N}_2}$ and $F_{E\text{N}_2}$ are easily obtained from known and measured values. Air is composed of nitrogen, oxygen, and carbon dioxide, and the

fractions of O₂ and CO₂ are known for room air (assumed to be the inspired air) and measured for expired air by the gas analyzers. Thus, N₂ can be calculated by simple subtraction:

$$F_I N_2 = 1 - [F_I O_2 + F_I CO_2] = 0.7904 \\ 0.2093 + 0.0003$$

and

$$F_E N_2 = 1 - [F_E O_2 + F_E CO_2] = \text{variables} \\ \text{measured by analyzers}$$

Therefore,

$$\dot{V}_I = \dot{V}_E \left(\frac{1 - [F_E O_2 + F_E CO_2]}{0.7904} \right) \quad \text{B.11}$$

$$\dot{V}_E = \dot{V}_I \left(\frac{0.7904}{1 - [F_E O_2 + F_E CO_2]} \right) \quad \text{B.12}$$

For example, given

$$\dot{V}_{STPD} = 10.8 \text{ L} \cdot \text{min}^{-1}$$

$$O_2 \% = 16.38$$

$$CO_2 \% = 4.03$$

the computation becomes

$$\begin{aligned}\dot{V}_{E\text{STPD}} &= 10.8 \text{ L} \cdot \text{min}^{-1} \left(\frac{0.7904}{1 - [0.1638 + 0.0403]} \right) \\ &= 10.73 \text{ L} \cdot \text{min}^{-1}\end{aligned}$$

Conversely, if we are given

$$\begin{aligned}\dot{V}_{E\text{STPD}} &= 10.6 \text{ L} \cdot \text{min}^{-1} \\ \text{O}_2\% &= 16.38 \\ \text{CO}_2\% &= 4.03\end{aligned}$$

the computation becomes

$$\begin{aligned}\dot{V}_{I\text{STPD}} &= 10.6 \text{ L} \cdot \text{min}^{-1} \left(\frac{1 - [0.1638 + 0.0403]}{0.7904} \right) \\ &= 10.67 \text{ L} \cdot \text{min}^{-1}\end{aligned}$$

Calculation of Oxygen Consumed

Now we are finally ready to go back to [Equation B.2](#) and solve it.

$$\dot{V}\text{O}_2 \text{ L} \cdot \text{min}^{-1} = (\dot{F}_I \text{O}_2 \times \dot{V}_I) - (\dot{F}_E \text{O}_2 \times \dot{V}_E)$$

The available information is as follows:

$$\begin{aligned}
 F_{\text{I}}\text{O}_2 &= 0.2093 (\text{assumed constant}) \\
 \dot{V}_{\text{I}}\text{STPD} &= 10.8 \text{ L} \cdot \text{min}^{-1} (\text{measured as } \dot{V}_{\text{I}}\text{ATPS by} \\
 &\quad \text{pneumscan and converted by Eq. B.6}) \\
 F_{\text{E}}\text{O}_2 &= 0.1638 (\text{measured by oxygen analyzer}) \\
 \dot{V}_{\text{E}}\text{STPD} &= 10.73 \text{ L} \cdot \text{min}^{-1} (\text{calculated by the Haldane} \\
 &\quad \text{transformation in Eq. B.12})
 \end{aligned}$$

Substituting the given values into [Equation B.2](#), we get

$$\begin{aligned}
 \dot{V}\text{O}_2 \text{ L} \cdot \text{min}^{-1} &= (0.2093 \times 10.8 \text{ L} \cdot \text{min}^{-1}) \\
 &\quad - (0.1638 \times 10.73 \text{ L} \cdot \text{min}^{-1}) \\
 &= 0.5 \text{ L} \cdot \text{min}^{-1} \text{ or } 500 \text{ mL} \cdot \text{min}^{-1}
 \end{aligned}$$

Calculation of Carbon Dioxide Produced

Theoretically, the amount of carbon dioxide produced is simply equal to the amount of carbon dioxide in expired air minus the amount of carbon dioxide in inspired air:

$$\text{B.13} \quad \dot{V}_{\text{I}}\text{CO}_2 \text{ prod} = \dot{V}_{\text{E}}\text{CO}_2 - \dot{V}_{\text{I}}\text{CO}_2$$

Because there is no way to obtain $\dot{V}_{\text{E}}\text{CO}_2$ or $\dot{V}_{\text{I}}\text{CO}_2$ directly, a working formula must again be used. Thus, the calculation of the amount of carbon dioxide produced is very similar to the calculation of oxygen consumed.

carbon dioxide produced ($\text{L} \cdot \text{min}^{-1}$) = [fraction of carbon dioxide in expired air \times volume of expired air ($\text{L} \cdot \text{min}^{-1}$)] – [fraction of inspired carbon dioxide \times volume of inspired air ($\text{L} \cdot \text{min}^{-1}$)].

$$\text{B.14} \quad \dot{V}_{\text{I}}\text{CO}_2 \text{ L} \cdot \text{min}^{-1} = (F_{\text{E}}\text{CO}_2 \times \dot{V}_{\text{E}}) - (F_{\text{I}}\text{CO}_2 \times \dot{V}_{\text{I}})$$

The $F_{\text{I}}\text{CO}_2$ is a constant 0.0003 because the percentage of carbon

dioxide in room air is assumed to be 0.03%. Because this number is so small and would have no meaningful effect on the calculation in [Equation B.14](#), it is generally considered to be 0. As a result, only the expired portion of the equation needs to be computed. $\dot{V}E$ is measured by a carbon dioxide analyzer as described in [Chapter 4](#). $\dot{V}E$ either is directly measured by a spirometer or pneumoscan or is calculated as previously described.

Using the values of CO₂% and $\dot{V}E$ given previously, the calculation becomes

$$\begin{aligned}\dot{V}CO_2 \text{ L} \cdot \text{min}^{-1} &= (0.0403 \times 10.73 \text{ L} \cdot \text{min}^{-1}) \\ &= 0.43 \text{ L} \cdot \text{min}^{-1} \text{ or } 430 \text{ mL} \cdot \text{min}^{-1}\end{aligned}$$

Note that as mentioned in the section describing the calculation of $\dot{V}E$ from $\dot{V}I$, the oxygen consumed (500 mL·min⁻¹) is not equaled by the amount of carbon dioxide produced (430 mL·min⁻¹).

Complete the practice problems at the end of the appendix to determine your understanding of these concepts and calculations.

The Calculation of Gross Energy Expenditure (Total Oxygen Consumed) Using Mechanical Work or Speed of Movement

In exercise situations where an accurate assessment of mechanical work or speed of movement is possible but the actual measurement of oxygen consumed and carbon dioxide produced is not, the oxygen consumed can be estimated. These situations include walking, running, cycling on an ergometer, and bench stepping. Specific formulas recommended by the [American College of Sports Medicine \(2022\)](#) are available for each activity. The resulting oxygen consumption values will not be as accurate

as the direct measurement of oxygen consumed, which was described in the last section. However, these estimated oxygen consumptions are much less difficult and much less expensive to obtain. They can be very useful in the practical field settings of health clubs, hospitals, and/or school gymnasias as the initial step in determining exercise prescriptions by MET level or determining the caloric cost of any activity. When using these equations to determine caloric expenditure, resting expenditure ($3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) should be subtracted first. Because of differences in economy ([Chapter 4](#)), none of these equations should be used for children. Because no systematic differences in economy have been found between males and females, all of these equations can be used for both sexes.

The following computations show you how to determine the oxygen consumption values of the four activities specified earlier. Remember from [Chapter 4](#) that converting to METs requires dividing the $\text{O}_2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ value by $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. Caloric cost (also discussed in [Chapter 4](#)) can be estimated by multiplying the $\text{O}_2 \text{ L}\cdot\text{min}^{-1}$ value by $5 \text{ kcal}\cdot\text{L}\cdot\text{min}^{-1}$, because $5 \text{ kcal}\cdot\text{L}\cdot\text{min}^{-1}$ is the estimated caloric equivalent if the RER is unknown.

Gross Energy Expenditure (Total Oxygen Consumed) during Horizontal (Level) and Vertical (Graded) Walking

Level Walking

To determine the oxygen consumed while walking on level ground or on a treadmill at 0% elevation, it is necessary to determine the walking speed in meters per minute ($\text{m}\cdot\text{min}^{-1}$). It is known that for speeds between 1.9 and $3.7 \text{ mi}\cdot\text{hr}^{-1}$ (or 50 – $100 \text{ m}\cdot\text{min}^{-1}$; $26.8 \text{ m}\cdot\text{min}^{-1} = 1 \text{ mi}\cdot\text{hr}^{-1}$), the net oxygen cost of level walking is $0.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ per $\text{m}\cdot\text{min}^{-1}$. Because this oxygen consumption constant is a net value, the oxygen consumed during rest (1 MET or $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) is added to obtain the total amount of oxygen consumed. Therefore, the equation can be stated as follows:

oxygen consumed during horizontal walking
 $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \text{horizontal}$
 component $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) + \text{resting}$
 component $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$
 or

$$\text{B.15} \quad \dot{V}_{\text{O}_2\text{cons}} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \left[\text{speed} (\text{m} \cdot \text{min}^{-1}) \times \frac{0.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

For example, if an individual were walking on a treadmill at 0% grade and 3 mi·hr⁻¹, it would first be necessary to convert the speed to meters per minute (3 mi·hr⁻¹ × 26.8 m·min⁻¹/mi·hr⁻¹ = 80.4 m·min⁻¹). Then, substituting this value into [Equation B.15](#), we get

$$\begin{aligned} \dot{V}_{\text{O}_2\text{cons}} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[80.4 (\text{m} \cdot \text{min}^{-1}) \right. \\ &\quad \left. \times \frac{0.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \dot{V}_{\text{O}_2\text{cons}} &= 11.54 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \end{aligned}$$

Graded Walking

To determine the oxygen consumed during uphill walking or walking on a treadmill at a grade, a vertical component is added to [Equation B.15](#). The vertical component is determined by the percent grade expressed as a decimal. Each m·min⁻¹ of vertical rise consumes an additional 1.8 mL·kg⁻¹·min⁻¹ for each m·min⁻¹ of speed.

The following formula incorporates the vertical component in [Equation B.15](#):

oxygen consumed during vertical walking

$(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$

= horizontal component $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$

+ resting component $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$

+ vertical component $(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$

or

$\dot{V}_{\text{O}_2\text{cons}} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \left[\text{speed} (\text{m} \cdot \text{min}^{-1}) \right.$

$\times \frac{0.1 \text{ mL} \times \text{kg}^{-1} \times \text{min}^{-1}}{\text{m} \times \text{min}^{-1}} \left. \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

$+ \left[\text{decimal grade} \times \text{speed} (\text{m} \cdot \text{min}^{-1}) \right.$

$\times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \left. \right]$

B.16

If we change the percent grade in the previous example from 0% to 5%, we now have only to calculate the vertical component.

vertical component = $\left[0.05 \times 80.4 \text{ m} \cdot \text{min}^{-1} \right.$

$\times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \left. \right] = 7.24 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

The vertical component is then added to the horizontal and resting components to get the total amount of oxygen consumed while walking at a speed of $3 \text{ mi} \cdot \text{hr}^{-1}$ with a 5% grade on a treadmill.

Horizontal component = $8.04 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

Resting component = $3.50 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

Vertical component = $7.24 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

Total $\dot{V}_{\text{O}_2\text{cons}} = 18.78 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$

Gross Energy Expenditure (Total Oxygen

Consumed) during Horizontal (Level) and Vertical (Graded) Running

The formulas for estimating the amount of oxygen consumed during running differ from those for walking only in two factors. The first is that the net oxygen cost of level running is $0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ per $\text{m} \cdot \text{min}^{-1}$, or double the same cost for walking. Conversely, the additional cost for the vertical components is only half as much as for walking, because of differences in the biomechanics of the two gaits, especially the greater forefoot push-off in running. This difference necessitates multiplying the vertical component by 0.5. Thus, $1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \times 0.5 = 0.9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

Running is defined as speeds greater than $5 \text{ mi} \cdot \text{hr}^{-1}$ ($134 \text{ m} \cdot \text{min}^{-1}$). This leaves a gap between the top walking speeds ($3.7 \text{ mi} \cdot \text{hr}^{-1}$ or $100 \text{ m} \cdot \text{min}^{-1}$) and the lowest running speed. The best formula for these intermediate speeds depends on whether the individual is actually running or walking.

The formula for determining the oxygen consumed while running on level ground or a treadmill is as follows:

$$\text{B.17} \quad \dot{V}\text{O}_{2\text{cons}}(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \left[\text{speed}(\text{m} \cdot \text{min}^{-1}) \times \frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Graded running adds a vertical component to [Equation B.17](#):

$$\text{B.18} \quad \dot{V}\text{O}_{2\text{cons}}(\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = \left[\text{speed}(\text{m} \cdot \text{min}^{-1}) \times \frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} + \left[\text{decimal grade} \times \text{speed}(\text{m} \cdot \text{min}^{-1}) \times \frac{0.9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right]$$

If the individual in the previous example switches from walking

at 3 mi·hr⁻¹ up a 5% grade to running the same grade at 7 mi·hr⁻¹ (188 m·min⁻¹), this example works out as follows:

$$\begin{aligned}\dot{V}O_{2\text{cons}} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[188 \text{ m} \cdot \text{min}^{-1} \right. \\ &\times \left. \frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ &+ \left[0.05 \times 188 \text{ m} \cdot \text{min}^{-1} \right. \\ &\times \left. \frac{0.9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right] \\ \text{Horizontal component} &= 37.60 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Resting component} &= 3.50 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Vertical component} &= 8.46 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Total } \dot{V}O_{2\text{cons}} &= \underline{49.56 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}\end{aligned}$$

Gross Energy Expenditure (Total Oxygen Consumed) during Cycling Using Either the Legs or the Arms

The formulas for calculating the amount of oxygen consumed during cycling are intended only for cycling on an ergometer, which permits the actual measurement of the workload. The mechanical work completed (as described in [Chapter 4](#)) is determined by multiplying the force exerted, in the form of the load or resistance overcome, in kilograms, by the distance traveled (m·min⁻¹) using the following equation:

$$\begin{aligned}\text{work rate} (\text{kgm} \cdot \text{min}^{-1}) &= \text{kg of resistance} \times \text{m} \cdot \text{rev}^{-1} \\ \text{B.19} \quad &\times \text{rev} \cdot \text{min}^{-1}\end{aligned}$$

The distance traveled per revolution is the circumference off the flywheel. For the often-used Monark, distance is 6 m; for the Tunturi and Bodyguard friction ergometers, this distance is 3 m. The pedaling rate, or revolutions per minute value, is typically displayed on the console of the ergometer and can vary from 30

to 110 rev·min⁻¹, with 50 or 60 rev·min⁻¹ being the most commonly used by fitness exercisers.

Leg Cycling

Oxygen consumed during leg cycling can be estimated using the following equation. This formula is most accurate when the work rate is between 300 and 1,200 kg·m·min⁻¹ (50–200 W), but it may be used up to rates of 4,200 kg·m·min⁻¹. The oxygen cost against the external load is equal to that of vertical walking, or 1.8 mL·kg⁻¹·min⁻¹ per m·min⁻¹, and the oxygen associated with unloaded cycling is 3.5 mL·kg⁻¹·min⁻¹. In the example below, this latter value has been added to the resting component to equal 7.0 mL·kg⁻¹·min⁻¹.

$$\begin{aligned} &\text{oxygen consumed during leg} \\ &\text{cycling (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = [\text{oxygen consumed during} \\ &\text{the resistance component (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) \\ &\text{divided by body weight (kg)}] + \text{oxygen} \\ &\text{consumed during the resting component} \\ &\text{(mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) + \text{oxygen associated with} \\ &\text{unloaded cycling} \\ &\text{or} \\ &\dot{V}\text{O}_2\text{cons (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) \\ &= \left[\frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times \text{work rate (kgm} \cdot \text{min}^{-1}) \right. \\ &\quad \left. \div \text{body weight (kg)} \right] + 7 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \end{aligned}$$

B.20

For example, if a 50-kg individual pedals a Monark bike at 60 rev·min⁻¹ at a load of 2 kg (denoted as 2 kp on the ergometer itself; 1 kg = 1 kp), he or she consumes 32.92 mL·kg⁻¹·min⁻¹, which is calculated as follows:

$$\begin{aligned}\dot{V}O_2 (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[\frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right. \\ &\quad \times (2 \text{ kg} \times 6 \text{ m} \cdot \text{rev}^{-1} \times 60 \text{ rev} \cdot \text{min}^{-1}) \div 50 \text{ kg} \Big] \\ &\quad + 7 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Resistance component} &= 25.92 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Rest + unloaded component} &= 7.00 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Total } \dot{V}O_{2\text{cons}} &= 32.92 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

Arm Cranking

Cycling with the arms is more properly referred to as arm cranking. The formula for arm cranking differs from leg cycling in the constant for oxygen use per kilogram of resistance. The arm musculature used in cranking is smaller than the leg musculature used in cycling; hence, there is no need for the addition of an unloaded cycling cost. However, additional muscles in the shoulders, back, and chest are recruited to stabilize the arms, elevating the oxygen cost. Thus, instead of a constant $1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ per $\text{m} \cdot \text{min}^{-1}$, the value used is $3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ per $\text{m} \cdot \text{min}^{-1}$. In addition, although some locations may modify a regular leg cycle ergometer for use with the arms, most places use a specialized arm crank ergometer. In this case, the flywheel size and thus distance are typically smaller. For the Monark arm ergometer, this value is 2.4 m. Hence, the workload and $\dot{V}O_2$ consumed will be lower at any given resistance and number of revolution per minute. This formula is appropriate for power outputs between 150 and $750 \text{ kgm} \cdot \text{min}^{-1}$ (25–125 W).

$$\begin{aligned}&\text{oxygen consumed during arm cranking } (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) \\ &= [\text{oxygen consumed during the resistance component} \\ &\quad (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \cdot \text{per } \text{m} \cdot \text{min}^{-1}) \text{ divided by body weight}] \\ &\quad + \text{oxygen consumed during rest} \\ \dot{V}O_{2\text{cons}} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[\frac{3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right. \\ &\quad \times \text{work rate } (\text{kgm} \cdot \text{min}^{-1}) \div \text{body weight (kg)} \Big] \\ &\quad + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

B.21 + 3.5 mL · kg⁻¹ · min⁻¹

If the individual in the previous example switches from leg cycling to arm cranking and uses an arm ergometer, he or she now consumes $20.78 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ of oxygen, calculated as follows:

$$\begin{aligned}\dot{V}\text{O}_2\text{cons} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[\frac{3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \right. \\ &\quad \times (2 \text{ kg} \times 2.4 \text{ m} \cdot \text{rev}^{-1} \times 60 \text{ rev} \cdot \text{min}^{-1}) \div 50 \text{ kg} \left. \right] \\ &\quad + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Resistance component} &= 17.28 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Resting component} &= 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ \text{Total } \dot{V}\text{O}_2\text{cons} &= 20.78 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

The Gross Energy Expenditure (Total Oxygen Consumed) during Bench Stepping

Like cycling, bench stepping allows for the exact computation of the work being done if the height of the step, the rate of stepping, and the body weight of the stepper are known.

oxygen consumed during bench stepping
($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) = [oxygen consumed during
the horizontal component ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)
+ oxygen consumed during the vertical
component ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)

B.22 + oxygen consumed at rest]

or

$$\begin{aligned}\dot{V}\text{O}_2\text{cons} (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[\frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{steps} \cdot \text{min}^{-1}} \times (\text{stepping rate})(\text{steps} \cdot \text{min}^{-1}) \right] \\ &\quad + \left[1.33 \times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times (\text{step height}) \right. \\ &\quad \times (\text{m} \cdot \text{step}^{-1}) \times (\text{stepping rate})(\text{steps} \cdot \text{min}^{-1}) \\ &\quad \left. + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \right]\end{aligned}$$

In the first component, $0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ is the oxygen cost of stepping back and forth along the horizontal plane. In the second component, as with walking or running, each $\text{m} \cdot \text{min}^{-1}$ of vertical rise requires $1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ of oxygen. The 0.33 in the constant 1.33 accounts for the fact that bench stepping has both a positive (up) action and a negative (down) action. Negative work in this situation requires one third as much oxygen as positive work. The equation is most accurate for step heights between 1.6 and 15.7 in (0.04–0.40 m) and step rates of 12 and 30 steps·min⁻¹.

For an individual stepping up and down on a 10-in (0.254-m) bench at 24 steps·min⁻¹, oxygen consumption is calculated as follows:

$$\begin{aligned} \dot{V}\text{O}_2 \text{ cons (mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) &= \left[24 \text{ steps} \cdot \text{min}^{-1} \right. \\ &\quad \times \left. \frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{steps} \cdot \text{min}^{-1}} \right] + \left[0.254 \text{ m} \cdot \text{step}^{-1} \right. \\ &\quad \times 24 \text{ steps} \cdot \text{min}^{-1} \times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times 1.33 \left. \right] \\ &\quad + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \end{aligned}$$

$$\text{Horizontal component} = 4.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

$$\text{Vertical component} = 14.6 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

$$\text{Resting component} = 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

$$\text{Total } \dot{V}\text{O}_2 \text{ cons} = \overline{22.9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}$$

The Calculation of Maximal Oxygen Consumption from Cardiovascular Endurance Field Tests

The direct measurement of maximal oxygen consumption (

$\dot{V}O_2 \text{ max}$) is the best indicator of cardiovascular respiratory fitness. However, the direct

measurement of $\dot{V}O_2 \text{ max}$ requires expensive equipment, trained technicians, and considerable time. This makes it unsuitable for mass testing. Therefore, as indicated in [Chapter 11](#), field tests are often used to estimate

$\dot{V}O_2 \text{ max}$. The calculation of

$\dot{V}O_2 \text{ max}$ from the 1-mi run/walk is explained in [Chapter 11](#). This section details the calculation of

$\dot{V}O_2 \text{ max}$ from the PACER (originally known as the 20-m shuttle test) and from the Rockport Fitness Walking Test (RFWT).

Pacer

Several different formulas are available to determine

$\dot{V}O_2 \text{ max}$ from the results of the PACER test. Two have been included here to cover as wide an age span as possible. The first equation should be used for both male and female children and adolescents ([Leger et al., 1988](#)). It requires knowing only the final speed at which the individual ran and the age of the individual. Level 1 (minute 1) is run at a speed of 8.5 km·hr⁻¹. Each additional level (min) adds 0.5 km·hr⁻¹.

$$\dot{V}O_2 \text{ max} = 31.025 + 3.238 (\text{final speed in km} \cdot \text{hr}^{-1}) \\ \text{B.23} \quad -3.248 (\text{age in yr}) + 0.1536 (\text{final speed} \times \text{age})$$

For example, if a 9-year-old boy had a final speed of 11 km·hr⁻¹, we would substitute into the equation as follows:

$$\dot{V}O_2 \text{ max} = 31.025 + 3.238(11) - 3.248(9) \\ + 0.1536(11 \times 9) = 52.62 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

The second equation should be used for young ≥ 18 -year adult males and females (Leger et al., 1988; Plowman and Liu, 1999). It requires knowing only the maximal speed the individual completed. As stated above, level 1 (minute 1) is run at a speed of 8.5 km·hr⁻¹. Each additional level (min) adds 0.5 km·hr⁻¹.

$$\text{B.24} \quad \dot{V}O_2 \text{ max} = -23.4 + 5.8 (\text{max speed in km} \cdot \text{hr}^{-1})$$

Thus, if a 20-year-old college student ran for 10 minutes, the calculation would be (13 km·hr⁻¹)

$$\dot{V}O_2 \text{ max} = -23.4 + 5.8(13 \text{ km} \cdot \text{hr}^{-1}) = 52 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Rockport Fitness Walking Test (RFWT)

$\dot{V}O_2 \text{ max}$

The RFWT can be used to estimate in male and female adults from approximately age 30–70 years (Kline et al., 1987) and adolescents 14–18 years (McSwegin et al., 1998), but not young adults.

Five variables are needed to estimate

$\dot{V}O_2 \text{ max}$

: body weight in pounds, age in years, sex (females are coded as 0 and males are coded as 1), time

for completion of the mile walk (in minutes, including a decimal), and heart rate in beats per minute (recorded by a heart rate monitor during the final quarter mile or manually in the 15-seconds immediately postexercise).

$$\begin{aligned} \dot{V}O_2 \text{ max} = & 132.853 - 0.0769 (\text{body weight})(\text{lb}) \\ & - 0.3877 (\text{age})(\text{yr}) + 6.3150 (\text{sex}) \\ & - 3.2649 (\text{walk time})(\text{min}) \\ \text{B.25} \quad & - 0.1565 (\text{heartrate})(\text{b} \cdot \text{min}^{-1}) \end{aligned}$$

For example, if a 64-year-old, 195-lb male completed the mile walk in 19.01 minutes with a last quarter heart rate of 113 b·min⁻¹, the calculation would be as follows:

$$\begin{aligned} \dot{V}O_2 \text{ max} = & 132.853 - 0.0769(195) - 0.3877(64) \\ & + 6.3150(1) - 3.2649(19.01) - 0.1565(113) \\ = & 19.61 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \end{aligned}$$

Standard Error of the Estimate

Because all of these formulas are estimates, knowing the accuracy of the estimate is helpful. Accuracy is determined during the development of the equations when the estimated values are compared to the actual values statistically and a standard error of the estimate (SEE) is obtained. The PACER equation for children and adolescents (Eq. B.23) has an SEE of 5.9 mL·kg⁻¹·min⁻¹. The PACER equation for young adults (Eq. B.24) has an SEE of 4.6–4.7 mL·kg⁻¹·min⁻¹. The RFWT equation (Eq. B.25) has an SEE of 5.0 mL·kg⁻¹·min⁻¹. Each SEE means that the calculated

$\dot{V}O_2 \text{ max}$ could differ from an actual measured value by one or two times the amount of the SEE. Most scores (68%) should vary only plus or minus one SEE from the calculated estimated value.

$\dot{V}O_2 \text{ max}$

Thus, the calculated for the college student from $52 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ Equation B.24 could actually be anywhere from 47.3 to 56.7 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. Obviously, the smaller the SEE, the greater the equation's accuracy (Jackson, 1989). All of these estimation equations are deemed to have acceptable accuracy.

Practice Problems

Use the data in **Table B.3** to work the problems that follow. Answers are provided at the end of the section. Because we are dealing with percentages (xx.xx) expressed as decimals (.xxxx), carry calculations out to four (4) decimals before any rounding.

TABLE B.3 Practice Problem Data

| Subjects | | | | |
|---|--------|--------|-------|-------|
| Variables | No. 1 | No. 2 | No. 3 | No. 4 |
| Descriptive Information | | | | |
| Sex | Female | Female | Male | Male |
| Age (yr) | 15 | 24 | 32 | 58 |
| Weight (lb) | 108 | 132 | 175 | 201 |
| Maximal Treadmill Data | | | | |
| \dot{V}_E ATPS (L·min ⁻¹) | 45 | | 130 | |
| P _B (mmHg) | 740 | 742 | 738 | 739 |
| T (°C) | 20 | 23 | 21 | 24 |
| F _E O ₂ (%) | 16.08 | 16.2 | 16.04 | 16.75 |
| F _E CO ₂ (%) | 4.94 | 5.01 | 5.12 | 4.73 |
| \dot{V}_I ATPS (L·min ⁻¹) | | 70 | | 75 |
| RH (%) | | 50 | | 25 |
| Submaximal Exercise Data | | | | |
| Speed (m·min ⁻¹) | 90 | 161 | | |
| Grade | 11 | 0 | | |
| Ergometer load (kp) | | | 4 | 3 |
| Pedaling rate (rev·min ⁻¹) | | | 80 | 50 |
| Flywheel circumference (m) | | | 6 | 2.4 |
| Step height (m) | | 0.305 | | |
| Step rate (steps·min ⁻¹) | | 30 | | |
| Field Test Data | | | | |
| PACER speed (km·hr ⁻¹) | 10 | | | |
| PACER time (min) | 12.5 | 9 | 16 | |
| 1-mi walk time (min) | | | | 17.6 |
| HR (b·min ⁻¹) | | | | 108 |

1. Correct the expired minute ventilations from ATPS to STPD for subjects No. 1 and 3.
2. Correct the inspired minute ventilations from ATPS to STPD for subjects No. 2 and 4.
3. Calculate the inspired minute ventilations from the expired minute ventilations using the Haldane transformation for

subjects No. 1 and 3.

4. Calculate the expired minute ventilations from the inspired minute ventilations using the Haldane transformation for subjects No. 2 and 4.

$$\dot{V}O_2 \text{ cons}$$

5. Calculate $\dot{V}O_2$ substituting the information given and computed from the maximal treadmill results in Equation B.2 for subjects No. 1, 2, 3, and 4. Present your answer in both relative ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and absolute ($\text{L}\cdot\text{min}^{-1}$) oxygen units.

$$\dot{V}CO_2$$

6. Calculate the $\dot{V}CO_2$ produced for subjects No. 1, 2, 3, and 4 using the information given and computed from the maximal treadmill results in $\text{L}\cdot\text{min}^{-1}$.
7. Calculate the oxygen consumed during submaximal walking for subject No. 1.
8. Calculate the oxygen consumed during submaximal running for subject No. 2.
9. Calculate the oxygen consumed during submaximal cycling, assuming that subject No. 3 is doing leg work and subject No. 4 is doing arm cranking.
10. Calculate the oxygen consumed during bench stepping for subject No. 2.

$$\dot{V}O_2 \text{ max}$$

11. Calculate the estimated $\dot{V}O_2$ for subject No. 1 and No. 2 using the appropriate formula for the information presented.

Solutions and Answers

1. Subject No. 1

$$\dot{V}_{E\text{STPD}} =$$

$$45 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^\circ}{273^\circ + 20^\circ} \right) \times \left(\frac{740 - 17.5 \text{ mmHg}}{760} \right)$$

$$= 39.86 \text{ L} \cdot \text{min}^{-1}$$

Subject No. 3

$$\dot{V}_{E\text{STPD}} =$$

$$130 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^\circ}{273^\circ + 21^\circ} \right) \times \left(\frac{738 - 18.7 \text{ mmHg}}{760} \right)$$

$$= 114.25$$

2. Subject No. 2

$$\dot{V}_{E\text{STPD}} =$$

$$70 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^\circ}{273^\circ + 23^\circ} \right) \times \left(\frac{742 - [0.5 \times 21.1] \text{ mmHg}}{760 \text{ mmHg}} \right)$$

$$= 62.13 \text{ L} \cdot \text{min}^{-1}$$

Subject No. 4

$$\dot{V}_{E\text{STPD}} =$$

$$75 \text{ L} \cdot \text{min}^{-1} \left(\frac{273^\circ}{273^\circ + 24^\circ} \right) \times \left(\frac{739 - [0.25 \times 22.4] \text{ mmHg}}{760} \right)$$

$$= 66.52 \text{ L} \cdot \text{min}^{-1}$$

3. Subject No. 1

$$\dot{V}_{E\text{STPD}} = 39.86 \text{ L} \cdot \text{min}^{-1} \left(\frac{1 - [0.1608 + 0.0494]}{0.7904} \right)$$

$$= 39.83$$

Subject No. 3

$$\dot{V}_{E\text{STPD}} = 114.25 \text{ L} \cdot \text{min}^{-1} \left(\frac{1 - [0.1604 + 0.0512]}{0.7904} \right)$$

$$= 113.96$$

4. Subject No. 2

$$\dot{V}_{E\text{STPD}} = 62.13 \text{ L} \cdot \text{min}^{-1} \left(\frac{0.7904}{1 - [0.1620 + 0.0510]} \right)$$

$$= 62.33$$

Subject No. 4

$$\dot{V}_{\text{E STPD}} = 66.52 \text{ L} \cdot \text{min}^{-1} \left(\frac{0.7904}{1 - [0.1675 + 0.0473]} \right)$$

$$= 66.96 \text{ L} \cdot \text{min}^{-1}$$

5. Subject No. 1

$$\dot{V}\text{O}_{2\text{cons}} = (0.2093 \times 39.83 \text{ L} \cdot \text{min}^{-1})$$

$$- (0.1608 \times 39.86 \text{ L} \cdot \text{min}^{-1}) = 1.93 \text{ L} \cdot \text{min}^{-1}$$

$$= 1930 \text{ mL} \cdot \text{min}^{-1} \div 49.1 \text{ kg}$$

$$= 39.31 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Subject No. 2

$$\dot{V}\text{O}_{2\text{cons}} = (0.2093 \times 62.13 \text{ L} \cdot \text{min}^{-1})$$

$$- (0.1620 \times 62.33 \text{ L} \cdot \text{min}^{-1}) = 2.91 \text{ L} \cdot \text{min}^{-1}$$

$$= 2910 \text{ mL} \cdot \text{min}^{-1} \div 60 \text{ kg}$$

$$= 48.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Subject No. 3

$$\dot{V}\text{O}_{2\text{cons}} = (0.2093 \times 113.96 \text{ L} \cdot \text{min}^{-1})$$

$$- (0.1604 \times 114.25 \text{ L} \cdot \text{min}^{-1}) = 5.53 \text{ L} \cdot \text{min}^{-1}$$

$$= 5530 \text{ mL} \cdot \text{min}^{-1} \div 79.55 \text{ kg}$$

$$= 69.52 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Subject No. 4

$$\dot{V}\text{O}_{2\text{cons}} = (0.2093 \times 66.52 \text{ L} \cdot \text{min}^{-1})$$

$$- (0.1675 \times 66.96 \text{ L} \cdot \text{min}^{-1}) = 2.71 \text{ L} \cdot \text{min}^{-1}$$

$$= 2710 \text{ mL} \cdot \text{min}^{-1} \div 91.36 \text{ kg}$$

$$= 29.66 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

6. Subject No. 1

$$\dot{V}\text{CO}_2 \text{ prod} = (0.0494 \times 39.86 \text{ L} \cdot \text{min}^{-1})$$

$$= 1.97 \text{ L} \cdot \text{min}^{-1}$$

Subject No. 2

$$\dot{V}\text{CO}_2 \text{ prod} = (0.0501 \times 62.33 \text{ L} \cdot \text{min}^{-1})$$

$$= 3.12 \text{ L} \cdot \text{min}^{-1}$$

Subject No. 3

$$\dot{V}\text{CO}_2 \text{ prod} = (0.0512 \times 114.25 \text{ L} \cdot \text{min}^{-1})$$

$$= 5.85 \text{ L} \cdot \text{min}^{-1}$$

Subject No. 4

$$\dot{V}\text{CO}_2 \text{ prod} = (0.0473 \times 66.96 \text{ L} \cdot \text{min}^{-1})$$

$$= 3.17 \text{ L} \cdot \text{min}^{-1}$$

7. Subject No. 1

$$\dot{V}\text{O}_2 \text{ cons} = 90 \text{ m} \cdot \text{min}^{-1} \times \frac{0.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}}$$

$$+ 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} + (0.11 \times 90 \text{ m} \cdot \text{min}^{-1}$$

$$\times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}}) = 30.32 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

8. Subject No. 2

$$\dot{V}\text{O}_2 \text{ cons} = 161 \text{ m} \cdot \text{min}^{-1} \times \frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}}$$

$$+ 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} = 35.7 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

9. Subject No. 3

$$\dot{V}\text{O}_2 \text{ cons} = \left[\frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times (4 \text{ kg} \times 6 \text{ m} \cdot \text{rev}^{-1} \right.$$

$$\left. \times 80 \text{ rev} \cdot \text{min}^{-1}) \div 79.55 \text{ kg} \right]$$

$$+ 7 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

$$= 50.44 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

Subject No. 4

$$\dot{V}\text{O}_2 \text{ cons} = \left[\frac{3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times (3 \text{ kg} \times 2.4 \text{ m} \cdot \text{rev}^{-1} \right.$$

$$\left. \times 50 \text{ rev} \cdot \text{min}^{-1}) \div 91.4 \text{ kg} \right]$$

$$+ 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

$$= 15.32 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

10. Subject No. 2

$$\begin{aligned}\dot{V}O_{2\text{cons}} &= \left[\frac{0.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times 30 \text{ steps} \cdot \text{min}^{-1} \right] \\ &+ \left[1.33 \times \frac{1.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{\text{m} \cdot \text{min}^{-1}} \times 0.350 \text{ m} \cdot \text{step} \right. \\ &\quad \left. \times 30 \text{ steps} \cdot \text{min}^{-1} \right] + 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \\ &= 31.41 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

$\dot{V}O_2 \text{ max}$

11. **Subject No. 1** PACER
B.23)

(Eq.

$$\begin{aligned}\dot{V}O_2 \text{ max} &= 31.025 + 3.238 (10) \\ &\quad - 3.248 (15) + 0.1536 (10 \times 15) \\ &= 37.73 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\end{aligned}$$

$\dot{V}O_2 \text{ max}$

Subject No. 2 PACER
B.24)

(Eq.

$$\dot{V}O_2 \text{ max} = -23.4 + 5.8 (12.5) = 49.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$$

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Appendix C

Answers to Check Your Comprehension

CHAPTER 1

Check Your Comprehension 1

1. Static exercise
2. Static exercise
3. Short-term, light to moderate submaximal aerobic exercise
4. Incremental aerobic exercise to maximum
5. Long-term, moderate to heavy submaximal aerobic exercise
6. Very short-term, high-intensity anaerobic exercise
7. Dynamic resistance exercise
8. Long-term, moderate to heavy submaximal aerobic exercise
9. Incremental aerobic exercise to maximum
10. Short-term, light to moderate submaximal aerobic exercise
11. Dynamic resistance exercise
12. Very-short-term, high-intensity anaerobic exercise

Check Your Comprehension 2

Pattern (C) is the best. This incorporates step loading with recovery/rejuvenation microcycles programmed into the overload progression. Pattern (B) remains for too long at one level and makes too large a jump between levels. Pattern (A) progresses in

small increments but does not plan for any rest or recovery cycles. Overtraining is most likely to occur in individuals when large increases in training occur abruptly and when the rest and recovery periods included in the periodization program are insufficient.

Check Your Comprehension 3—Case Study 1

These responses may be discussed in any order.

1. **Specificity**—While cross-training can be good, Mark has insufficient specificity in his workouts to improve his 10-km time. His workouts are also too short as his race time shows. Suggestion: Mark needs to increase the number of days per week that he runs to at least 3; he also needs to gradually increase the distance he runs.

2. **Overload**—Mark's workouts are sufficient to maintain his fitness as indicated by his lack of change in his race time from the previous year. However, they are insufficient to improve his fitness and hence his time. Suggestion: Mark needs to prepare a miniperiodization steploading plan (remembering that he has only 3 months until the Turkey Trot) and follow it.

Note: This may be stated in reverse as the maintenance principle with the suggestions being to overload.

3. **Progression**—There is no indication that Mark has any progression in either the intensity or the duration of his workouts.

Suggestion: Mark needs to use steploading to progress to at least 10 km distance; he also needs to insert some high-intensity speed workouts.

4. **Individualization**—By letting Kristi set the pace, the training was individualized for her, but not for Mark.

Suggestion: Mark and Kristi need to do at least some of their weekly workouts separately—perhaps in the same area but at individual paces.



CHAPTER 2

Check Your Comprehension 1

See Table 2.1.

Check Your Comprehension 2

The actual number of ATP produced from the 18-carbon fatty acid stearate.

1. $n \div 2 - 1 = 18 \div 2 - 1 = 8$ cycles of beta-oxidation
2. $\text{FADH}_2 = 8 \times 1.5 \text{ ATP} = 12 \text{ ATP}$
 $\text{NADH}_2 = 8 \times 2.5 \text{ ATP} = 20 \text{ ATP}$
 $8 \times 4 \text{ ATP} = 32 \text{ ATP}$
3. Acetyl-CoA = 9:
 $9 \times 10 \text{ ATP} = 90 \text{ ATP}$
4. Activation energy = -2 ATP

$$\begin{array}{r} \text{Total: } 90 \text{ ATP} \\ + 32 \text{ ATP} \\ \hline 122 \text{ ATP} \\ - 2 \text{ ATP} \\ \hline 120 \text{ ATP} \end{array}$$

5.

CHAPTER 3

Check Your Comprehension 1—Case Study 1

Recommend that this client (Yeen Kuen) do aerobic activity first, followed by resistance activity.

This sequence does cause a slightly larger EPOC, elevation in energy expenditure, for the first 10 minutes of recovery. This is unlikely to even be noticed by the exerciser. However, on the

basis of the significantly higher HR and $\dot{V}O_2$ measures during the RE-RU sequence, it is easier physiologically to do the run first. If resistance work is done first, it could negatively impact the adherence of the beginning exerciser. Conversely, if a client was interested primarily in weight control and had been training consistently for some time, more calories would be burned doing the resistance activity before the aerobic activity.

Check Your Comprehension 2—Case Study 2

Miriam's maximal lactate steady state is at $10 \text{ km}\cdot\text{hr}^{-1}$. Her best time goal is approximately 1 hour and 30 minutes ($15 \text{ km} \div 10 \text{ km}\cdot\text{hr}^{-1} = 1.5 \text{ hr}$). Typically, however, MLSS indicates the pace that is sustainable for 30–60 minutes only. Given the extended duration of this event, she may have to run some portion of the race slower than this and come in somewhat over this time. At $9 \text{ km}\cdot\text{hr}^{-1}$, her time would be 1 hour 40 minutes ($15 \text{ km} \div 9 \text{ km}\cdot\text{hr}^{-1} = 1.66 \text{ hr}$), so between 1 hour 30 minutes and 1 hour 40 minutes is a reasonable goal.

CHAPTER 4

Check Your Comprehension 1—Case Study 1

Yes, this is a true max. $\text{RER} = 1.34$ (> 1.1 criterion level); $\text{HR} = 200 \text{ b}\cdot\text{min}^{-1}$ (predicted $220 - 22 = 198 \pm 12 = 186 - 210$); $\dot{\text{V}}\text{O}_2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ difference minutes 27 - 28 = 2.07 (less than half the expected $5.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$); $\dot{\text{V}}\text{O}_2$ reaches a plateau. In addition, the verification run meets the $\dot{\text{V}}\text{O}_2 \text{ max}$ criterion (it is within 2% of the incremental value), and the HR max is within $4 \text{ b}\cdot\text{min}^{-1}$ for both tests.

Check Your Comprehension 2

Minute 2: $\text{RER} = 0.99$; $\text{CHO} = 96.6\%$; $\text{FAT} = 3.4\%$

Minute 14: $\text{RER} = 0.96$; $\text{CHO} = 86.4\%$; $\text{FAT} = 13.6\%$

Minute 28: $\text{RER} = 1.34$; $\text{CHO} = 100\%$

Check Your Comprehension 3

$\text{RER} = 0.96$

$\dot{\text{V}}\text{O}_2 \text{ L}\cdot\text{min}^{-1} = 2.36$

$\text{kcal}\cdot\text{L O}_2^{-1} = 4.998$ (from **Table 5.4**)

$4.998 \text{ kcal}\cdot\text{L O}_2^{-1} \times 2.36 \text{ L O}_2\cdot\text{min}^{-1} = 11.80 \text{ kcal}\cdot\text{min}^{-1}$

$11.80 \text{ kcal}\cdot\text{min}^{-1} \times 4.18 \text{ kJ}\cdot\text{kcal}^{-1} = 49.30 \text{ kJ}\cdot\text{min}^{-1}$

Check Your Comprehension 4—Case Study 2

$$1,200 \text{ mL} \cdot \text{min}^{-1} \text{ O}_2 = 1.2 \text{ L} \cdot \text{min}^{-1} \text{ O}_2$$

$$1.2 \text{ L} \cdot \text{min}^{-1} \times 5 \text{ kcal} \cdot \text{L}^{-1} = 6 \text{ kcal} \cdot \text{min}^{-1}$$

$$300 \text{ kcal} \div 6 \text{ kcal} \cdot \text{min}^{-1} = 50 \text{ min}$$

Check Your Comprehension 5

$$\frac{35.75 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}} = 10.21 \text{ METs}$$

Check Your Comprehension 6—Case Study 3

Computing the $\% \dot{V}\text{O}_2$ values for the given oxygen costs of each individual at each speed, we get:

| Oxygen Cost | Daughter | Son | Mother | Father | Grandmother |
|-------------------------|----------|-----|--------|--------|-------------|
| 10 min·mi ⁻¹ | 77 | 78 | 60 | 62 | 83 |
| 9 min·mi ⁻¹ | 83 | 86 | 64 | 65 | 90 |
| 8 min·mi ⁻¹ | 94 | 98 | 76 | 79 | 100 |
| 7 min·mi ⁻¹ | 100 | 100 | 86 | 88 | — |

Therefore, the family could probably stay together easily at a 10-min·mi⁻¹ pace, a little less easily 9-min·mi⁻¹ because of the

grandmother's $\dot{V}\text{O}_2$. Perhaps the grandmother and the kids should run together somewhere between 9 and 10-min·mi⁻¹ and let mom and dad go faster.

CHAPTER 5

Check Your Comprehension 1

The matches for Columns A and B are 1 = C; 2 = D; 3 = A; 4 = B.

Check Your Comprehension 2

The shift to a higher amount and percentage of aerobic metabolism as a result of the warm-up is beneficial because when more oxygen is available sooner, less reliance is placed on anaerobic metabolism and less lactate accumulates at any given heavy workload. The dancers should be able to perform longer before feeling the consequences of the H⁺ from lactic acid.

Check Your Comprehension 3—Case Study 1

| Variable | Pretraining Value | Posttraining Value |
|---|-------------------|--------------------|
| $\dot{V}O_2$ max mL·kg ⁻¹ ·min ⁻¹ | 42.3 | 48.3 |
| RERmax | 1.06 | 1.11 |
| RER at 6 mph (160 m·min ⁻¹) | 0.93 | 0.84 |
| [La ⁻¹]max mmol·L ⁻¹ | 12 | 13.5 |
| [La ⁻¹] mmol·L ⁻¹ at 6 mph (160 m·min ⁻¹) | 2.8 | 2.3 |
| $\dot{V}O_2$ mL·kg ⁻¹ ·min ⁻¹ at 6 mph (160 m·min ⁻¹) | 35 | 34 |
| Resting ATP mM·g wet muscle ⁻¹ | 3.5 | 5.5 |
| Resting PC mM·g wet muscle ⁻¹ | 10 | 15 |
| Resting glycogen mM·g wet muscle ⁻¹ | 80 | 111 |
| Number of mitochondria mmol ³ | 0.45 | 1.08 |
| Glycogen phosphorylase mM·kg wet muscle ⁻¹ | 5 | 7 |
| Running velocity at LT2 mph (m·min ⁻¹) | 7 (190) | 8.5 (230) |

Yes, this individual is now more fit than before training as improvements have been made in all variables tested.

CHAPTER 6

Check Your Comprehension 1

Rank order for the glycemic load (GL) (from lowest to highest):

1. Gatorade = 16.7; 2. Snickers bar = 20.7; 3. Banana = 25.3;
4. Bagel = 36.1; 5. Pizza (supreme) = 37.4; 6. PowerBar (chocolate) = 39.0

The calculation of glycemic load (GL) alters the impact of some foods but not others. For example, Gatorade has the highest GI but the lowest GL; the pizza has the lowest GI but next to the highest GL. Both the banana and the PowerBar have moderate GI, but the banana has a moderate GL and the PowerBar the highest GL among the selected foods. Conversely, the bagel has both a high GI and GL, whereas the Snickers bar has both a low GI and GL. This occurs because of the higher number of grams of CHO in the typical portion of some foods than others.

Check Your Comprehension 2—Case Studies 1 and 2

- A. Female tennis player, 21 years, 117 lbs. Total caloric estimated daily need from Table 6.2 = 2,400 kcal

| | | | |
|--|--|---|--|
| Situation: Training 10:00–12:00 AM; 6:00–7:30 PM | | | |
| | Grams | Kilocalories | Percentage |
| Carbohydrate | $5\text{--}7 \text{ g}\cdot\text{kg}\cdot\text{d}^{-1}$ $117 \text{ lbs} \div 2.2$ $\text{lb}\cdot\text{kg}^{-1} =$ 53 kg $53 \text{ kg} \times 5$ $\text{g}\cdot\text{kg}\cdot\text{d}^{-1}$ $= 265; 53 \text{ kg}$ $\times 7 = 371 \text{ g}$ | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $265 \text{ g} \times 4$ $\text{kcal}\cdot\text{g}^{-1} =$ $1,060 \text{ kcal};$ 371×4 $\text{kcal}\cdot\text{g}^{-1} =$ $1,484 \text{ kcal}$ | $\text{CHO kcal} \div$ total kcal $1,060 \text{ kcal} \div$ $2,400 \text{ kcal}$ $= 45\%;$ $1,484 \text{ kcal/}$ $2,400 \text{ kcal}$ $= 62\%$ |
| Protein | $1.2\text{--}1.7 \text{ g}\cdot\text{kg}\cdot\text{d}^{-1}$ $64\text{--}90 \text{ g}$ | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $256\text{--}360 \text{ kcal}$ | $\text{PRO kcal} \div$ total kcal $11\text{--}15\%$ |
| Fat | $0.5\text{--}1.5 \text{ g}\cdot\text{kg}\cdot\text{d}^{-1}$ $27\text{--}80 \text{ g}$ | $\text{g} \times 9 \text{ kcal}\cdot\text{g}^{-1}$ $243\text{--}715$ | $\text{Fat kcal} \div$ total kcal $10\text{--}30\%.$ *Note the 10% would be too low. |
| Situation: Immediate postexercise recovery. Note the athlete has <8 hr to recover. | | | |
| | Grams | Kilocalories | |
| Carbohydrate | $1\text{--}1.2 \text{ g}\cdot\text{kg}\cdot\text{d}^{-1}$ $53\text{--}64 \text{ g}$ | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $212\text{--}256 \text{ kcal}$ | |
| Protein | $0.25 \text{ g}\cdot\text{kg}^{-1}$ 13 g | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ 52 kcal | |
| Fat | No recommendation | | |
| Suggest specific drinks, bars, and/or gels from Tables 6.4, 6.5, and 6.6 | For example: 8 oz whole chocolate milk (26 g CHO; 8 g PRO) plus Clif Bar (42 g CHO; 10 g PRO) = $272 \text{ kcal CHO} + 72 \text{ kcal PRO} = 344 \text{ kcal}$ | | |

B. Male marathon runner, 35 years, 145 lbs. Projected finish time 3:15.

| | | |
|--|---|--|
| Situation: 24 hr prior to event | Grams | Kilocalories |
| Carbohydrate | $10\text{--}12 \text{ g}\cdot\text{kg}^{-1}$ $145 \text{ lb} \div 2.2 \text{ lb}\cdot\text{kg}^{-1}$ $= 65.9 \text{ kg}$ rounded to 66 kg $66 \text{ kg} \times 10 \text{ g}\cdot\text{kg}^{-1}$ $= 660 \text{ g}$; $66 \text{ kg} \times$ $12 \text{ g}\cdot\text{kg}^{-1} = 792 \text{ g}$ | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $660 \text{ g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $= 2,540 \text{ kcal}$; 792 $\times 4 \text{ kcal}\cdot\text{g}^{-1} =$ $3,168 \text{ kcal}$ |
| Situation: 1–4 hr prior to event | Grams | Kilocalories |
| Carbohydrate | $1\text{--}4 \text{ g}\cdot\text{kg}^{-1}$ $66\text{--}264 \text{ g}$ | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $264\text{--}1,056 \text{ kcal}$ |
| Protein | Minimal if any | |
| Fat | Minimal if any | |
| Situation: 5 min prior to event | Grams | Kilocalories |
| Carbohydrate | 50 g | 200 kcal |
| Protein | Minimal if any | |
| Fat | Minimal if any | |
| Situation: During the event | Grams | Kilocalories |
| Carbohydrate | $90 \text{ g}\cdot\text{hr}^{-1}$; $45 \text{ g}\cdot 30$ min^{-1} ; $22 \text{ g}\cdot 15$ min^{-1} Blend of glucose and fructose. Fluid | $\text{g} \times 4 \text{ kcal}\cdot\text{g}^{-1}$ $360 \text{ kcal}\cdot\text{hr}^{-1}$; 180 $\text{kcal}\cdot 30 \text{ min}^{-1}$; 90 $\text{kcal}\cdot 15 \text{ min}^{-1}$ |
| Suggest specific drinks, bars, and/or gels from Tables 6.4, 6.5, and 6.6 | 6–8% CHO concentration in drinks. For example: Accelerade (S, F, GP) $80 \text{ kcal}\cdot 8 \text{ oz}^{-1}$; 14 g CHO; 6% concentration or Powerade Ion4 (F, GP); $75 \text{ kcal}\cdot 8\cdot\text{oz}^{-1}$; 18 g CHO; 8% concentration or GU gel (F, GP); 100 kcal per packet, 23 g CHO plus 8 oz water | |

CHAPTER 7

Check Your Comprehension 1—Case Study

$$\text{FFW} = 132 \text{ lb} \times [(100 - 27.2) \div 100] = 96.1 \text{ lb}$$

$$\text{WT2} = [(100 \times 96.1) \div (100\% - 22\%)] = 123.2 \text{ lb}$$

$$\Delta \text{WT} = 132 \text{ lb} - 123.2 \text{ lb} = 8.8 \text{ lb}$$

Phyllis needs to lose 8.8 lb to obtain 22% BF, assuming she maintains her muscle mass (FFW).

Check Your Comprehension 2—Case Studies 1–5

1. Enrico: sex = M; age = 27 years; height = 5 ft 11 in.; weight = 198 lbs; waist circumference = 44 in.

$$5 \text{ ft } 11 \text{ in.} = 71 \text{ in.} \times 2.54 \text{ cm} \cdot \text{in.}^{-1} = 180.34 \text{ cm} =$$

$$1.8 \text{ m} \times 1.8 \text{ m} = 3.24 \text{ m}^2$$

$$198 \text{ lb} \div 2.2 \text{ lb} \cdot \text{kg}^{-1} = 90 \text{ kg}$$

$$\text{BMI} = 90 \text{ kg} \div 3.24 \text{ m}^2 = 27.8 \text{ kg} \cdot \text{m}^{-2}$$

$$\text{Waist circumference} = 44 \text{ in.} \times 2.54 \text{ cm} \cdot \text{in.}^{-1} = 112 \text{ cm}$$

Overweight by $\text{BMI} \geq 25 \text{ kg} \cdot \text{m}^{-2}$; abdominal obesity by $\text{WC} \geq 99\text{--}102 \text{ cm}$

2. Zoe: sex = F; age = 20 years; height = 5 ft 4 in.; weight = 105 lbs; waist circumference = 25 in.; hip circumference = 30 in.

$$5 \text{ ft } 4 \text{ in.} = 64 \text{ in.} \times 2.54 \text{ cm} \cdot \text{in.}^{-1} = 162.56 \text{ cm} =$$

$$1.63 \text{ m} \times 1.63 \text{ m} = 2.66 \text{ m}^2$$

$$105 \text{ lbs} \div 2.2 \text{ lb} \cdot \text{kg}^{-1} = 47.73 \text{ kg}$$

$$\text{BMI} = 47.7 \text{ kg} \div 2.66 \text{ m}^2 = 17.9 \text{ kg} \cdot \text{m}^{-2}$$

$$\text{Waist circumference} = 25 \text{ in.} \times 2.54 \text{ cm} \cdot \text{in.}^{-1} =$$

63.5 cm

Hip circumference = $30 \text{ in.} \times 2.54 \text{ cm}\cdot\text{in.}^{-1} = 76.2 \text{ cm}$

Waist-to-hip ratio = $63.5 \text{ cm} \div 76.2 \text{ cm} = 0.83$

Underweight by BMI less than $18.5 \text{ kg}\cdot\text{m}^{-2}$; no abdominal obesity by $\text{WC} \leq 88/89 \text{ cm}$; W/H ratio ≤ 0.84

3. Abdullah: sex = M; age = 12 years; height = 4 ft 7 in.; weight = 120 lbs.

$4 \text{ ft } 7 \text{ in.} = 55 \text{ in.} \times 2.54 \text{ in.}\cdot\text{cm}^{-1} = 139.7 \text{ cm} = 1.4 \text{ m}$
 $1.4 \text{ m} \times 1.4 \text{ m} = 1.96 \text{ m}^2$

$120 \text{ lbs} \div 2.2 \text{ lb}\cdot\text{kg}^{-1} = 54.5 \text{ kg}$

$\text{BMI} = 54.5 \text{ kg} \div 1.96 \text{ m}^2 = 27.8 \text{ kg}\cdot\text{m}^{-2}$

Obese by CDC BMI Growth Chart Figure 7.8A.

4. Rudie: sex = F; age = 66 years; height = 5 ft 6 in.; weight = 187 lbs; waist circumference = 36 in.; hip circumference = 39 in.

$5 \text{ ft } 6 \text{ in.} = 66 \text{ in.} \times 2.54 \text{ cm}\cdot\text{in.}^{-1} = 167.6 \text{ cm} = 1.68 \text{ m}$
 $1.68 \text{ m} \times 1.68 \text{ m} = 2.8 \text{ m}^2$

$187 \text{ lbs} \div 2.2 \text{ lb}\cdot\text{kg}^{-1} = 85 \text{ kg}$

$\text{BMI} = 85 \text{ kg} \div 2.8 \text{ m}^2 = 30.4 \text{ kg}\cdot\text{m}^{-2}$

Waist circumference = $36 \text{ in.} \times 2.54 \text{ cm}\cdot\text{in.}^{-1} = 91 \text{ cm}$

Hip circumference = $39 \text{ in.} \times 2.54 \text{ cm}\cdot\text{in.}^{-1} = 99 \text{ cm}$

Waist-to-hip ratio = $91 \text{ cm} \div 99 \text{ cm} = 0.92$

Class I obesity by $\text{BMI} \geq 30 \text{ kg}\cdot\text{m}^{-2}$; abdominal obesity by $\text{WC} > 88/89 \text{ cm}$; WH ratio ≥ 0.90

5. Yourself

BMI = ? Waist circumference = ? W/H ratio = ?
Classifications = ?

CHAPTER 8

Check Your Comprehension 1—Case Study 1

The body weight planner have shown a caloric value of 2,312 calories/day for Pamela to reach her goal of 140 lbs in 180 days. The total daily caloric intake to maintain her current 152 lbs is 2,650 calories/day. $2,650 \text{ kcal} - 2,312 \text{ kcal} = 338 \text{ kcal} \cdot \text{d}^{-1} \times 180 \text{ d} = 60,840 \text{ kcal}$ total deficit. $60,840 \text{ kcal} \div 12 \text{ lbs} = 5,070 \text{ kcal} \cdot \text{lb}^{-1}$. This is 1,570 kcal more per pound than expected from the 3,500 kcal·lb⁻¹ rule. To maintain her weight at 140 lbs, Pamela will need to consume 161 kcal less per day than to maintain her original 152 lbs weight. Changing the activity level allows Pamela to ingest 202 more calories per day during the weight loss program (2,514 kcal·d⁻¹ with more exercise vs. 2,312 kcal·d⁻¹ with no change in exercise) and 206 more calories in maintaining her weight at 140 lbs (2,695 kcal·d⁻¹ with the additional exercise vs. -2,489 kcal·d⁻¹ with no change in exercise).

Check Your Comprehension 2—Case Study 2

$$\begin{aligned}\text{Danladi: HT} &= 6'2'' = 74 \text{ in.} \times 2.54 \text{ cm} \cdot \text{in.}^{-1} = 187.96 \text{ cm} \\ &= 188 \text{ cm}\end{aligned}$$

$$\text{WT} = 191 \text{ lb} \div 2.2 \text{ lb} \cdot \text{kg}^{-1} = 86.8 \text{ kg}$$

$$\text{RMR} = 88.362 + (4.799 \times \text{HT cm}) + (13.397 \times \text{WT kg})$$

$$- (5.677 \times \text{age yr})$$

$$= 88.362 + (4.799 \times 188) + (13.397 \times 86.8)$$

$$- (5.677 \times 48)$$

$$= 1,881.2 \text{ kcal} \cdot \text{d}^{-1}$$

RMR PAL = 1.4; $1.6 - 1.4 = 0.2$; $0.2 \times 1,881.2 \text{ kcal}\cdot\text{d}^{-1} = 376.24 \text{ kcal}\cdot\text{d}^{-1}$, more physical activity needed

Basketball = 6 METs

$$1 \text{ MET} = 1 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} \div 60 \text{ min}\cdot\text{hr}^{-1} = \text{kcal}\cdot\text{min}^{-1}$$

$$6 \text{ MET} = 6 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} \times 86.8 \text{ kg} \div 60 \text{ min}\cdot\text{hr}^{-1}$$

$$= 8.68 \text{ kcal}\cdot\text{min}^{-1}$$

$$376.24 \text{ kcal}\cdot\text{d}^{-1} \div 8.68 \text{ kcal}\cdot\text{min}^{-1} = 43 \text{ min}\cdot\text{d}^{-1}$$

Walking = 3.3 METs

$$1 \text{ MET} = 1 \text{ kcal}\cdot\text{kg}^{-1} \text{ hr}^{-1} \div 60 \text{ min}\cdot\text{hr}^{-1} = \text{kcal}\cdot\text{min}^{-1}$$

$$3.3 \text{ MET} = 3.3 \text{ kcal}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1} \times 86.8 \text{ kg} \div 60 \text{ min}\cdot\text{hr}^{-1} = 4.77 \text{ kcal}\cdot\text{min}^{-1}$$

$$376.24 \text{ kcal}\cdot\text{d}^{-1} \div 4.77 \text{ kcal}\cdot\text{min}^{-1} = 78.9 \text{ min} = 79 \text{ min}$$

$$3.3 \text{ METs} = 3.0 \text{ mi}\cdot\text{hr}^{-1}; 60 \text{ min}\cdot\text{hr}^{-1} \div 3.0 = 20 \text{ min}\cdot\text{mi}^{-1}; 78 \text{ min} \div 20 \text{ min}\cdot\text{mi}^{-1} = 3.95 \text{ mi}$$

Most of the physical activities should come from sports or planned physical activity because the professor's job is primarily sedentary. Higher MET level activities will allow him to spend less time in activity.

Check Your Comprehension 3—Case Study 3

Zachary's weight should be 132 lb. He needs to lose 6 lb. These results are computed as follows using Equation 8.2.

$$\text{DB} = 1.0982 - \left[0.000815(8+9+12) + 0.0000084(8+9+12)^2 \right]$$

$$= 1.0982 - [0.023635 + 0.0070644]$$

$$= 1.0982 - 0.0306994 = 1.0675 \text{ g}\cdot\text{cc}^{-1}$$

For a 14-year-old male, the %BF formula from Table 7.1 is

$$\%BF = ([5.07 \div 1.0675] - 4.64) \times 100 = 10.9$$

Using [Equation 7.4](#)

$$FFW = 138 \text{ lb} \times ([100\% - 10.9\%] \div 100) = 123 \text{ lb}$$

Zachary should wrestle at no less than 7%BF with a weight loss not exceeding 7% of body weight. Using [Equation 7.5](#)

$$WT_2 = ([100\% \times 123 \text{ lb}] \div [100\% - 7\%]) = 132.26 \text{ lb}$$

Using Equation 7.6

$$132 \text{ lb} - 138 \text{ lb} = -6 \text{ lb}$$

CHAPTER 9

Check Your Comprehension 1

1. Pattern A: Alveolar ventilation

$$\begin{aligned} & [(600 \text{ mL}\cdot\text{br}^{-1}) - (150 \text{ mL}\cdot\text{br}^{-1})] \times (10 \text{ br}\cdot\text{min}^{-1}) \\ & = (450 \text{ mL}\cdot\text{br}^{-1}) \times (10 \text{ br}\cdot\text{min}^{-1}) = 4,500 \text{ mL}\cdot\text{min}^{-1} \div \\ & 1,000 \text{ mL}\cdot\text{L}^{-1} \\ & = 4.5 \text{ L}\cdot\text{min}^{-1} \end{aligned}$$

Pattern B: Alveolar ventilation

$$\begin{aligned} & [(200 \text{ mL}\cdot\text{br}^{-1}) - (150 \text{ mL}\cdot\text{br}^{-1})] \times (30 \text{ br}\cdot\text{min}^{-1}) \\ & = (50 \text{ mL}\cdot\text{br}^{-1}) \times (30 \text{ br}\cdot\text{min}^{-1}) = 1,500 \text{ mL}\cdot\text{min}^{-1} \div \\ & 1,000 \text{ mL}\cdot\text{L}^{-1} \\ & = 1.5 \text{ L}\cdot\text{min}^{-1} \end{aligned}$$

At identical minute ventilations, alveolar ventilation is greatly reduced as the depth (tidal volume) of the ventilation decreases. Increasing the frequency of breathing does not compensate for a small tidal volume at the alveolar level.

Therefore, shallow, frequent breathing is not as effective as deep, infrequent breathing.

2. Both situations decrease alveolar ventilation. When tidal volume is low, as when trying to inhale without exhaling first or in taking short, quick gulps of air, the volume of the dead space has a negative impact on the amount of air available for exchange (the alveolar ventilation). This result is seen in the calculations in problem 1. Inadequate alveolar ventilation can lead to dizziness or unconsciousness, which are dangerous situations, especially in water.
3. A snorkel extends the dead space. Thus, the tidal volume must be increased sufficiently to compensate for that volume as well as the anatomical dead space to maintain effective alveolar ventilation.

Check Your Comprehension 2



| Site | PO ₂ | PCO ₂ |
|---------------------|---|--|
| Alveoli | 104 mmHg | |
| Pulmonary capillary | 40 mmHg, arterial end; 104 mmHg, venous end | |
| Left side of heart | 95 mmHg | |
| Systemic arteries | 95 mmHg | 40 mmHg |
| Tissue (resting) | 40 mmHg | 45 mmHg |
| Systemic capillary | 95 mmHg, arterial end; 40 mmHg, venous end | 40 mmHg, arterial end; 45 mmHg, venous end |
| Systemic veins | 40 mmHg | 45 mmHg |
| Right side of heart | 40 mmHg | 45 mmHg |
| Pulmonary artery | 40 mmHg | 45 mmHg |
| Alveoli | | 40 mmHg |
| Pulmonary capillary | | 45 mmHg, arterial end; 40 mmHg, venous end |
| Left side of heart | | 40 mmHg |

Check Your Comprehension 3—Case Study 1

$$\text{HbO}_2 \text{ mLO}_2\cdot\text{dL}^{-1} = \text{Hb gm}\cdot\text{dL}^{-1} \times 1.34 \text{ mLO}_2\cdot\text{gmHb}^{-1} \times \text{SbO}_2\% \text{ (as decimal)}$$

$$\text{Last year: } 13.2 \times 1.34 \times 0.97 = 17.16 \text{ mLO}_2\cdot\text{dL}^{-1}$$

$$\text{This year: } 11.9 \times 1.34 \times 0.97 = 15.47 \text{ mLO}_2\cdot\text{dL}^{-1}$$

The difference in oxygen-carrying capacity between this year and last is $17.16 \text{ mLO}_2\cdot\text{dL}^{-1} - 15.47 \text{ mLO}_2\cdot\text{dL}^{-1} = 1.69 \text{ mLO}_2\cdot\text{dL}^{-1}$ or a reduction of almost 10%.

CHAPTER 10

Check Your Comprehension 1—Case Study 1

| | Carter (M) | Sydney (F) | Expected Change |
|---|---------------|---------------|---|
| 1-mile run | 8:08 | 10:04 | Increase—longer time to cover same distance; because of the decrease in $\text{SaO}_2\%$ and PaO_2 at altitude |
| $\dot{V}\text{O}_2$ max $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ | 43 | 36.5 | Decrease—because of the decrease in $\text{SaO}_2\%$ and PaO_2 at altitude |
| Curl-ups | 32 | 26 | No change |
| 90-degree Push-ups | 28 | 12 | No change |
| Back saver sit-and-reach | 8 | 12 | No change |
| BMI $\text{kg}\cdot\text{m}^{-2}$ | 21.5 | 23.4 | No change or decrease; it is possible that some weight will be lost until acclimation of food intake |
| Trunk lift | 12 | 12 | No change |

Check Your Comprehension 2

Both exercise-induced arterial hypoxemia and altitude decrease maximal exercise performance. The common mechanism is a decrease in $\text{SaO}_2\%$ and PaO_2 .

CHAPTER 11

Check Your Comprehension 1

| Heart Period | Valves Open | Blood Flowing From | Blood Flowing To |
|--------------|------------------|--------------------|----------------------------|
| VFP | A-V valves | Atria | Ventricles |
| ICP | None | None | None |
| VEP | Semilunar valves | Ventricles | Aorta and pulmonary artery |
| IRP | None | None | None |

The force of ventricular contraction produces the force to eject blood from the ventricles during VEP. During the ICP, blood does not leave the heart because the semilunar valves are closed.

Check Your Comprehension 2

| Normal | Increased sympathetic nervous stimulation |
|--|---|
| EDV = 150; SV ~ 80 mL · b ⁻¹ | EDV = 150; SV ~ 90 mL · b ⁻¹ |
| EDV = 200; SV ~ 100 mL · b ⁻¹ | EDV = 200; SV ~ 105 mL · b ⁻¹ |
| EDV = 250; SV ~ 105 mL · b ⁻¹ | EDV = 250; SV ~ 112 mL · b ⁻¹ |
| 1. EDV = 350; SV ~ 95 mL · b ⁻¹ | EDV = 300; SV ~ 110 mL · b ⁻¹ |
| EF = $\frac{80}{150} = 53.3\%$ | EF = $\frac{90}{150} = 60\%$ |
| EF = $\frac{100}{200} = 50.0\%$ | EF = $\frac{105}{200} = 52.5\%$ |
| EF = $\frac{105}{250} = 42\%$ | EF = $\frac{112}{250} = 44.8\%$ |
| 2. EF = $\frac{95}{300} = 31.7\%$ | EF = $\frac{110}{250} = 44\%$ |

Check Your Comprehension 3

| | HR (b·min ⁻¹) | SV (mL·b ⁻¹) | Q̇ (L·min ⁻¹) |
|-------|---------------------------|--------------------------|---------------------------|
| Mike | 80 | 90 | 7.20 |
| Keiko | 60 | 120 | 7.20 |
| Kirk | 122 | 146.5 | 17.87 |
| Don | 72 | 88.05 | 6.34 |
| Nora | 58 | 98 | 5.68 |

Check Your Comprehension 4

$$PP = SBP - DBP = 150 - 90 = 60 \text{ mmHg}$$

$$MAP = \frac{PP}{3} + DBP = \frac{60}{3} + 90 = 110 \text{ mmHg}$$

$$TPR = \frac{MAP}{\dot{Q}} = \frac{110 \text{ mmHg}}{5.1 \text{ L} \cdot \text{min}^{-1}} = 21.57$$

CHAPTER 12

Check Your Comprehension 1

| Condition | MAP (mmHg) | TPR (units) | RPP (units) |
|-----------------------------|------------------|-------------|-------------|
| Rest | 102 ^a | 17.0 | 107 |
| Light aerobic | 118 ^a | 11.8 | 195 |
| Heavy aerobic | 129 ^a | 9.9 | 263 |
| Maximal aerobic | 144 ^a | 9.6 | 360 |
| Sustained static | 155 ^a | 19.4 | 284 |
| Dynamic resistance exercise | 136 ^a | 13.6 | 227 |

^aUse Equation 11.5a.

^bUse Equation 11.5b.

Check Your Comprehension 2—Case Study 1

The test was stopped because of the exaggerated blood pressure

response. Although it is normal for systolic blood pressure to increase during an incremental exercise test, the magnitude of Janet’s increase was abnormal. Furthermore, her diastolic blood pressure also increased. Based on her borderline high blood pressure and her exaggerated blood pressure response, the exercise specialist recommended that she follow up with her primary care physician.

Check Your Comprehension 3

| | MAP (mmHg) | HR (b·min ⁻¹) | SV (mL) | Q (L·min ⁻¹) | TPR (units) |
|-----------------------------|---------------|------------------------------|------------|-----------------------------|----------------|
| Preexercise | 90 | 68 | 75 | 5.1 | 17.6 |
| Immediately postexercise | 110 | 192 | 130 | 25.0 | 4.4 |
| Calculate percent change | 22.2% | 182.4% | 73.3% | 390.2% | −300.0% |

Yes, this is a normal, expected response. The drastic decrease in TPR occurs to increase blood flow to the active tissue while also limiting the rise in MAP. Furthermore, to facilitate increased blood flow to the working muscles, HR and SV both increase resulting in an increased Q.

Check Your Comprehension 4

No, it is not appropriate for Kara to exercise at the same HR on the stair climber, treadmill, and arm ergometer. Exercise prescriptions should be adjusted when an individual is doing upper body exercise. Upper body exercise, particularly when it involves a static component, leads to greater sympathetic stimulation than occurs during lower body exercise and results in higher blood pressure and total peripheral resistance.

Check Your Comprehension 5—Case Study 2

| | MAP (mmHg) | HR (b·min ⁻¹) | SV (mL) | Q (L·min ⁻¹) | TPR (units) |
|--------------------------------|---------------|------------------------------|------------|-----------------------------|----------------|
| Preexercise | 110 | 68 | 75 | 5.1 | 21.6 |
| Immediately postexercise | 150 | 90 | 80 | 7.2 | 20.8 |
| Calculate percent change | 36.4% | 32.4% | 6.7% | 41.2% | -3.7% |

During static muscular contractions, blood vessels embedded within the muscle are compressed, resulting in a rapid increase in both systolic and diastolic blood pressure (referred to as the pressor response). This results in a drastic rise in MAP. To help blood move against the large pressure response observed during static contractions, TRP does not decrease as greatly as it does during aerobic exercises. As is seen with aerobic exercise, the greater the exercise intensity, the greater the rise in HR. Due to increased intrathoracic pressure, as occurs during static contractions, preload may decrease while afterload typically increases. The change in preload and afterload results in decreased SV. While SV decreases during static contractions, the rise in HR counteracts the reduction in SV and Q increases.

CHAPTER 13

Check Your Comprehension 1

%HR_{max} Method

Mei:

$$\text{Estimated HR}_{\text{max}} = 220 - 50 = 170 \text{ b} \cdot \text{min}^{-1}$$

$$\text{Training HR} = 170 \times 0.57 = 97 \text{ b} \cdot \text{min}^{-1}$$

$$\text{Training HR} = 170 \times 0.63 = 107 \text{ b} \cdot \text{min}^{-1}$$

On the basis of the % HR_{max} method, a light exercise

for Mei elicits a heart rate between 97 and 107 $\text{b}\cdot\text{min}^{-1}$.

Serena:

$$\text{Estimated HR}_{\text{max}} = 220 - 60 = 160 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HR} = 160 \times 0.57 = 91 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HR} = 160 \times 0.63 = 101 \text{ b}\cdot\text{min}^{-1}$$

Using the % HR_{max} method, a light exercise for Serena elicits a heart rate between 91 and 101 $\text{b}\cdot\text{min}^{-1}$.

% HRR Method

Mei:

$$\text{Estimated HR}_{\text{max}} = 220 - 50 = 170 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HRR} = [(170 - 62) \times 0.30] + 62 = 94 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HRR} = [(170 - 62) \times 0.39] + 62 = 104 \text{ b}\cdot\text{min}^{-1}$$

On the basis of the % HRR method, a light exercise for Mei elicits a heart rate between 94 and 104 $\text{b}\cdot\text{min}^{-1}$.

Serena:

$$\text{Estimated HR}_{\text{max}} = 220 - 60 = 160 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HRR} = [(160 - 82) \times 0.30] + 82 = 105 \text{ b}\cdot\text{min}^{-1}$$

$$\text{Training HRR} = [(160 - 82) \times 0.39] + 82 = 112 \text{ b}\cdot\text{min}^{-1}$$

Using the % HRR method, a light exercise for Serena elicits a heart rate between 105 and 112 $\text{b}\cdot\text{min}^{-1}$.

Check Your Comprehension 2—Case Study

1

$\dot{V}O_2R$ or

1. A moderate workout represents 40–59% HRR. 4 mph is too low for Juan. 7, 8, and 9 mph are too high for everyone. 6 mph is moderate only for Juan. 5 mph falls within the moderate range for all runners.

| Individual | 40% | 59% |
|------------|--|--|
| Janet | $52 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $48.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.4$ $19.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{+3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $22.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ | $52 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $48.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.59$ $28.6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{\pm 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $32.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ |
| Juan | $64 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $60.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.4$ $24.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{+3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $27.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ | $64 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $60.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.59$ $35.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{\pm 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $39.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ |
| Mark | $49 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $45.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.4$ $18.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{+3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $21.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ | $49 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $45.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.59$ $26.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{\pm 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $30.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ |
| Gail | $56 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $52.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.4$ $21 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{+3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $24.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ | $56 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{-3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $52.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\times 0.59$ $31.0 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ $\underline{\pm 3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}}$ $34.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ |

2. To determine the anticipated heart rate during the 5-mph

run, first determine what percent $\dot{V}O_2 \text{ max}$ (as a fraction) each individual is working at. Refer to the box in

the chapter to find the $\dot{V}O_2 \text{ max}$ for each individual and the oxygen cost of running 5 mph.

Now rearrange the $TE_x \dot{V}O_2$ equation to solve for $\% \dot{V}O_2 R$.

$$\% \dot{V}O_2 R = \frac{Ex \dot{V}O_2 - \dot{V}O_{2 \text{ rest}}}{\dot{V}O_2 R}$$

Janet:

$$\frac{30.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{52 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}} = \frac{26.8}{48.5} = 0.553$$

Juan:

$$\frac{30.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{64 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}} = \frac{26.8}{60.5} = 0.443$$

Mark:

$$\frac{30.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{49 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}} = \frac{26.8}{45.5} = 0.589$$

Gail:

$$\frac{30.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}}{56 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} - 3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}} = \frac{26.8}{52.5} = 0.511$$

These percentages (as fractions) can then be used in the HRR equation.

| Janet | Juan | |
|----------------|----------------|--|
| 220 | 220 | |
| <u>-23</u> | <u>-35</u> | Age (yr) |
| 197 | 185 | Predicted HRmax (b·min ⁻¹) |
| <u>-60</u> | <u>-48</u> | RHR (b·min ⁻¹) |
| 137 | 137 | |
| <u>× 0.553</u> | <u>× 0.443</u> | % $\dot{V}O_2R$ /%HRR (b·min ⁻¹) |
| 76 | 61 | |
| <u>+60</u> | <u>+48</u> | RHR (b·min ⁻¹) |
| 136 | 109 | Exercise HR (b·min ⁻¹) |
| Mark | Gail | |
| 220 | 220 | |
| <u>-22</u> | <u>-28</u> | Age (yr) |
| 198 | 192 | Predicted HRmax (b·min ⁻¹) |
| <u>-64</u> | <u>-58</u> | RHR (b·min ⁻¹) |
| 134 | 134 | |
| <u>× 0.589</u> | <u>× 0.511</u> | % $\dot{V}O_2R$ /%HRR (b·min ⁻¹) |
| 79 | 69 | |
| <u>64</u> | <u>±58</u> | RHR (b·min ⁻¹) |
| 143 | 127 | Exercise HR (b·min ⁻¹) |

CHAPTER 14

Check Your Comprehension 1—Case Study 1

Based on the symptoms, Martha was likely suffering from exertional heat exhaustion due to the strenuous work in unaccustomed heat. It is important that Martha take steps (or is assisted) to cool down and rehydrate. If the symptoms do not improve or progress to blurred vision or collapse, medical attention should be sought as heat exhaustion can escalate to heat injury or even heatstroke, which is a serious medical condition that can lead to a fatality.

Check Your Comprehension 2

Coach Brown should observe the temperature, humidity, degree of direct sunlight on the field, and degree to which the uniforms interfere with heat dissipation or increase absorption of radiant heat. He should assess environmental conditions (using WBGT or heat stress index) and adjust the practice appropriately. The coach should cancel practice or limit activity if conditions are extreme (see Figure 14.1). He can have some drills performed in the shade or devise drills that can be performed without full gear to lessen heat stress.

The coach should be mindful of the fitness level, degree of acclimatization, and hydration level of his players. He should develop a periodization plan for general conditioning for all potential players to follow during the summer and before reporting for formal practice. He should begin the season with limited practice/workouts including some that can be safely done without helmets until players become acclimatized to the heat. He must make sure that players are hydrated at the onset of practice by having players weigh in at the beginning of practice to ensure that they do not have large fluctuations in daily weight attributable primarily to body water loss. Those whose weight loss appears to be hydration related need to be counseled about drinking throughout the day and possibly their NaCl intake needs to be checked. Water and sports drinks should be readily available during practice, and the coach should encourage drinking throughout practice and after practice. Coach Brown should also carefully monitor his players for signs and symptoms of heat stress and ensure that he has a way to cool and rehydrate any player he thinks may be suffering from early signs of heat illness. The culture of the team should encourage players to stop and seek shade and fluid if they feel any symptoms.

CHAPTER 15

Check Your Comprehension 1—Case Study

1

Yes, this is a very serious condition. Prediabetes that is not addressed often develops into diabetes, which is a devastating metabolic disease. Prediabetes also accelerates cardiovascular disease. Aging, obesity, and physical inactivity are associated with greater risk of prediabetes. While Jansson cannot stop aging, he can begin eating a more healthy diet, lose weight, and increase his physical activity. Furthermore, given the serious nature of the problem, he should schedule regular follow-ups with his physician to track his blood glucose levels.

Check Your Comprehension 2

The metabolic syndrome is diagnosed when an individual has three or more of the risk factors listed below:

1. Abdominal obesity defined as a waist circumference of greater than 102 cm for males and greater than 88 cm for females
2. Plasma TG ≥ 150 mg·dL⁻¹
3. HDL-C less than 40 mg·dL⁻¹ in males and less than 50 mg·dL⁻¹ for females
4. BP $\geq 135/85$ mmHg
5. Fasting glucose ≥ 110 mg·dL⁻¹ (American Heart Association, 2002)

Check Your Comprehension 3—Case Study 2

The table below presents the CVD risk analysis for Vivian:

How many total risk factors does Vivian have?

Vivian has five total risk factors. Refer to the table below to see which risk factors Vivian has and which risk factors she does not.



| Name the Six Major Modifiable Risk Factors | Provide Cutoff Numerical Values Including Units for Each Risk Factor | Provide Values for Vivian from Her History and Determine Whether She Has This Risk Factor |
|--|---|---|
| Cigarette smoking | Currently or stopped <6 months | Stopped smoking only 1 month ago. Yes, has this risk factor |
| Hypertension | SBP 130-139 or DBP 80-89 | 124/82 mmHg. No, does not have this risk factor, but she does have elevated blood pressure |
| Cholesterol-lipid fractions | $\geq 240 \text{ mg}\cdot\text{dL}^{-1}$ TC $< 40 \text{ mg}\cdot\text{dL}^{-1}$ HDL | TC = $280 \text{ mg}\cdot\text{dL}^{-1}$; HDL-C = $34 \text{ mg}\cdot\text{dL}^{-1}$. Yes, has this risk factor |
| Impaired fasting glucose | $\geq 100 \text{ mg}\cdot\text{dL}^{-1}$ on two separate occasions | Fasting glucose = 116 and $118 \text{ mg}\cdot\text{dL}^{-1}$. Yes, has this risk factor |
| Obesity | Waist/hip ratio > 0.86 for female | $95 \text{ cm} \div 103 \text{ cm} = 0.92$. Yes, has this risk factor |
| Physically inactive/sedentary lifestyle | No regular exercise program or less than SG recommendations | No regular exercise. Yes, has this risk factor |

CHAPTER 16

Check Your Comprehension 1

1. Low- to moderate-impact loading activities are recommended, including hiking, cross-country skiing, stair climbing activities on commercially available machines, and weight lifting. These are activities that promote bone health

while minimizing the risk of injury.

2. High-impact loading activities, such as sprinting, jumping, and soccer, would be recommended in order to promote the attainment of a high peak bone mass. High-impact activities increase the likelihood that an individual will attain her genetic potential for peak bone mass. She should also be careful to ensure adequate calcium intake in her diet.

Check Your Comprehension 2—Case Study 1

It is likely that Allison has a stress fracture. Stress fractures are often due to an abrupt increase in training volume, and this is more likely to cause a problem if footwear fits improperly or if running is performed on hard surfaces such as asphalt or concrete. Allison should review her risk factors, including her nutrient intake, and make training adjustments, particularly decreasing her mileage. If pain persists, she should consult a physician.

CHAPTER 17

Check Your Comprehension 1—Case Study 1

Based on his success in long-distance events, it is likely that Jackamo has a higher percentage of SO fibers. SO fibers have higher mitochondrial density, higher capillary density, and a higher myoglobin content. SO fibers also have higher triglyceride stores and higher oxidative enzymes than do fast-twitch fibers. These structural and metabolic properties of SO fibers are well suited to supporting long-distance events and account for the relative fatigue resistance of SO fibers.

Check Your Comprehension 2

To determine the percentage of ST fibers, divide the number of ST fibers (~12) by the total number of fibers (~41) and multiply by 100. The percentage of FT fibers can also be determined by subtracting the percentage of ST fibers from 100. (Some staining techniques also permit the calculation of the percentage of the subcategories of FT [FOG and FG] using the same procedure as above, although this is not possible in this example.)

Total number of fibers = 41

Number of FT fibers = 12

Number of ST fibers = 29

$$\begin{aligned}\% \text{ of FT} &= \frac{\text{Number of FT}}{\text{total fiber count}} \times 100 \\ &= \frac{12}{41} \times 100 = 29\%\end{aligned}$$

$$\% \text{ of ST} = \frac{\text{Number of ST}}{\text{total fiber count}} \times 100$$

This biopsy most likely came from Jackamo.

CHAPTER 18

Check Your Comprehension 1—Case Study 1

Claire and Louise are suffering from delayed-onset muscle soreness (DOMS). This was caused by the unaccustomed eccentric muscle contractions associated with running **down** the trail. Typically, the pain peaks during 24–48 hours and subsides within 96 hours. Research is not definitive, but they may find that they get some relief from taking a nonsteroidal anti-inflammatory

medication, using ice or contrast therapy, or using a compression garment. Perhaps the best advice is to do some activity, like gentle walking and gentle stretching until the pain subsides.

Check Your Comprehension 2

1. and 2.

| Name | Absolute Strength MVC (kg) | Relative Strength (kg·kg ⁻¹) | 50% MVC |
|-------|----------------------------|--|---------|
| Jody | 40.0 | 0.66 | 20.0 |
| Jill | 60.0 | 0.88 | 30.0 |
| Pat | 36.0 | 0.51 | 18.0 |
| Scott | 50.0 | 0.79 | 25.0 |
| Tom | 72.0 | 0.88 | 36.0 |
| Mike | 71.0 | 1.01 | 35.5 |

3. Tom; Mike

4. Tom

Check Your Comprehension 3

1. No, bending the knees (changing knee angle) does not eliminate the involvement of the thigh muscles. If the feet are supported (held down), the thigh muscles are more active than the rectus abdominis.
2. External obliques.
3. Not held.
4. From the available choices, you should have selected feet unsupported and knees bent at a 105-degree position in order to maximize the use of the abdominal muscles. These results indicate the best form of the sit-up from the standpoint of hip angle and foot support, but they do not take into account arm position. Also, they do not permit comparison with a curl-up or crunch (in which the head, shoulders, and trunk are lifted off the floor only about 30

degrees). Other research has actually shown that the abdominal muscles are responsible for only the first 30–45 degrees of the sit-up motion, and it is easier on the spinal discs if only a partial sit-up and not a full sit-up is performed. Combining this information, it must be concluded that on the basis of currently available information, a curl-up test with knees bent and feet unsupported is the exercise of choice for the abdominal muscles for most individuals.

CHAPTER 19

Check Your Comprehension 1

This player had indicated that her goal is to increase her strength during the preparation phase of her training. Since she is starting her fourth season as a forward, she should be considered intermediate or advanced in strength training. To maximize her strength gains, she should perform multiple sets of 6–12 repetitions. For rest between sets, 1–2 minutes would be appropriate.

Check Your Comprehension 2

Lifts should be performed in the following order: power cleans, lat pull-downs, back squat, bench press, leg extension, shoulder press, calf raises, shrugs, sit-ups, and forearm curls.

Check Your Comprehension 3—Case Study 1

This woman has indicated that her goal is to gain muscle strength. Since she is just beginning a program, she should be considered a novice. You should therefore recommend that she becomes familiar with proper technique for each lift and then begin a program that includes all the major muscles of the body

(probably 6–10 exercises). She should perform 1–3 sets of 8–12 reps at 60–70% of her 1-RM for each exercise. She should perform this exercise 2–3 d·wk⁻¹.

CHAPTER 20

Check Your Comprehension 1

1. A ballistic technique is to stand on a step and bounce on your toes so that your heel goes below step height and then above it. This exercise will not work on a level surface.
2. Walk on your heels for 10 yards both forward and backward.
3. A static technique is the wall stretch. Extend one leg straight back and stretch it, with the other bent at the knee and forward. Hold the position.
4. A CR PNF technique is to assume the long sitting position, a jump rope (or a towel or sweats) around your foot in a neutral position. Resist a maximal isometric contraction of foot, attempting to plantar flex. Relax and pull your foot into dorsiflexion with the implement. Repeat.
5. A CRAC PNF technique is the same as the CR technique, except that you actively contract the shin muscles (dorsiflex). Repeat.

CHAPTER 21

Check Your Comprehension 1—Case Study 1

Numerous hormones play a role in regulating metabolic responses to exercise, including insulin, glucagon, growth hormone, and cortisol. Cytokines released from the muscle are also implicated in regulating metabolic response. Collectively, these hormones

maintain blood glucose, increase fuel (free fatty acids and glucose) availability, and enhance the oxidation of these fuels to provide energy to support increased muscular activity—in this case, cycling.

Check Your Comprehension 2—Case Study 2

A bout of resistance training will result in increased levels of GH, IGF, and testosterone. These hormones play a role in the recovery from exercise and in the increase in muscle protein following exercise.

Chapter 22

Check Your Comprehension 1

Nick's fall and the abrasion he received resulted in tissue damage and may have allowed a foreign antigen to enter the body. The swelling, redness, and pain were the result of the inflammatory process, a process mediated by several chemical factors (including cytokines, complement, histamines, and prostaglandins) that increase vasodilation and capillary permeability and cause leukocytes to move to the site of injury. The inflammatory response helps to promote tissue repair by getting leukocytes to the damaged area so that they can ingest (and destroy) foreign invaders.

Check Your Comprehension 2

IL-6 is released primarily from skeletal muscle and thus is called *myokine*. IL-6 plays a key regulatory role, helping to regulate pro- and anti-inflammatory responses to exercise, affecting other hormones, and influencing metabolism.

Check Your Comprehension 3—Case Study

1



| Day | Training Session | Duration (min) | RPE: CR-10 scale | Load |
|---------------------------------|-----------------------|----------------|------------------|----------|
| Candace | | | | |
| M | Resistance | 90 | 5 | 450 |
| T | Speed, agility, power | 45 | 7 | 315 |
| W | Endurance running | 60 | 4 | 240 |
| TH | Speed, agility, power | 45 | 7.5 | 337.5 |
| F | Resistance | 90 | 5 | 450 |
| S | Speed, agility, power | 45 | 6.5 | 292.5 |
| SU | Endurance | 60 | 4 | 240 |
| Weekly load | | | | 2,325 |
| Standard deviation (SD) | | | | 88.2 |
| Daily load (weekly load/7) | | | | 332 |
| Monotony (daily load/SD) | | | | 3.76 |
| Strain (weekly load × monotony) | | | | 8,742 |
| Maya | | | | |
| M | Resistance | 90 | 4 | 360 |
| T | Speed, agility, power | 45 | 7.5 | 337.5 |
| W | Endurance running | 60 | 5 | 300 |
| TH | Speed, agility, power | 45 | 7.5 | 337.5 |
| F | Resistance | 90 | 3.5 | 315 |
| S | Speed, agility, power | 45 | 7.5 | 337.5 |
| SU | Endurance | 60 | 5 | 300 |
| Weekly load | | | | 2,287.5 |
| Standard deviation (SD) | | | | 22.4 |
| Daily load (weekly load/7) | | | | 326.8 |
| Monotony (daily load/SD) | | | | 14.6 |
| Strain (weekly load × monotony) | | | | 33,397.5 |

Maya is most likely to suffer from maladaptation if the training load continues as is. Although both players have comparable

daily training loads, Maya has a low standard deviation, indicating little day-to-day variation in load, that is, high monotony. As a result, she also has the higher training strain.



| Day | Training Session | Duration (min) | RPE: CR-10 scale | Load |
|--|-----------------------|----------------|------------------|---------|
| Candace | | | | |
| M | Resistance | 90 | 5 | 450 |
| T | Speed, agility, power | 45 | 7 | 315 |
| W | Endurance running | 60 | 4 | 240 |
| TH | Rest day | | | 0 |
| F | Resistance | 90 | 5 | 450 |
| S | Speed, agility, power | 45 | 6.5 | 292.5 |
| SU | Endurance | 60 | 4 | 240 |
| Weekly load | | | | 1,987.5 |
| Standard deviation (SD) | | | | 96.6 |
| Daily load (weekly load/7) | | | | 331 |
| Monotony (daily load/SD) | | | | 3.43 |
| Strain (weekly load \times monotony) | | | | 579.5 |
| Maya | | | | |
| M | Resistance | 90 | 4 | 360 |
| T | Speed, agility, power | 45 | 7.5 | 337.5 |
| W | Endurance running | 60 | 5 | 300 |
| TH | Rest day | | | 0 |
| F | Resistance | 90 | 3.5 | 315 |
| S | Speed, agility, power | 45 | 7.5 | 337.5 |
| SU | Endurance | 30 | 5 | 150 |
| Weekly load | | | | 1,800 |
| Standard deviation (SD) | | | | 76.3 |
| Daily load (weekly load/7) | | | | 300 |
| Monotony (daily load/SD) | | | | 3.93 |
| Strain (weekly load \times monotony) | | | | 458 |

Neither player had a day of complete rest and both should have. Eliminating the TH speed, agility, and power workout is

recommended. In addition, if one of the endurance days for Maya is reduced from 60 to 30 min, her monotony and strain scores drop precipitously.

Glossary

1-RM The maximal weight that an individual can lift once during a dynamic resistance exercise.

Absolute Submaximal Workload A set exercise load performed at any intensity from just above resting to just below maximum.

Acclimatization The adaptive changes that occur when an individual undergoes prolonged or repeated exposure to a stressful environment; these changes reduce the physiological strain produced by such an environment.

Action Potential Reversal of polarity or change in electrical potential.

Adaptive Thermogenesis Any change in heat production or energy expenditure in response to a changing internal or external environment indicating either an increase or a decrease in the efficiency of energy utilization.

Adenosine Triphosphate (ATP) Stored chemical energy that links the energy-yielding and energy-requiring functions within all cells.

Adequate Intake (AI) Used when an RDA cannot be determined. The AI is an estimate of intake by healthy individuals.

Aerobic In the presence of, requiring, or utilizing oxygen.

Afterload Resistance presented to the contracting ventricle.

All-or-None Principle When a motor neuron is stimulated, all of the muscle fibers in that motor unit contract to their fullest extent or do not contract at all.

Alveolar Ventilation (VA) The volume of air available for gas exchange; calculated as tidal volume minus dead space volume times frequency.

Anabolic Resistance The decline in anabolic response provided by a particular anabolic stimulus as a result of aging.

Anaerobic In the absence of, not requiring, nor utilizing, oxygen.

Anorexia Athletica (AA) An eating disorder that is characterized

by a food intake less than that required to support the training regimen and by a body weight less than 95% of normal.

Anorexia Nervosa (AN) An eating disorder characterized by marked self-induced weight loss and an intense fear of fatness.

Apolipoprotein The protein portion of lipoproteins.

Archimedes' Principle The principle that a partially or fully submerged object will experience an upward buoyant force equal to the weight or the volume of fluid displaced by the object.

Arteriosclerosis The natural aging changes that occur in blood vessels, including thickening of the walls, loss of elastic connective tissue, and hardening of the vessel wall.

Arteriovenous Oxygen Difference (a-vO₂diff) The difference between the amount of oxygen originally carried in arterial blood and the amount returned in venous blood.

Atherosclerosis A pathological process that results in the buildup of plaque inside blood vessels.

Ballistic Stretching A form of stretching, characterized by an action-reaction bouncing motion, in which the involved joints are placed into an extreme range of motion by fast, active contractions of agonistic muscle groups.

Basal Metabolic Rate (BMR) The level of energy required to sustain the body's vital functions in the waking state, when the individual is in a fasted condition, at normal body and room temperature, and without psychological stress.

Beta-Oxidation A cyclic series of steps that breaks off successive pairs of carbon atoms from FFA, which are then used to form acetyl-CoA.

Binge-Eating Disorder (BED) Recurring episodes of excessive eating and feeling out of control and/or guilty about it.

Body Composition The partitioning of body mass into FFM (weight or percentage) and fat mass (weight or percentage).

Body Mass Index (BMI) A ratio of the total body weight to height.

Bone Modeling The process of altering the shape of bone by bone resorption and bone deposition.

Bone Remodeling The continual process of bone breakdown (resorption) and formation (deposition of new bone).

Bulimia Nervosa (BN) An eating disorder marked by an unrealistic appraisal of body weight and/or shape that is manifested by alternating bingeing and purging behavior.

Caloric Balance Equation The mathematical summation of the

caloric intake (+) and energy expenditure (–) from all sources.

Caloric Cost Energy expenditure of an activity performed for a specified period of time. It may be expressed as total calories (kcal), calories or kilojoules per minute ($\text{kcal}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{min}^{-1}$), or relative to body weight ($\text{kcal}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ or $\text{kJ}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$).

Caloric Equivalent The number of kilocalories produced per liter of oxygen consumed.

Calorimetry The measurement of heat energy liberated or absorbed in metabolic processes.

Capacitance Vessels Another name for veins because of their distensibility, which enables them to pool large volumes of blood and become reservoirs for blood.

Carbohydrate Loading (Glycogen Supercompensation)

A process of nutritional modification that results in an additional storage of glycogen in muscle fiber up to two to three times the normal levels.

Carbon Dioxide Produced (VCO_2) The amount or volume of carbon dioxide generated during metabolism.

Cardiac Cycle One complete sequence of contraction and relaxation of the heart.

Cardiac Output (Q) The amount of blood pumped per unit of time, in liters per minute.

Cardiovascular Drift The changes in observed cardiovascular variables that occur during prolonged, heavy submaximal exercise without a change in workload.

Cardiovascular Fitness The ability to deliver and use oxygen under the demands of intensive, prolonged exercise or work.

Cellular Respiration The process by which cells transfer energy from food to ATP in a stepwise series of reactions. It relies heavily on the use of oxygen to produce energy.

Central Cardiovascular Adaptations Adaptations that occur in the heart that increase the ability to deliver oxygen.

Central Cardiovascular Responses Responses directly related to the heart.

Cholesterol A derived fat that is essential for the body but may be detrimental in excessive amounts.

Coenzyme A nonprotein substance derived from a vitamin that activates an enzyme.

Concentric Contraction A dynamic muscle contraction that produces tension during shortening.

Concurrent Training The integration of endurance- and resistance-

based training into a training program.

Contractility The force of contraction of the heart; the ability of a muscle to respond to a stimulus by shortening.

Contraction Tension-producing process of the contractile elements within muscle.

Coronary Heart Disease (CHD) Also called coronary artery disease or ischemic heart disease, CHD results from damage to the coronary arteries supplying the heart muscle (myocardium).

Coupled Reactions Linked chemical processes in which a change in one substance is accompanied by a change in another.

Criterion Test The most accurate tests for any given variable; the measurement standard against which other tests are judged.

Cross-Bridging Cycle The cyclic events necessary for the generation of force or tension within the myosin heads during muscle contraction.

Cross-Training The development or maintenance of cardiovascular fitness by alternating between or concurrently training in two or more modalities.

Cutting Decreasing body fat and body water content to very low levels in order to increase muscle definition.

Cytokines Proteins or peptides that are released from immune cells and other tissues (notably skeletal muscle and adipose tissue) and are involved in communication between immune cells (especially lymphocytes and phagocytes) and other cells of the body.

Delayed-Onset Muscle Soreness (DOMS) DOMS is a condition characterized by muscle tenderness, pain on palpitation, and mechanical stiffness that appears approximately 8 hours after exercise, increases and peaks over the next 24–48 hours, and usually subsides within 96 hours.

Densitometry The measurement of mass per unit volume.

Detraining The partial or complete loss of training-induced adaptations as a result of a training reduction or cessation.

Diastole The relaxation phase of the cardiac cycle.

Diastolic Blood Pressure (DBP) The force exerted on the wall of blood vessels by blood during relaxation of the heart (diastole).

Diet (a) The food regularly consumed during the course of normal living; (b) a restriction of caloric intake.

Diffusion The tendency of gaseous, liquid, or solid molecules to move from an area of higher concentration to an area of lower concentration by constant random action.

Doppler Echocardiography A technique that calculates stroke

volume from measurements of aortic cross-sectional area and time-velocity integrals in the ascending aorta.

Dose-Response relationship A description of how a change in one variable is associated with a corresponding change in another variable.

Dual-Energy X-Ray Absorptiometry The new criterion measure for determining body composition in a three-compartment model that use x-ray technology.

Dynamic Balance The ability to make necessary postural adjustments while the center of gravity and the base of support are in motion.

Dynamic Contraction A muscle contraction in which the force exerted varies as the muscle shortens to accommodate change in muscle length and/or joint angle throughout the range of motion while moving a constant external load.

Dyslipidemia Triad The combination of hypertriglyceridemia (high triglycerides), low HDL-C, and small, dense LDL-C, which is associated with a much higher risk of CVD than any single factor.

Dyspnea Labored or difficult breathing.

Eating Disorders (ED) Disturbances of eating habits or weight-control behavior that can result in significant impairment of physical health or psychosocial functioning.

Eating Disorders Not Otherwise Specified (EDNOS) Conditions of disordered eating that do not meet the complete criteria for AN, BED, or BN.

Eccentric Contraction A dynamic muscle contraction that produces tension (force) while lengthening.

Economy The oxygen cost of any activity, but particularly walking or running at varying speeds.

Ejection Fraction (EF) The percentage of end-diastolic volume that is ejected from the heart.

Elasticity The ability of a muscle to return to resting length after being stretched.

Electrocardiogram (ECG) Tracing that provides a graphic illustration of the electrical current generated by excitation of the heart muscle.

Electromyography (EMG) The measurement of the neural or electrical activity that brings about muscle contraction.

Electron Transport System (ETS) The final metabolic pathway, which proceeds as a series of chemical reactions in the mitochondria that transfer electrons from the hydrogen atom carriers NAD and FAD

to oxygen; water is formed as a by-product; the electrochemical energy released by the hydrogen ions is coupled to the formation of ATP from ADP and P_i .

End-Diastolic Volume (EDV) The volume of blood in the ventricle at the end of diastole.

End-Systolic Volume (ESV) The volume of blood in the ventricle at the end of systole.

Endothelium Single layer of epithelial tissue.

Energy Availability (EA) The amount of dietary energy remaining for all other metabolic processes after the energy cost of training is subtracted from the daily energy intake.

Energy System Capacity The total amount of energy that can be produced by an energy system.

Energy System Power The maximal amount of energy that can be produced per unit of time.

Enzyme A protein that accelerates the speed of a chemical reaction without itself being changed by the reaction.

Eupnea Normal respiration rate and rhythm.

Excess Postexercise Oxygen Consumption (EPOC) Oxygen consumption during recovery that is above normal resting values.

Exchange Vessels Another name for capillaries because this is the site of gas and nutrient exchange between the blood and tissues.

Excitation-Contraction Coupling The sequence of events by which an action potential in the sarcolemma initiates the sliding of the myofilaments, resulting in contraction.

Exercise A single acute bout of bodily exertion or muscular activity that requires an expenditure of energy above resting level and that in most, but not all, cases results in voluntary movement.

Exercise-Associated Hyponatremia (EAH) The occurrence of hyponatremia during or up to 24 hours after prolonged physical activity; it is diagnosed by a plasma sodium concentration below normal values (usually $135 \text{ mmol}\cdot\text{L}^{-1}$).

Exercise-Induced Arterial Hypoxemia (EIAH) A condition in which the amount of oxygen carried in arterial blood is severely reduced by $\geq 4\%$ consistently.

Exercise Modality or Mode The type of activity or sport; usually classified by energy demand or type of muscle action.

Exercise Physiology A basic and an applied science that describes, explains, and uses the body's response to exercise and adaptation to exercise training to maximize human physical potential.

Exercise Response The pattern of homeostatic disruption or change

in physiological variables during a single acute bout of physical exertion.

Exertional Heat Exhaustion A moderate illness characterized by an inability to maintain adequate cardiac output at moderate (38.5°C) to high ($>40^{\circ}\text{C}$) body temperatures.

Exertional Heat Illness (EHI) A range of multisystem illnesses related to elevated body core temperature and the cardiovascular and metabolic processes that result from exercise and the body's thermoregulatory response.

Exertional Heat Injury A moderate to severe progressive multisystem disorder, with hyperthermia accompanied by organ damage or severe dysfunction.

Exertional Heatstroke A life-threatening illness characterized by high body temperature and central nervous system dysfunction.

Extensibility The ability of a muscle to be stretched or lengthened.

External Respiration The exchange of gases between the lungs and the blood.

Fartlek Workout A type of training session, named from the Swedish word meaning "speed play," that combines the aerobic demands of a continuous run with the anaerobic demands of sporadic speed intervals.

Fast Glycolytic (FG, Type IIX or IIX) Fibers Fast-twitch muscle fibers that perform primarily under glycolytic conditions.

Fast Oxidative Glycolytic (FOG, Type IIA or IIA) Fibers Fast-twitch muscle fibers that can work under oxidative and glycolytic conditions.

Fat-Free Weight The weight of body tissue excluding extractable fat.

Fatigue Index (FI) Percentage of peak power drop-off during high-intensity, short-duration work.

Female Athlete Triad A portion of the relative energy deficiency in sport syndrome of interrelated conditions including disordered eating, menstrual dysfunction, and skeletal demineralization.

Pick Equation An equation used to calculate cardiac output from oxygen consumption ($\dot{V}\text{O}_2$) and arteriovenous oxygen difference ($a\text{-vO}_2\text{diff}$).

Field Test A performance-based test that can be conducted anywhere and that estimates the values measured by the criterion test.

First Law of Thermodynamics or the Law of Conservation of Energy Energy can neither be created nor destroyed but only changed in form.

Flavin Adenine Dinucleotide (FAD) A hydrogen carrier in cellular respiration.

Flexibility The range of motion in a joint or series of joints that reflects the ability of the musculotendon structures to elongate within the physical limits of the joint.

Food Efficiency An index of the number of calories an individual needs to ingest to maintain a given weight or percent body fat.

Gluconeogenesis The creation of glucose in the liver from noncarbohydrate sources, particularly glycerol, lactate or pyruvate, and alanine.

Glycemic Index (GI) A measure that compares the elevation in blood glucose caused by the ingestion of 50 g of any carbohydrate food with the elevation caused by the ingestion of 50 g of white bread or glucose.

Glycogen Stored form of carbohydrate composed of chains of glucose molecules chemically linked together.

Glycogenolysis The process by which stored glycogen is broken down (hydrolyzed) to provide glucose.

Glycolysis The energy pathway responsible for the initial catabolism of glucose in a 10- or 11-step process that begins with glucose or glycogen and ends with the production of pyruvate (aerobic glycolysis) or lactate (anaerobic glycolysis).

Health-Related Physical Fitness That portion of physical fitness directed toward the prevention of or rehabilitation from disease, the development of a high level of functional capacity for the necessary and discretionary tasks of life, and the maintenance or enhancement of physiological functions in biological systems that are not involved in performance but are influenced by habitual activity.

Heart Rate (HR) The number of cardiac cycles per minute.

Heart Rate Recovery (HR_{rec}) Decrease in heart rate during standard time period (usually 1 min) after exercise test.

Heart Rate Variability (HRV) The beat-to-beat variation in the time of the R to R intervals.

Heat Index A scale used to determine the risk of heat stress from measures of ambient temperature and relative humidity.

Heat Strain The physiological responses and resulting thermoregulatory processes to combat heat stress.

Heat Stress The physical work and environmental components that combine to create heat load on an individual.

Hematocrit The ratio of blood cells to total blood volume, expressed as a percentage.

Hemoglobin (Hb) The protein portion of the red blood cell that binds with oxygen, consisting of four iron-containing pigments called hemes and a protein called globin.

High-Density Lipoprotein (HDL-C) A lipoprotein in blood plasma composed primarily of protein and a minimum of cholesterol or triglyceride whose purpose is to transport cholesterol from the tissues to the liver.

Homeostasis The state of dynamic equilibrium (balance) of the internal environment of the body.

Hormones Chemical substances that originate in glandular tissue (or cells) and are transported through body fluids to a target cell to influence physiological activity.

Hydrolysis A chemical process in which a substance is split into simpler compounds by the addition of water.

Hydrostatic Weighing The historical criterion measure for determining body composition through the calculation of body density.

Hyperplasia Growth in a tissue or organ through an increase in the number of cells.

Hyperpnea Increased pulmonary ventilation that matches an increased metabolic demand, such as during exercise.

Hypertension High blood pressure, defined as adult values equal to or greater than 130/80 mmHg.

Hyperthermia The increase in body temperature with exercise.

Hyperventilation Increased pulmonary ventilation, especially ventilation that exceeds metabolic requirements; carbon dioxide is blown off, leading to a decrease in its partial pressure in arterial blood.

Hypokinetic Diseases Diseases caused by and/or associated with lack of physical activity.

Hypothermia A core temperature less than 35°C (95°F), resulting in the loss of normal function.

Ideal Cardiovascular Health The absence of manifest CVD together with the simultaneous presences of optimal levels of seven cardiovascular health metrics: not smoking, getting sufficient physical activity, having a healthy diet pattern, normal body weight, optimal total cholesterol, optimal blood pressure, and optimal fasting blood glucose.

Immune System A precisely ordered system of cells, hormones, and chemicals that regulate susceptibility to, severity of, and recovery from infection and illness.

- Impulse** An electrical charge transmitted through certain tissue that results in the stimulation or inhibition of physiological activity.
- Incidence** The rate of new cases of a disease in a specific population.
- Intercalated Discs** The junction between adjacent cardiac muscle cells that forms a mechanical and electrical connection between cells.
- Internal Respiration** The exchange of gases between the blood and the tissues at the cellular level.
- Interval Training** An aerobic and/or anaerobic workout that consists of three elements: a selected work interval (usually a distance), a target time for that distance, and a predetermined recovery period before the next repetition of the work interval.
- Irritability** The ability of a muscle to receive and respond to stimuli.
- Isokinetic Contraction** A muscle fiber contraction in which the velocity of the contraction is kept constant.
- Isokinematic Contraction** A muscle contraction in which the rate of limb displacement or joint rotation is held constant with the use of specialized equipment.
- Isometric Contraction** A muscle fiber contraction that does not result in a length change in muscle fiber.
- Isotonic Contraction** A muscle fiber contraction in which the tension generated by the muscle fiber is constant through the range of motion.
- Kilocalorie** The amount of heat needed to raise the temperature of 1 kg of water by 1°C at 1 atmosphere.
- Krebs Cycle** A series of eight chemical reactions that begins and ends with the same substance; energy is liberated for direct substrate phosphorylation of ATP from ADP, and P_i carbon dioxide is formed and hydrogen atoms removed and carried by NAD and FAD to the electron transport system; does not directly utilize oxygen but requires its presence.
- Laboratory Test** Precise, direct measurement of physiological functions for the assessment of exercise responses or training adaptations; usually involves monitoring, collection, and analysis of expired air, blood, or electrical signals.
- Lactate Thresholds** Points on the linear-curvilinear continuum of lactate accumulation that appear to indicate sharp rises, often labeled as the first (LT1) and second (LT2) lactate threshold.
- Leukocytosis** An increase in circulating leukocytes (WBC).
- Lipoprotein** Water-soluble compound composed of apolipoprotein and lipid components that transport fat in the bloodstream.
- Load** Force exerted on the muscle.

Locomotor-Respiratory Coupling (LRC) A form of entrainment that involves the synchronization of limb movement and breathing frequency that accompanies rhythmical exercise.

Long Slow Distance (LSD) Workout A continuous aerobic training session performed at a steady-state pace for an extended time or distance.

Low-Density Lipoprotein (LDL-C) A lipoprotein in blood plasma composed of protein, a small portion of triglyceride, and a large portion of cholesterol whose purpose is to transport cholesterol to the cells.

Maximal (max) Exercise The highest intensity, greatest load, or longest duration exercise of which an individual is capable.

Maximal Lactate Steady State (MLSS) The highest workload that can be maintained over time without a continual rise in blood lactate; it indicates an exercise intensity above which lactate production exceeds clearance.

Maximal Oxygen Consumption ($\dot{V}O_{2\max}$) The highest amount of oxygen an individual can take in, transport, and utilize to produce ATP aerobically while breathing air during heavy exercise.

Maximal Voluntary Contraction (MVC) The maximal force that the muscle can exert.

Mean Arterial Pressure (MAP) The weighted average of SBP and DBP, representing the mean driving force of blood throughout the arterial system.

Mean Power (MP) The average power (force times distance divided by time) exerted during short-duration (typically 30 seconds) work.

Mechanical Efficiency The percentage of energy input that appears as useful external work.

Mechanotransduction The process by which a bone responds to a mechanical force on it.

MET A unit that represents multiples of the resting rate of oxygen consumption for any given activity.

Metabolic Flexibility The ability to transition from relying primarily on fat use for ATP production to carbohydrates use for ATP production as exercise intensity increases.

Metabolic Pathway A sequence of enzyme-mediated chemical reactions resulting in a specified product.

Metabolic Syndrome A cluster of interrelated risk factors of metabolic origin that directly promotes the development of atherosclerotic CVD and increases the individual's risk of diabetes.

- Metabolism** The total of all energy transformations that occur in the body.
- Microcirculation** Smallest vessels of the vascular system, including arterioles, venules, arteriovenous anastomoses, metarterioles, and true capillaries.
- Minerals** Elements, not of animal or plant origin, that are essential constituents of all cells and of many functions in the body.
- Minute Ventilation or Minute Volume (V_I or V_E)** The amount of air inspired or expired each minute, or the pulmonary ventilation rate per minute; calculated as tidal volume times frequency of breathing.
- Mitochondria** Cell organelles in which the formation of acetyl-CoA, Krebs cycle, electron transport, and oxidative phosphorylation take place.
- Morbidity** The number of people with a sickness or disease in a population.
- Mortality** The number of deaths in a population.
- Motor Unit** A motor neuron and the muscle fibers it innervates.
- Muscle Tension** Force developed when a contracting muscle acts on an object.
- Muscle Tonus** A state of low-level muscle contraction at rest.
- Muscular Endurance** The ability of a muscle or muscle group to repeatedly exert force against a resistance.
- Myocardial Oxygen Consumption** The amount of oxygen used by the heart muscle to produce energy for contraction.
- Myocardium** The heart muscle.
- Myoclonus** A twitching or spasm in a maximally stretched muscle group.
- Myocytes** The contractile cells of the heart (cardiac muscle cells).
- Myofibril** Contractile organelles composed of myofilaments.
- Myofilaments** Contractile (thick and thin) proteins responsible for muscle contraction.
- Neurotransmitters** Chemical messengers with which neurons communicate with target cells of either other neurons or effector organs.
- Nicotinamide Adenine Dinucleotide (NAD)** A hydrogen carrier in cellular respiration.
- Non-Weight-Bearing Exercise** A movement performed in which the body weight is supported or suspended and thereby not working against the pull of gravity.

- Normal Weight Obese** The term used for an individual with a normal or low BMI but a high %BF.
- Nutrient Timing** A nutritional strategy to manipulate the time that specific nutrients are consumed to help achieve a desirable outcome.
- Nutritional Periodization** The intentional manipulation of nutritional intake to optimally influence training adaptations.
- One-Repetition Maximum (1 rep max; 1-RM)** The most weight that can be lifted one time.
- Osteoblasts** Bone cells that cause the deposition of bone tissue (bone-forming cells).
- Osteoclasts** Bone cells that cause the resorption of bone tissue (bone-destroying cells).
- Osteocytes** Mature osteoblasts surrounded by calcified bone that help regulate the process of bone remodeling.
- Osteopenia** A condition of decreased bone mineral density (BMD) defined as a T-score of -1 to -2.5 , which means a BMD value greater than one standard deviation (SD) below (but not more than 2.5 SD below) values for normal young adults.
- Osteoporosis** A condition of porosity and decreased bone mineral density defined as a T-score below -2.5 , which indicates a BMD greater than 2.5 SD below values for young, normal adults.
- Overreaching (OR)** A short-term decrement in performance capacity that generally lasts only a few days to 2 weeks and from which the individual easily recovers.
- Overtraining Syndrome (OTS)** A state of chronic decrement in performance and ability to train in which restoration may take several weeks, months, or even years.
- Oxidation** A gain of oxygen, a loss of hydrogen, or the direct loss of electrons by an atom or substance.
- Oxidative Phosphorylation (OP)** The process in which $\text{NADH} + \text{H}^+$ and FADH_2 are oxidized in the electron transport system and the energy released is used to synthesize ATP from ADP and P_i .
- Oxygen Consumption ($\dot{V}\text{O}_2$)** The amount or volume of oxygen taken up, transported, and used at the cellular level.
- Oxygen Deficit** The difference between the oxygen required during exercise and the oxygen supplied and utilized. Occurs at the onset of all activity.
- Oxygen Dissociation** The separation or release of oxygen from the RBCs to the tissues.
- Oxygen Drift** A situation that occurs in submaximal activity of long duration, or above 70% $\dot{V}\text{O}_{2\text{max}}$, or in hot and humid conditions

where the oxygen consumption increases, despite the fact that the oxygen requirement of the activity has not changed.

Partial Pressure of a Gas (PG) The pressure exerted by an individual gas in a mixture; determined by multiplying the fraction of the gas by the total barometric pressure.

Peak Power (PP) The maximum power (force times distance divided by time) exerted during very-short-duration (5 seconds or less) work.

Percent Saturation of Hemoglobin (SbO₂%) The ratio of the amount of hemoglobin combined with oxygen to the total hemoglobin capacity for combining with oxygen, expressed as a percentage; indicated generally as SbO₂% or specifically as SaO₂% for arterial blood or as SvO₂% for venous blood.

Perfusion of the Lung Pulmonary circulation, especially capillary blood flow.

Periodization Plan for training based on a manipulation of the fitness components with the intent of peaking the athlete for the competitive season or varying health-related fitness training in cycles of harder or easier training.

Peripheral Cardiovascular Adaptations Adaptations that occur in the vasculature or muscles that increase the ability to extract oxygen.

Peripheral Cardiovascular Responses Responses directly related to the vessels.

Phosphorylation The addition of a phosphate (Pi).

Physical Activity Level (PAL) The ratio of total energy expenditure (TEE) to 24-hour resting or basal/resting energy expenditure (RMR), that is, TEE/RMR.

Physical Fitness A physiological state of well-being that provides the foundation for the tasks of daily living, a degree of protection against hypokinetic disease, and a basis for participation in sport.

Power The amount of work done per unit of time; the product of force and velocity; the ability to exert force quickly.

Prediabetes Blood glucose levels between 100 and 125 mg·dL⁻¹ confirmed by measurements on at least two separate occasions; also called insulin resistance.

Prehypertension Designation selected to identify individuals at high risk of developing hypertension; defined as blood pressure values between 120/80 and 140/90 mmHg.

Preload Volume of blood returned to the heart.

Pressor Response The rapid increase in both systolic pressure and

diastolic pressure during static exercise.

Prevalence The number of cases of a disease in a specific population at a given time.

Proprioceptive Neuromuscular Facilitation (PNF) A stretching technique in which the muscle to be stretched is first contracted maximally. The muscle is then relaxed and either is actively stretched by contraction of the opposing muscle or is passively stretched.

Pulmonary Ventilation The process by which air is moved into and out of the lungs.

Rate-Pressure Product (RPP) An estimate of the myocardial oxygen consumption, calculated as the product of heart (HR) and systolic blood pressure (SBP).

Rating of Perceived Exertion A subjective impression of overall physical effort, strain, and fatigue during acute exercise.

Reciprocal Inhibition The reflex relaxation of the antagonist muscle in response to the contraction of the agonist.

Recommended Daily Allowance (RDA) The average daily intake level that is sufficient to meet the nutrient requirement of 97–98% of healthy individuals by age and sex.

Reduction A loss of oxygen, a gain of electrons, or a gain of hydrogen by an atom or substance.

Reflex Rapid, involuntary response to stimuli in which a specific stimulus results in a specific motor response.

Relative Energy Deficiency in Sport (RED-S) A syndrome of impaired physiological and/or psychological function caused by low energy availability.

Relative Humidity The moisture in the air relative to how much moisture (water vapor) can be held by the air at any given ambient temperature.

Relative Submaximal Workload A workload above resting but below maximum that is prorated to each individual; typically set as some percentage of maximum.

Residual Volume (RV) The amount of air left in the lungs following a maximal exhalation.

Resistance Training A systematic program of exercises involving the exertion of force against a load, used to develop strength, endurance, and/or hypertrophy of the muscular system.

Resistance Vessels Another name for arterioles because this is the site of greatest resistance to blood flow in the vascular system.

Respiratory Cycle One inspiration and expiration.

Respiratory Exchange Ratio (RER) Ratio of the volume of CO₂ produced divided by the volume of O₂ consumed in the body as a whole.

Respiratory Quotient (RQ) Ratio of the amount of carbon dioxide produced divided by the amount of oxygen consumed at cellular level.

Resting Metabolic Rate (RMR) The energy expended while an individual is resting quietly in a supine position.

Risk Factor An aspect of personal behavior or lifestyle, an environmental exposure, or an inherited characteristic that has been shown by epidemiological evidence to predispose an individual to develop a specific disease.

Sarcomere The functional unit (contractile unit) of muscle fibers.

Sarcopenia The loss of contractile muscle mass associated with aging.

Sarcoplasmic Reticulum (SR) The specialized muscle cell organelle that stores and releases calcium.

Sedentary Behavior Any waking behavior that expends ≤ 1.5 METs of energy; often, but not always, stipulated as being in a reclining or sitting posture.

Skinfolds The double thickness of skin plus the adipose tissue between the parallel layers of skin.

Sliding Filament Theory of Muscle Contraction The theory that explains muscle contraction as the result of myofilaments sliding over each other.

Slow Oxidative (SO, Type I) Fibers Slow-twitch muscle fibers that rely primarily on oxidative metabolism to produce energy.

Spirometry An indirect calorimetry method for estimating heat production, in which expired air is analyzed for the amount of oxygen consumed and carbon dioxide produced.

Sport-Specific Physical Fitness That portion of physical fitness directed toward optimizing athletic performance.

Sports Anemia A transient decrease in red blood cells and hemoglobin level (grams per deciliter of blood).

Static Balance The ability to make adjustments to maintain posture while standing still.

Static Contraction A muscle contraction that produces an increase in muscle tension but does not cause meaningful limb displacement or joint displacement and therefore does not result in movement of the skeleton.

Static Stretching A form of stretching in which the muscle to be

stretched is slowly put into a position of controlled maximal or near-maximal stretch by contraction of the opposing muscle group and held for 30–60 seconds.

Steady State A condition in which the energy provided during exercise is balanced with the energy required to perform that exercise, and factors responsible for the provision of this energy reach elevated levels of equilibrium.

Strength The ability of a muscle or muscle group to exert maximal force against a resistance in a single repetition.

Stress The state manifested by the specific syndrome that consists of all the nonspecifically induced changes within a biological system; a disruption in body homeostasis and all attempts by the body to regain homeostasis.

Stress Fracture A fine hairline break in bone that occurs without acute trauma, is clinically symptomatic, and is detectable by x-rays or bone scans.

Stress Reactions Maladaptive areas of bone hyperactivity where the balance between resorption and deposition is progressively lost such that resorption exceeds deposition.

Stroke Volume (SV) Amount of blood ejected from the ventricles with each beat of the heart.

Substrate Fuel substance acted on by an enzyme.

Substrate-Level Phosphorylation The transfer of P_i directly from a phosphorylated intermediate or substrates to ADP without any oxidation occurring.

Supramaximal Exercise An exercise bout in which the energy requirement is greater than what can be supplied aerobically at $V.O_{2max}$.

Synapse The gap, or junction, between terminal ends of the axon and other neurons, muscle cells, or glands.

Syncytium A group of cells of the myocardium that function collectively as a unit during depolarization.

Systole The contraction phase of the cardiac cycle.

Systolic Blood Pressure (SBP) The force exerted on the wall of blood vessels by the blood as a result of contraction of the heart (systole).

Thermic Effect of a Meal (TEM) The increased heat production as a result of food ingestion.

Thermogenesis The production of heat.

Thermoregulation The process whereby body temperature is maintained or controlled under a wide range of environmental

conditions.

Tidal Volume (VT) The amount of air that is inspired or expired in one breath.

Torque The capability of a force to produce rotation of a limb around a joint.

Total Lung Capacity (TLC) The greatest amount of air that the lungs can contain.

Total Peripheral Resistance (TPR) or Resistance (R) The factors that oppose blood flow.

Tracking A phenomenon in which a characteristic is maintained, in terms of relative rank, over a long time span or even a lifetime.

Training A consistent or chronic progression of exercise sessions designed to improve physiological function for better health or sport performance.

Training Adaptations Physiological changes or adjustments resulting from an exercise training program that promote optimal functioning.

Training Principles Fundamental guidelines that form the basis for the development of an exercise training program.

Training Taper A reduction in training load before an important competition that is intended to allow the athlete to recover from previous hard training, maintain physiological conditioning, and improve performance.

Training Volume The total amount of work done, usually expressed as mileage or load and calculated as frequency times duration for anaerobic or aerobic continuous exercise or number of sets times number of repetitions for resistance exercise.

Transamination The transfer of the NH_2 amino group from an amino acid to a keto acid.

Transverse Tubules (T Tubules) Organelles that carry the electrical signal from the sarcolemma into the interior of the cell.

Uncompensable Heat Stress A condition in which the evaporative cooling that is needed is greater than the evaporative cooling permitted by the environment.

Valsalva's Maneuver Breath holding that involves closing of the glottis and contraction of the diaphragm and abdominal musculature.

Velocity at $\dot{V}\text{O}_2\text{max}$ The speed at which an individual can run when working at his or her maximal oxygen consumption, based on both submaximal running economy and $\dot{V}\text{O}_2\text{max}$.

Ventilatory Equivalent The ratio of liters of air processed per liter of oxygen used ($\text{V}_E/\text{V}_{\text{O}_2}$).

Ventilatory Thresholds Points where the rectilinear rise in minute ventilation breaks from linearity during an incremental exercise to maximum.

Vital Capacity (VC) The greatest amount of air that can be exhaled following a maximal inhalation.

Vitamins Organic substances of plant or animal origin that are essential for normal growth, development, metabolic processes, and energy transformations.

Voluntary Dehydration Exercise-induced dehydration that develops despite an individual's access to unlimited water.

Weight-Bearing Exercise A movement performed in which the body weight is supported by muscles and bones.

Weight Cycling Repeated bouts of weight loss and regain.

Wolff's Law Bone forms in areas of stress and is resorbed in areas of nonstress.

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